Int Urogynecol J (2000) 11:361–376

International Urogynecology Journal

Review Article

The Role of the Levator Ani Muscle in Evacuation, Sexual Performance and Pelvic Floor Disorders

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Abstract: This paper reviews the role of the levator ani muscle (LAM) in evacuation, sexual performance and pelvic floor disorders. The LAM fixes the vesical neck, anorectal junction and vaginal fornices to the side wall of the pelvis by means of the suspensory sling and hiatal ligament. On contraction it shares in the mechanism of evacuation (urination, defecation). During the sexual act vaginal distension by the erect penis evokes the vaginolevator and vaginopuborectalis reflexes, with a resulting LAM contraction. The LAM also contracts upon stimulation of the clitoris or cervix uteri, an action mediated through clitoromotor and cervicomotor reflexes. LAM contraction leads to upper vagina ballooning, which acts as receptacle for semen collection, to uterine elevation and straightening and to elongation and narrowing of the vagina. These actions enhance the sexual response and prepare the uterus and vagina for the reproductive process. During ejaculation LAM contraction facilitates semen ejection. Levator subluxation and sagging leads to levator dysfunction syndrome, which may present as pudendal canal syndrome.

Keywords: Ejaculation; Micturition; Proctalgia; Pudendal canal syndrome; Uterine cervix; Vulvodynia

Introduction

For many decades the levator ani muscle has had an undisputed place in the medical literature as a muscle of the pelvic floor. However, when we carried out studies into the anatomical and physiological details of this muscle, we found that it has a multifunctional nature that directly involves and interferes with other areas of the pelvic floor.

The pelvic floor muscles include the levator ani and the puborectalis. This paper reviews the role of the levator ani in evacuation, sexual performance and pelvic floor disorders.

Anatomical Considerations

The levator ani consists essentially of the pubococcygeus, the iliococcygeus being rudimentary in humans [1]. The latter is usually thin, and may be absent or replaced largely by fibrous tissue [2]. It has been demonstrated that the puborectalis muscle does not belong to the levator muscle and that each of them has a different origin, innervation and function [1,3]. Whereas the levator is a dilator to the intrahiatal organs the puborectalis is a constrictor. The pubococcygeus (levator) is funnel-shaped, with a transverse portion called the levator plate and a vertical portion called the suspensory sling [1,3] (Figs 1, 2). It arises from the pubic body and obturator fascia and extends across the pelvis, forming the levator plate. At the vesical neck and anorectal junction the levator plate bends sharply downwards to form the suspensory sling.

The levator plate is a cone-shaped structure which stretches across the pelvis. The levator hiatus, an oval opening, occupies its anterior portion and the anococcygeal raphe exists posteriorly. It transmits the intrahiatal structures, which include the anal canal in both sexes, the prostate in males and the vagina and urethra in females. The levator plate muscle bundles were found to be arranged in two main groups, each with its own attachment and direction [3] (Fig. 2): the lateral bundles,

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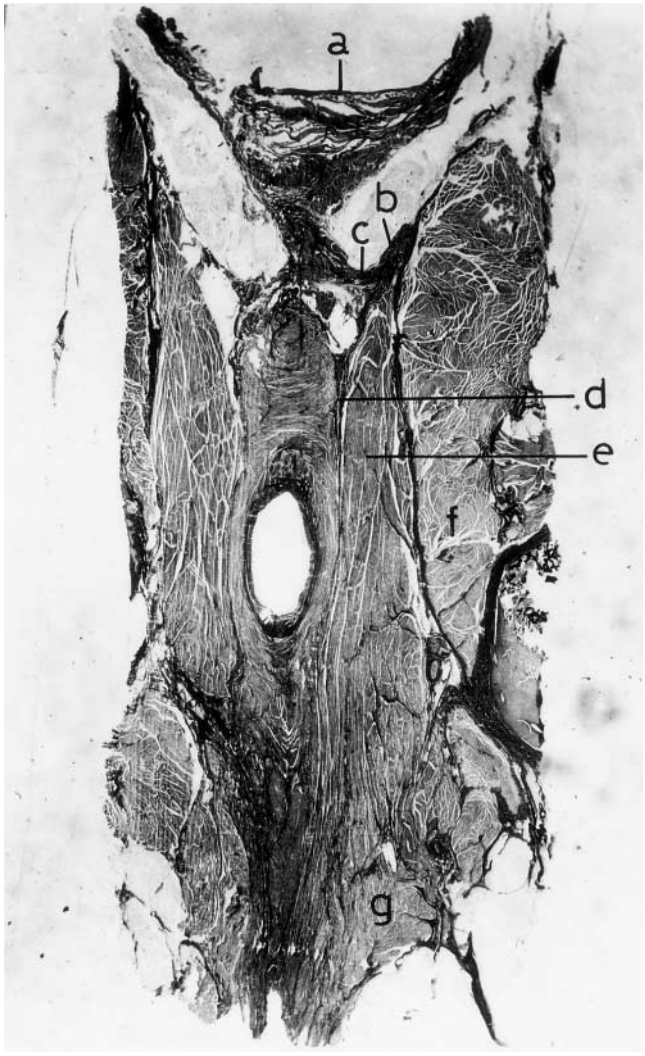

Fig. 1. Paracoronal section of urinary bladder and urethra in female cadaver. (Verhoeff–van Giessen stain ×5.) a, urinary bladder; b, levator plate; c, hiatal ligament; d, internal sphincter; e, suspensory sling; f, puborectalis; g, striated urethral sphincter (from Shafik [23]).

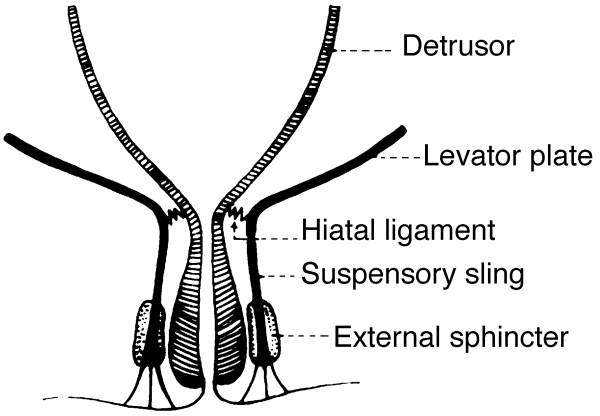

Fig. 3. Levator ani muscle and hiatal ligament (from Shafik [23]).

which form what I call the 'lateral masses', and the medial bundles, forming two strips which I termed the 'crura' (Fig. 2). Three patterns of crural origin could be identified: classic pattern, crural overlap and crural scissors [3].

Suspensory Sling

At the level of the levator hiatus the levator plate bends downward, forming the suspensory sling as a vertical muscular cuff around the intrahiatal organs [1,3] (Figs 1, 2). Detailed study of the latter has shown that it consists of longitudinally arranged striped muscle bundles impregnated with collagen. It was separated from the intrahiatal structures by a fascial septum which I call the 'tunnel septum' [3]. From a descriptive viewpoint, the suspensory sling can be divided into two parts: anal and urethral.

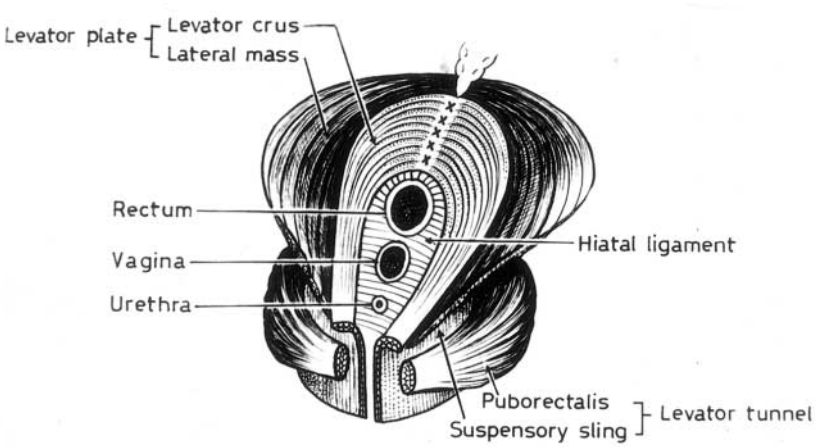

Fig. 2. Diagram illustrating the levator tunnel (from Shafik [3]).

Role of Levator Ani in Pelvic Organ Fixation

The levator plate consists of two zones which seem to be functionally different: a lateral 'visceral support' zone, represented by the two lateral masses, and a medial 'dilator' zone, represented by the two crura (Fig. 2). The two lateral masses are relatively fixed muscular sheets which, on contraction, together with the iliococcygeus, become elevated and function to support the viscera [3]. The two crura appear to represent the functionally active components of the levator plate.

The levator ani is connected to the vesical neck, anorectal junction and vaginal fornices by means of the hiatal ligament and suspensory sling. As the suspensory sling is a direct continuation of the levator plate, it seems to connect these structures to the side wall of the pelvis (Fig. 3). When the levator contracts on straining during evacuation (urine or stool) the suspensory sling also contracts, with a resultant slinging up and fixing of the urethra, anal canal and vagina [1,3] (Fig. 3).

While the suspensory sling fixes the urethra, anal canal and vagina in a vertical plane, the hiatal ligament provides a horizontal suspension for them (Figs 1,3) [1,3]. These structures are continuously exposed to intraabdominal tension variations due to respiratory movements and straining. The hiatal ligament, which constitutes a flexible mobile connection between levator plate and bladder neck, anorectal junction and vaginal fornices, appears to allow for a certain degree of mobility during respiration, urination and defecation. Moreover, it seals the levator hiatus and appears to prevent the intra-abdominal pressure transmitting through the hiatus to the infralevator structures and interfering with their functional activity (Figs 1, 2). The hiatal ligament is thus suggested to keep the infralevator compartment 'pressure-tight'. Our findings of the infralevator location of the urethra differ from the concept of other investigators, who claim its supralevator position [4–8]. This and other studies [1,3,9–11] have demonstrated that the levator ani is connected to the urethra at the bladder neck. The supralevator location of the urethra, as reported by some investigators [4–8], means that the levator extends downward to the lower end of the urethra, a description that could not be demonstrated in our study, nor by other investigators [1–3,9,10]. The suggestion of these investigators [4–8] is that the rise in intra-abdominal pressure compresses not only the bladder but also the urethra when it is located in the supralevator position. Meanwhile, other investigators reported that cases with an infralevator position of the urethra had no stress urinary incontinence [11–13], confirming the view that the urethra is protected in the infralevator pressure-tight compartment.

Role of Levator Ani in Evacuation

There are two components to the evacuation mechanism (urination and defecation): intrinsic and extrinsic. The intrinsic component comprises the detrusor and its sphincter; it acts involuntarily, being composed of smooth fibers. It is, however, under the voluntary control of the extrinsic component, which comprises the levator ani and the striated urethral or anal sphincter, the fibers of which are striped. The intrinsic component functions reflexly: as the detrusor contracts, the internal sphincter reflexly relaxes [14–16]. However, the continuation of the evacuation act depends essentially on the extrinsic component.

If evacuation is acceded to, the striated sphincter is voluntarily relaxed. Straining seems to have a role in evacuation: it elevates the intra-abdominal pressure, which serves two purposes: it compresses and helps evacuation of the detrusor, and it initiates levator contraction, which is mediated through the straining-levator reflex [17]. Although the intra-abdominal pressure compresses the detrusor, it spares the urethra, anal canal and vagina, owing to their location in the pressure-tight infralevator compartment.

The levator plate is connected to the vesical neck, anorectal junction and vaginal fornices along the hiatal ligament, and to the striated urethral or anal sphincter through the suspensory sling (Fig. 1). On straining at evacuation the levator crura contract and are elevated and retracted laterally. In so doing, they seem to pull the hiatal ligament, which in turn pulls open the vesical neck, anal canal and vaginal fornices [1,3,18,19] (Fig. 3). Meanwhile, the suspensory sling contracts simultaneously with the levator plate, being its downward prolongation. Its contraction is suggested to serve a threefold action: it shortens and widens the urethra, anal canal and vagina; it fixes and slings these structures to the side wall of the pelvis; and it pulls open the lower part of the striated urethral or anal sphincter by the action of the suspensory sling terminal fibers that pass through the sphincter; this leads to opening of the external urethral meatus, anal orifice or vaginal introitus [1,3,18,19] (Figs 1,3). The final result of levator contraction is that the urethra and anal canal are opened to evacuate their contents.

Physiology of Micturition

Studies have demonstrated that the action of the vesicourethral musculature during micturition is modulated and coordinated by a set of reflexes. Upon distension of the detrusor with urine stretch receptors are stimulated, resulting in detrusor contraction and internal sphincter relaxation [14–16]. Detrusor contraction triggers two reflexes: the vesicolevator [20] and the vesicopuborectalis [21], which act spontaneously but in opposite directions. Whereas the vesicolevator reflex effects levator contraction, which functions to open the vesical neck, a 'reflex' puborectalis contraction mediated through the vesicopuborectalis reflex keeps the vesical neck closed. Meanwhile, levator contraction evokes striated urethral sphincter contraction through the levator–urethral reflex [22].

Thus, although levator contraction on detrusor distension functions to open the vesical neck, urine control is secured by reflex double sphincter contraction: the puborectalis as a 'common' pelvic organ sphincter and the striated sphincter as an 'individual' urethral sphincter [23]. The vesical neck is kept temporarily closed by the contraction of these two sphincters, until a conscious decision is made whether to micturate or not. If conditions are favorable, the two sphincters relax voluntarily and micturition takes place.

If, however, circumstances are inopportune the vesical neck is kept closed by the 'voluntary' joint puborectalis and striated sphincter contraction, which evokes two reflex actions that lead eventually to abortion of the micturition reflex: reflex levator relaxation through the levator–puborectalis reflex [24], and reflex detrusor relaxation by means of the voluntary inhibition reflex [25]. Voluntary striated sphincter contraction, through the voluntary inhibition reflex, prevents internal sphincter relaxation, which results in reflex detrusor relaxation and abortion of the micturition reflex. When the urge to micturate wanes, the puborectalis and the striated sphincter relax and continence is maintained by the resting tone of the striated and internal urethral sphincters.

Role of the Levator Ani in the Performance of the Sexual Act

Levator Ani and Vagina

The vagina is related to the pelvic floor muscles, including the levator ani and puborectalis. A recent study has demonstrated a contraction of the levator ani muscle upon vaginal distension, and this was mediated through the vaginolevator reflex [19]. Upon contraction the levator plate is transformed from a cone into a flat shape [1,3], becoming elevated and laterally retracted, and pulling on the hiatal ligament which is attached to the vagina at the lateral fornices. These are pulled up and opened, resulting in elongation, narrowing and partial straightening of the vaginal tube, as well as elevation of the uterus [19] (Figs 4, 5). The upper part of the vagina, including the fornices, is widened and ballooned, the hiatal ligament being directly attached to it; the lower part is related to and supported by the sphincter vaginae and puborectalis muscle, which prevent its ballooning [26] (Figs 4, 5). Elongation and narrowing of the vaginal tube, together with the constrictive effect of the contracting puborectalis muscle and sphincter vaginae, could lead not only to adequate adaptation of the penis in

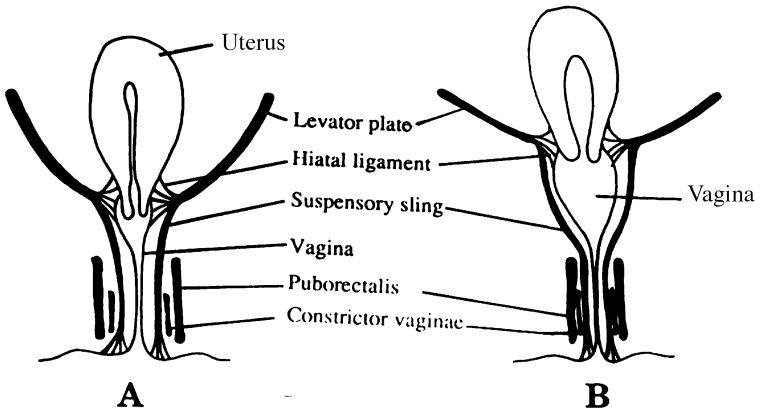

Fig. 4. Diagram illustrating the effect of levator ani and puborectalis muscles contraction on the vagina and uterus. Anteroposterior view (A) at rest; (B) upon levator contraction the muscle becomes elevated and laterally retracted, with a resulting elevation of the uterus as well as dilatation of the upper vagina and vaginal fornices. Puborectalis muscle contraction compresses the lower vagina (from Shafik [30]).

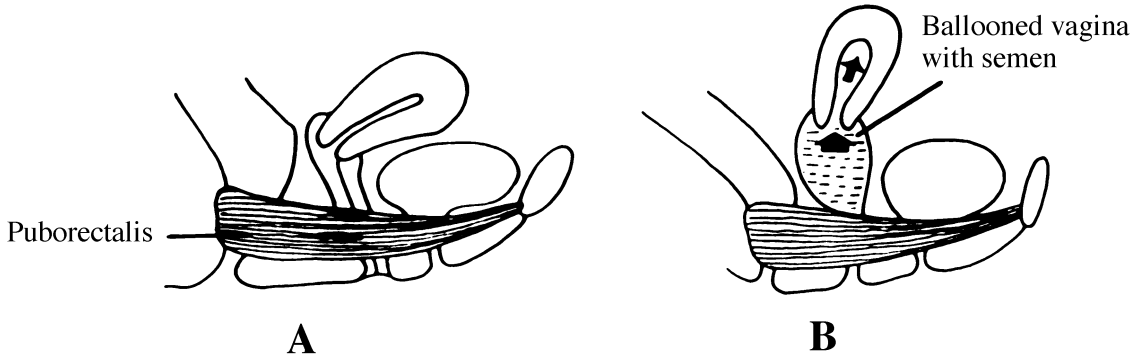

Fig. 5. Lateral view of the effect of levator ani and puborectalis muscle contraction on the vagina and uterus. (A) At rest; (B) upon levator and puborectalis contraction (from Shafik [30]).

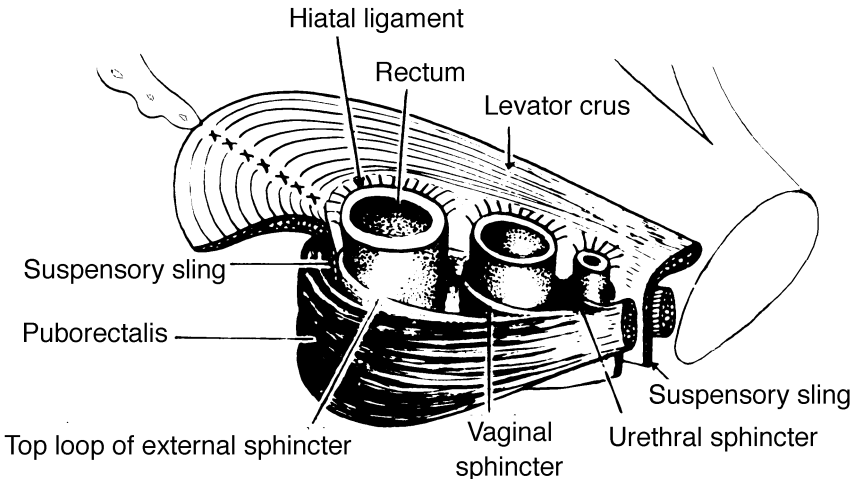

Fig. 6. Diagram illustrating the levator ani and puborectalis muscles with the 'individual' sphincters (from Shafik [23]).

the vagina, but also to an increase in penile rigidity. In the meantime, the suspensory sling contracts in association with the levator plate, resulting in eversion and widening of the vaginal introitus [19]. A previous study has demonstrated that the sphincter vaginae is derived from the puborectalis muscle [3,23] (Fig. 6).

During the sexual act, vaginal distension by the erect penis evokes the vaginolevator and vaginopuborectalis reflexes [19,26]. Levator muscle contraction seems to lead to genital responses that facilitate sexual performance. The patent and everted vaginal introitus invites the entry of the penis. Furthermore, the vaginolevator reflex could provide an explanation for the particular events that occur during the sexual act. The causes of the rise of the uterus in the pelvis, the elongation of the vagina and the ballooning of its upper part, as well as the irregular contractions of the vaginal vault that occur during the sexual act, had not been fully understood [27–29] and have been attributed by some investigators to contraction of the smooth muscle fibers within the parametrial tissues supporting the uterus and upper vagina [27]. However, these fibers are only scattered and do not constitute a formed muscle that could serve such functions. We believe that the above-mentioned genital responses result from levator contraction evoked by vaginal distension on penile thrusting. On contraction the levator plate becomes elevated and laterally retracted, pulling up the upper end of the vagina and the cervix uteri, straightening and elongating the vagina as well as ballooning its upper part [19]. The contractions at the vaginal vault during the sexual act seem to be simply levator muscle contractions that occur with penile thrusting.

These physiogenital changes in the female probably facilitate male sexual performance [19]. Thus, vaginal elongation and uterine elevation provide an appropriate penile–vaginal adaptation which adds to penile arousal. Furthermore, uterine elevation pulls the cervix out of the way of the deeply thrusted penis, thus avoiding the discomfort caused by buffeting the cervix. However, the function of the ballooning of the upper part of the vagina is not clear. The ballooned part, including the vaginal fornices, could act as a receptacle to collect semen, preventing its spilling outside the vagina [19] (Fig. 4). Its proximity to the cervix would also assist in conception.

Levator Ani and Clitoris and Cervix Uteri

An earlier study demonstrated contraction of both the levator ani and puborectalis muscles upon clitoral or cervix uteri stimulation, as evident from the increase in both vaginal pressure and EMG activity of the two muscles [30,31]. This was found to be initiated by the clitoromotor and cervicomotor reflexes [30,31]. The uterovaginal changes that occur upon clitoral or cervical stimulation are believed to serve a dual purpose: they enhance the sexual response in both sexes, and they prepare the uterus and vagina for the reproductive process.

LA contraction on clitoral or cervical stimulation pulls on the hiatal ligament, which opens the upper end of the vagina at the fornices. The upper vagina is ballooned, with a resultant lowering of pressure. Meanwhile, contraction of the puborectalis muscle with its derivative, the sphincter vaginae, compresses the lower two-thirds of the vaginal tube, the two muscles being related to this part of the vagina. This leads to an increase in vaginal pressure. However, the vaginal pressure in the lower third was higher than in the middle third, probably because the bulbocavernosus muscle shares the contraction of the puborectalis and sphincter vaginae [32,33]. The bulbocavernosus muscle encircles the lower vagina at the introitus. Accordingly, on clitoral or cervical stimulation the upper vagina balloons, with a drop in vaginal pressure, and the lower two-thirds become narrow, with a pressure increase. Furthermore, clitoral stimulation is associated with decline in uterine pressure [30]. The clitoro- and cervicomotor reflexes seem to play an important role in the sexual act that needs to be clarified.

Levator Ani and Penile Thrusting

With penile thrusting during coitus the clitoris and cervix may be repeatedly buffeted. Mechanical clitoral and cervical stimulation by the thrusting penis may evoke certain reflexes that enhance the sexual response in both male and female. These include the clitoro- and cervicomotor, and vagino- and cervicocavernosus reflexes [30–33], which lead to levator ani, puborectalis and cavernosus muscle contraction. The resulting intermittent narrowing of the vaginal introitus and lower two-thirds of the vagina may increase penile tumescence and rigidity.

Levator Ani and Reproductive Process

These changes seem to help the reproductive process. Thus, the ballooned upper vagina acts as a receptacle for semen collection, which appears to facilitate insemination. Moreover, on withdrawing from the vagina, the penis may be 'milked' by the contracting bulbocavernosus and puborectalis muscles, and some of the semen still contained in the urethra would be evacuated into the vagina [26,32,33]. In addition, the narrowing of the middle and lower vagina might help not only to close the vagina and prevent seepage of the semen through it to the exterior, but also to push the semen in the lower vagina up to the receptacle. Meanwhile, the elevation of the cervix probably brings the latter to a better position to receive the collected semen. The cervical os, instead of looking backward in the vaginal fornix, seems to be elevated and looks downward, facing the pool of semen in the vaginal receptacle.

The studies [30,31] show also that the uterus adapts to the reproductive process upon stimulation of the glans clitoris. The lowering of the uterine pressure upon glans clitoris stimulation is believed to help the 'suction' of the collected semen into the uterine cavity, especially as the reduced uterine pressure is lower than that in the upper vagina. Thus, the repeated successive narrowing of the lower vagina due to puborectalis contraction, the ballooning of the upper vagina, the rising of the cervix and uterus, and the drop in uterine pressure, seem to work collectively and reflexly through the clitoromotor reflex. The aim of these reflex physiologic changes is most likely to augment and facilitate the reproductive process.

Diagnostic Role of the Vaginolevator and Cervicomotor Reflexes

The available diagnostic techniques in female sexual dysfunction are poor. They include measurement of episodic vaginal blood flow changes during sleep [34] and vaginal responses to erotic stimuli in the waking state [35,36]. However, the diagnostic value of these tests is not yet clear. Sacral evoked responses have been shown to be impaired in women with orgasmic dysfunction [37]. The vaginolevator and cervicomotor reflexes may be of diagnostic significance in disorders of female sexual performance. Evoking the reflex is simple: it involves introducing a concentric needle electrode into the levator ani muscle, balloon inflation of the vagina or mechanical striking of the cervix, and recording the response on a standard EMG apparatus [19,31]. Absence of the reflex, a prolongation of latency or changes in the motor unit action potentials may indicate a defect in the reflex pathway. Therefore, the measurement of the latency or evoked potentials of the reflex could be included in investigations of disorders of female sexual performance [19,31].

Role of Levator Ani in Erection and Ejaculation

Earlier studies have demonstrated the role of the pelvic floor muscles and sphincters in erection and ejaculation [38,39].

Erection

All of the pelvic floor muscles and sphincters show a significant increase in EMG activity during the whole erectile period. It seems that this increased activity is preparatory to ejaculation. The puborectalis muscle embraces the prostate and the upper part of the anal canal. As it passes backward from its origin in the symphysis pubis, it gives rise to individual sphincters which comprise the prostatic and striated urethral sphincters as well as the deep external anal sphincter [3,23] (Fig. 6). The increased EMG activity of the puborectalis muscle during erection most likely involves its individual sphincters. This might result in compression of the internal reproductive organs (prostate and lower parts of vasal ampulla and seminal vesicles), thus expressing their secretions into the posterior urethra to be expelled to the exterior by the ejaculatory mechanism [38,39]. The puborectalis muscle might act in conjunction with contraction of the intrinsic musculature of the internal reproductive organs.

The function of increased EMG activity in the levator ani during erection is not known. However, the muscle may have a role during ejaculation, as will be discussed later.

The increased EMG activity of the external anal and striated urethral sphincters during erection might act to abort any urge to defecate or urinate and to prevent leakage of urine, feces and flatus during coitus [38,39]. Under normal physiologic conditions the internal sphincter (anal or urethral) relaxes when the detrusor (rectal or vesical) contracts [14–16]. If there is an urge but no desire to evacuate the external sphincters contract, and this results in reflex detrusor relaxation, accommodation for the contents and waning of the urge to evacuate [25]. This is mediated through the voluntary inhibition reflex [25]. It seems that sphincter contraction

during coitus not only aborts evacuation but prevents leakage of urine or stool by means of the sustained contraction of the external anal and striated urethral sphincters throughout erection.

Role of Puborectalis and Levator Ani Muscles, and External Anal and Striated Urethral Sphincters at Ejaculation

Ejaculation consists of two phases: emission and ejection [40,41]. The puborectalis muscle might, as previously suggested, share with the intrinsic musculature of the internal reproductive organs expressing the genital fluid into the posterior urethra, thus assisting the process of emission. The levator ani muscle may have a role during ejaculation. It is connected to the bladder neck by the hiatal ligament [1,3]. Upon contraction the muscle is elevated and retracted laterally, with a resulting elevation of the bladder neck and prostate. The prostatic elevation may partially straighten the kink at the prostatomembraneous urethra that occurs with penile elevation during erection and which may interfere with semen ejection at ejaculation [38,39].

The pelvic floor sphincters also seem to have a role at ejaculation. Orgasm and ejaculation may be associated with generalized skeletal muscle activity [42,43]. Abdominal muscle contractions may be so forceful that they compress the urinary bladder and rectum, with possible leakage of their contents. Sphincter contraction during orgasm most likely aims to prevent such leaks. Moreover, the rhythmic contraction of the striated urethral sphincter during ejaculation might act as a suction–ejection pump [38,39]. The striated urethral sphincter, while relaxing after contraction, might create a negative pressure in the posterior urethra and ejaculatory duct which could help evacuation of the seminal vesicles and vasal ampullae. Upon contraction the striated urethral sphincter ejects the secretions collected in the posterior urethra to the bulbous one, where they are ejected to the exterior by the rhythmic bulbocavernosus muscle contractions.

Role of Levator Ani in Defecation

The functions of the anorectal musculature at defecation are initiated and harmonized by voluntary impulses and reflex actions, as demonstrated in previous publications [14–16,44,45]. When the rectal detrusor is distended with fecal mass and the stretch receptors are stimulated, the rectoanal inhibitory reflex [14–16] is initiated whereby the rectal detrusor contracts and the internal sphincter relaxes. Detrusor contraction triggers two reflexes: the rectopuborectalis [44] and the rectolevator [45]. These two act simultaneously, yet have opposite functions: on detrusor contraction the rectolevator reflex effects a reflex levator contraction which opens the anal canal. At the same time, the reflex puborectalis contraction, activated by the rectopuborectalis reflex, functions to close or keep closed the anal canal until a conscious decision is made to defecate.

Voluntary puborectalis contraction evokes two reflex actions: reflex levator relaxation through the levator-puborectalis reflex [24], and reflex detrusor relaxation by means of the voluntary inhibition reflex [25]. Meanwhile, it aborts the rectoanal inhibitory reflex which relaxes the internal sphincter. Hence voluntary puborectalis contraction, through the voluntary inhibition reflex, prevents internal sphincter relaxation, which results in reflex detrusor relaxation, and waning of the urge to defecate. However, as soon as circumstances permit defecation and the sensation of desire has returned, the puborectalis muscle relaxes voluntarily and the detrusor evacuates its contents.

Role of Levator Ani in Genesis of Pathologic Conditions

The levator ani, being a principal muscle of the pelvic floor and sharing in the functional activity of the urinary bladder, vagina, rectum and internal reproductive organs (prostate, seminal vesicles, vasal ampulla), may undergo functional disorders that might lead to deranged (fecal and urinary) evacuation and sexual disorder.

Levator Dysfunction Syndrome

Under normal physiological conditions the main brunt of increased intra-abdominal pressure caused by straining at stool is borne by the levator plate, and in particular by its rectococcygeal raphe, which is its most dependent and durable part [1,3]. The levator hiatus, being plugged by the viscera, which are firmly fixed to the levator plate by the hiatal ligament, is immune to increased intra-abdominal pressure. Moreover, the hiatal ligament attachment to the vesical neck, anorectal junction and vaginal fornices firmly seals the pelvic floor and prevents intra-abdominal pressure leak to the infralevator structures (Fig. 7A). This mechanism is maintained as long as the increased intra-abdominal pressure is within its physiological limit. An increase beyond this limit, as in chronic straining, would tend to throw its load on the rectococcygeal raphe, hiatal ligament and levator hiatus. The rectococcygeal raphe and hiatal ligament, both tendinous, become overstretched and subluxated. Consequently the levator plate sags, leading to suspensory sling subluxation [1,3] (Fig. 7B). Being continuously exposed to increased intra-abdominal pressure, the sagging levator plate atrophies and acquires a vertically oblique position; the levator hiatus is consequently overwidened and lowered, so that most of the anal canal, urethra and vagina lie above it (Fig. 7B).

These changes in the levator plate and its ligaments would interfere with the normal evacuation (defecation/urination) mechanism, with the ultimate development of ‘levator dysfunction syndrome’ [1,3,46]. Thus, on

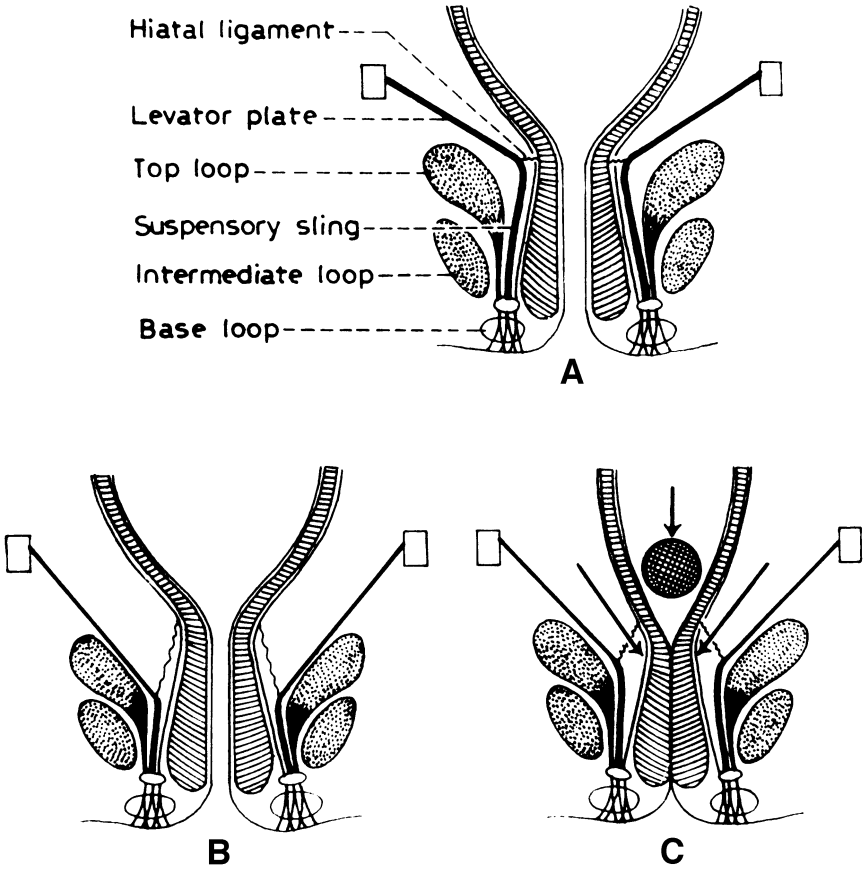

Fig. 7. The mechanism of defecation in levator dysfunction syndrome. (A) Normal findings; (B) pathological findings in levator dysfunction syndrome at rest; sagging of the levator plate as well as hiatal ligament and suspensory sling subluxation. Levator hiatus is widened and lowered so as to expose the anal canal to the intra-abdominal pressure; (C) levator dysfunction syndrome at defecation. On straining at stool, contraction of both the sagging levator plate and the subluxated suspensory sling is too weak to effect anal canal opening in front of the descending fecal mass. The increased intra-abdominal pressure leaks through the abnormally wide levator hiatus to the anal canal, closing it, with a resulting fecal obstruction (from Shafik [46]).

straining at stool or urination, the contraction of both the sagging atrophic levator plate and the subluxated suspensory sling is too weak to effect anal canal or vesical neck opening (Fig. 7C). This would explain the failure of the anal canal to expand with straining during digital anal palpation in the levator dysfunction syndrome. In the meantime, levator plate sagging with widening and lowering of the levator hiatus exposes the anal canal and urethra directly to intra-abdominal pressure. Thus, on straining at defecation and urination, the increased intra-abdominal pressure is transmitted through the abnormally wide levator hiatus to the anal canal and urethra, leading to their obstruction [1,3,46] (Fig. 7C). It seems that direct anal canal compression by the increased intra-abdominal pressure causes the high anal pressure detected during straining in all patients with levator dysfunction syndrome [46].

The ultimate result of levator dysfunction is impairment of the urination and defecation mechanisms, so that straining tends to close rather than open the anal canal and vesical neck. This is essentially attributable to atrophy and the vertically oblique position of the levator plate, leading to its inadequate contraction and thus to the failure of the anal canal and vesical neck to open and to leakage of the high intra-abdominal pressure through the widened levator hiatus to the anal canal and urethra, thereby obstructing them [1,3,46].

Causes of Levator Dysfunction

Conditions leading to chronic straining at stool or micturition or during a prolonged second stage of labor or forceps application would disturb levator function and ultimately result in levator dysfunction syndrome [46]. In view of the evident effect of chronic straining, first on the levator plate and subsequently on the whole of the defecation or urination mechanism, straining is to be considered the first stage in the developing of levator dysfunction syndrome.

It seems there is an individual susceptibility to straining that could depend on the different levator crural and rectococcygeal raphe patterns [3]. The crural overlap and scissors, as well as the triple-decussation

pattern of the levator ani, could hinder the full hiatal dilatation necessary for evacuation that occurs under normal physiologic conditions [3,46]. This, in consequence, would necessitate extra straining effort to achieve full dilatation. Meanwhile, these patterns seem to maintain the intrahiatal structures in position more firmly, so that a prolapse is less liable to occur in such cases despite the extra straining.

However, other possible causes of chronic straining should also be considered, such as occupational straining, severe loss of muscle tone from a debilitating disease or senility, or the increased intra-abdominal pressure due to a fat-laden viscera in obese, stoutly built subjects.

Treatment

The prime aim in the treatment of the levator dysfunction syndrome is to improve levator function. All traceable causes of straining should be eliminated. Straining at evacuation is to be strictly avoided. Levator muscle exercises and electrical stimulation, although they improve muscle tone, have only a temporary effect.

Levatorplasty

Using a para-anal incision the sagging levator muscle is exposed and elevated and its medial edge sutured to the anal canal close to the anorectal junction [46] (Fig. 8). The effectiveness of levator muscle repair in the treatment of levator dysfunction syndrome has been demonstrated in a previous study [46]. The beneficial effects of the operation could be attributed to many factors:

1. The correction of the levator plate position from the vertically oblique to the normal flat horizontal position affects anal canal and vesical neck opening on levator contraction.
2. Suturing of the levator plate medial border to the anorectal junction creates an artificial fibrous hiatal ligament instead of the subluxated functionless one; thus, on contraction, the levator plate becomes elevated and laterally retracted, and pulls on the hiatal ligament which opens the anorectal junction and vescial neck.
3. The transverse flat levator position opposes increased intra-abdominal pressure on straining more efficiently than does the vertically oblique one.

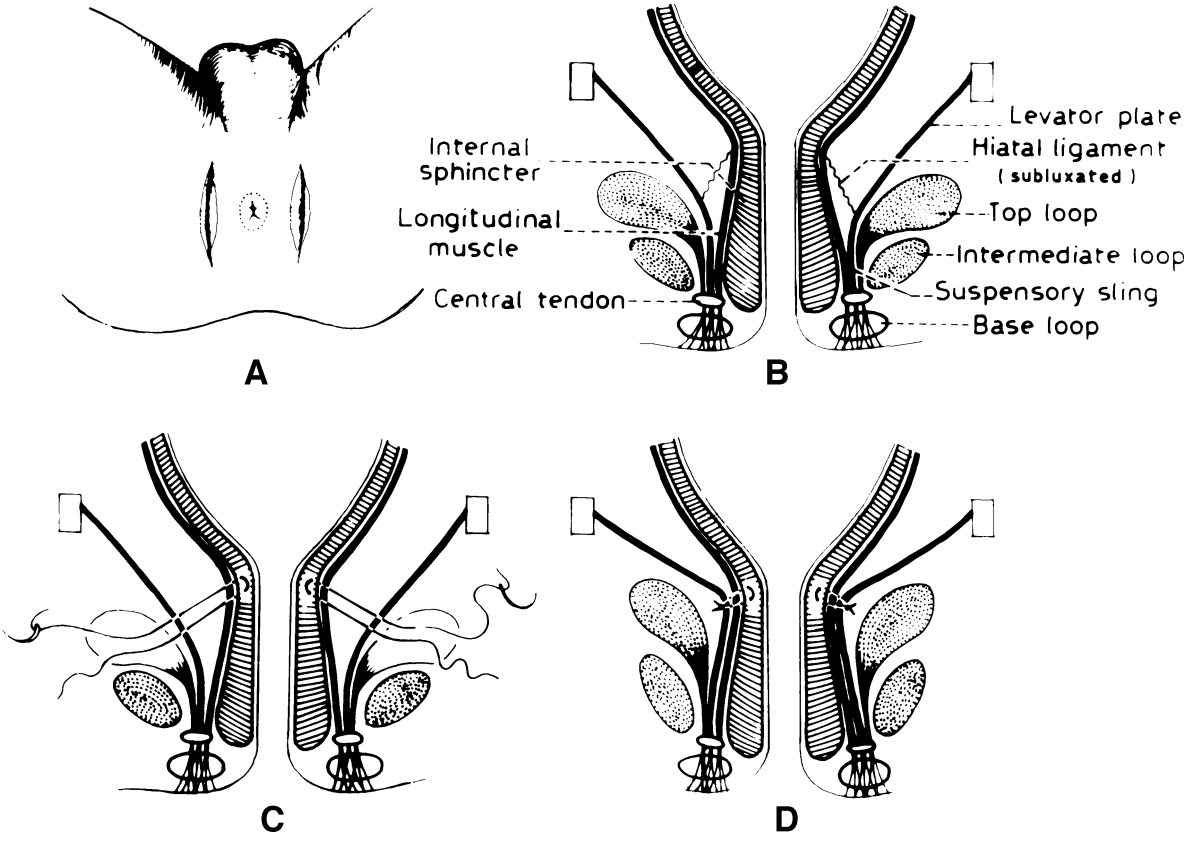

Fig. 8. Diagram illustrating the technique of levator muscle repair in levator dysfunction syndrome. (A) Para-anal incision; (B) findings at operation; the levator plate is lying vertically oblique, with subluxation of both the suspensory sling and hiatal ligament. Levator hiatus is widened; (C) levator plate is being sutured to upper anal canal inlet; (D) levator plate is repaired (from Shafik [46]).

4. Levator plate elevation not only places the anal canal and urethra below the pelvic diaphragm, thus protecting them from the direct intra-abdominal pressure effect, but also corrects the suspensory sling subluxation. It further achieves the narrowing of the abnormally wide levator hiatus, thereby helping to prevent intra-abdominal pressure leak to the anal canal or urethra, obstructing them.

Beside the levator dysfunction syndrome, two other syndromes have been described as affecting the levator ani muscle: the detrusor–levator [47] and the levator paradoxical [48]. These two syndromes differ from levator dysfunction in that the levator muscle is lying normally and not sagging, despite being functionally disordered.

Detrusor–Levator Dyssynergia Syndrome

This was found to be common in females, with an average age of 38.2 years. The main complaints were constipation and straining at stool. Physical examination was normal. On straining and rectal distension, the levator ani muscle relaxed instead of contracting [47]. Stool frequency and weight were normal. The anal canal pressure was normal at rest and on squeezing. It was reduced on straining, but only slightly, and not to the level occurring under normal conditions. The rectal pressure was normal. External anal sphincter EMG was normal at rest and on squeezing and straining. Levator EMG showed normal resting activity which did not change on voluntary squeezing. On straining and on balloon rectal distension the levator resting activity disappeared, instead of augmenting as occurs under normal conditions.

These results demonstrate that the levator ani muscle did not contract on rectal distension, but rather relaxed. Levator relaxation at defecation leads to failure of the anal canal to open, with a resulting fecal arrest at the anal canal orifice. The patient then has to strain in order to defecate. The elevated intra-abdominal pressure compresses the rectal detrusor, pushing its contents into the anal canal. The results of biofeedback were satisfactory in some patients [47].

Levator Paradoxical Syndrome

Normally, the levator ani muscle contracts on straining and is relaxed on voluntary squeezing [1,3]. In this syndrome, levator activity was absent or weak on straining and increased on squeezing [48]. It was common in females. All patients complained of excessive straining at stool. Stool frequency was normal. Anal canal pressure recorded abnormally high values on straining instead of diminished values as under normal conditions. Levator EMG resting activity was absent at rest and increased on voluntary squeezing. On straining it showed no or only weak activity, in contrast to normal controls, in whom levator EMG showed resting activity, no activity on squeezing and increased activity on straining. Direct levator stimulation showed normal activity in all patients. The external anal sphincter EMG activity was normal.

Results suggest that levator action is paradoxical. Normally, the levator muscle contracts to open the anal canal at defecation [1,3]. Levator paradoxical action results in failure of the anal canal to open on straining at stool. Fecal arrest at the anal canal initiates excessive straining and forcible detrusor contractions which eventually succeed in forcing fecal mass into the anal canal. The cause of levator paradoxical action is unknown. Three theories have been put forward: developmental, myopathic and neurologic [48]. Some patients improved with biofeedback treatment.

Levator Ani Muscle and the Pudendal Nerve

Levator ani subluxation and sagging occurs as previously mentioned in conditions of chronic straining, a prolonged second stage of labor or forceps application [1,3,46]. It pulls on the pudendal nerve, and in particular on the part lying in the pudendal canal, leading eventually to pudendal neuropathy, entrapment and pudendal canal syndrome [49,50]. In this regard a description of the surgical anatomy of the pudendal canal and nerve seems necessary.

Pudendal Canal

The anatomy of the pudendal canal (PC) was studied in 26 cadavers: 10 stillborn and 16 adults (mean age 48.2 years) [51]. Two approaches were used: gluteal and perineal. The PC was an obliquely lying tube with a mean length of 0.8 cm in the stillborn and 1.6 cm in the adult cadavers (Fig. 9). It started at a mean distance of 0.8 cm from the ischial spine in the stillborn and 1.6 cm in the adult cadavers, and ended at a mean distance of 0.7 cm and 2.6 cm, respectively, from the lower border of the symphysis pubis. The PC wall was formed by splitting of the obturator fascia and not by the lunate fascia. It contained the pudendal nerve and vessels embedded in loose areolar tissue. The three branches of the neurovascular bundle arose inside the canal in all but 3 cadavers. The wall of the PC consisted of collagen and elastic fibers, but that of the obturator fascia contained collagen only. The PC seems to be structurally adapted to serve certain functions.

Structural–Functional Adaptation of the PC

The anatomical structure of the PC seems to provide it with maximal functional performance [51]. Thus, the loose areolar tissue in which the neurovascular bundle is embedded allows for changes in vessel diameter in response to pelvic organ activities, especially during

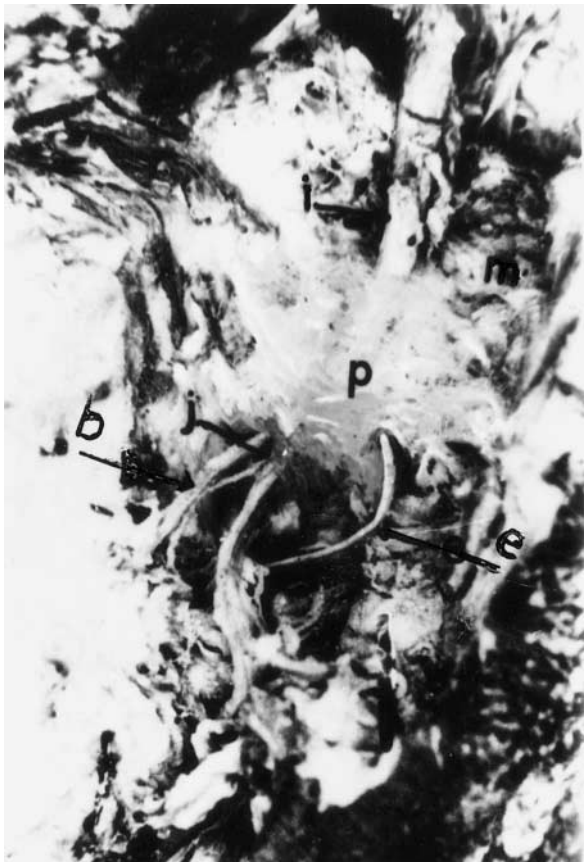

Fig. 9. Dissected cadaveric specimen showing the pudendal canal. The inferior rectal nerve (e) pierces the medial wall of the pudendal canal (p) and the perineal (j) and dorsal (b) nerves coming out of its anterioinferior end. i, pudendal nerve; m, sacrospinous ligament (from Shafik [51]).

sexual arousal and erection, without blood supply embarrassment. The functional adaptation of the PC is further provided by its histologic structure. The criss-cross plywood arrangement of the collagen fibers gives the PC wall a textile nature that allows the canal to change its shape in adaptation to pressure changes in the pudendal vessels. The PC can thus expand, giving the pudendal vessels space to engorge during sexual arousal and intercourse. Meanwhile, the elastic fibers included in the wall effect spontaneous return of the PC to its original size by their elastic recoil. With such a structure the PC is suggested to act as a 'pump' that assists venous return in the pudendal veins [51]. Furthermore, the elastic fibers seem to prevent collagen overstretch and PC subluxation as a result of its continuous distension by vessel engorgement.

'Pulley' Action of the PC

It appears that the PC also acts as a 'pulley' for the pudendal neurovascular bundle [51]. The latter, arising in the pelvis, passes through the PC on its journey to the ischiorectal fossa. It seems that the pulley action of the PC not only fixes the bundle during its travel to the pelvic floor muscles, but also prevents its being traumatized by the continuous movement of these muscles.

Pudendal Nerve

The pudendal nerve is an important motor and sensory nerve to the pelvic organs and perineum. It supplies the anal and urethral sphincters and pelvic floor muscles, as well as providing cutaneous and skeletal motor innervation of the penis and clitoris. Pudendal neuropathy or nerve injury leads to pathologic changes in these structures [49]. Pudendal nerve compression in the pudendal canal causes the pudendal canal syndrome (an entrapment syndrome) with a resulting incompetence of the anal and urethral sphincters [49,50]. Electrodes have been applied to the pudendal nerve or its roots or branches to treat anal and urethral sphincter insufficiency [52–56].

Knowledge of the anatomy of the pudendal nerve allows its precise localization for the performance of an accurate nerve block and stimulation to assess the functional integrity of the nerve and the pelvic floor musculature. Furthermore, successful use of implantable neuroprotheses [52–56] to treat fecal or urinary incontinence or erectile dysfunction would depend on proper screening studies of the pudendal nerve. The nerve can be used in biofeedback training and in nerve conduction studies or evoked potential recordings. It is also exposed in patients with pudendal canal syndrome, where the canal is decompressed by slitting open its roof [49,50,57–60].

The pudendal nerve arises from the anterior rami of S2–4, and is formed of three roots and two cords [61,62] (Fig. 10). It may also take a contribution from S1 and S5. The first root continues as the upper cord while the second and third roots unite to form the lower cord. The two cords fuse to form the pudendal nerve. In neurostimulation of the pudendal nerve, identification of the cords helps in applying the neuroprosthesis to the appropriate cords. The upper cord is longer and thinner than the lower.

Role of Levator Ani in Idiopathic Pelvic Pain (Vulvodynia, Proctalgia, Scrotalgia and Prostatodynia) and the Pudendal Canal Syndrome

Levator dysfunction might occur as a result of straining at stool or during a prolonged second stage of labor or forceps application, with a resulting pudendal canal syndrome. The mechanism is described later. Patients with prostatodynia, scrotalgia, vulvodynia and proctalgia have a common clinical presentation and investigative findings of pudendal canal syndrome [50,63–65]. These comprise the neurogenic nature of the pain, the motor and sensory changes being localized to pudendal nerve distribution, and the increased pudendal nerve terminal

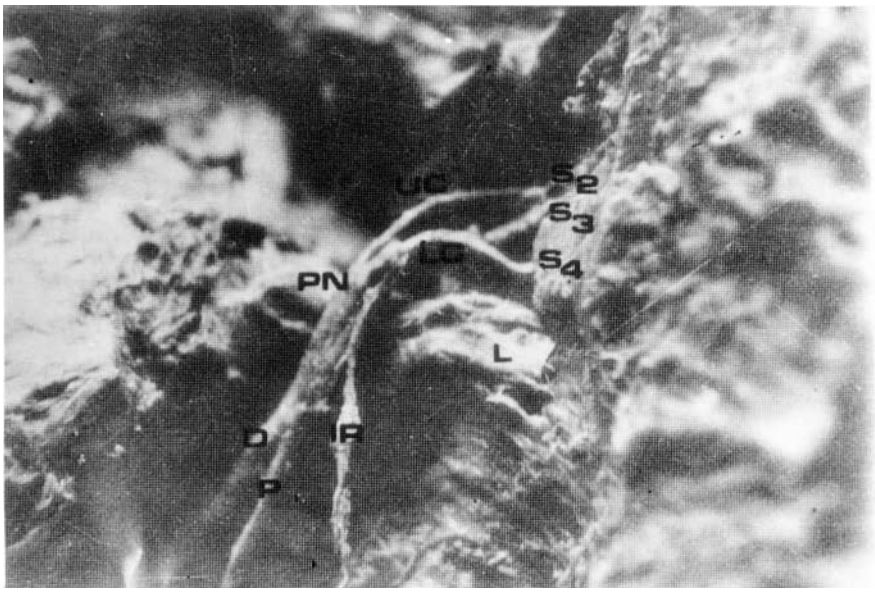

Fig. 10. Photograph of an adult cadaver showing the pudendal nerve. S2 = sacral root forming upper cord. S3, S4 = the two sacral roots unite to form the lower cord. The two cords unite to form the pudendal nerve (PN), which crosses the sacrospinous ligament (L) and gives rise to three branches: the inferior rectal (IR), the perineal (P) and the dorsal nerve of the penis (D) (from Shafik et al. [61]).

motor latency (PNTML), which indicates the involvement of the distal part of the nerve. The diagnosis of pudendal canal syndrome is further confirmed by the temporary disappearance of the pelvic pain following bilateral pudendal nerve block.

Mechanism of Pudendal Nerve Entrapment

The pudendal nerve is a mixed nerve. Arising from the sacral plexus, it leaves the pelvic cavity through the greater sciatic foramen and passes over the sacrospinous ligament close to the ischial spine to enter the perineum through the lesser sciatic foramen [61,62]. It then passes forward in the pudendal canal. Its branches comprise the inferior rectal, perineal and dorsal nerves of the penis or clitoris. The inferior rectal nerve supplies the external anal sphincter, the levator ani, the mucous membrane of the lower half of the anal canal and the perianal skin. The perineal nerve supplies the striated urethral sphincter and the skin on the posterior surface of the scrotum. The dorsal nerve of the penis or clitoris is distributed to the penis or clitoris.

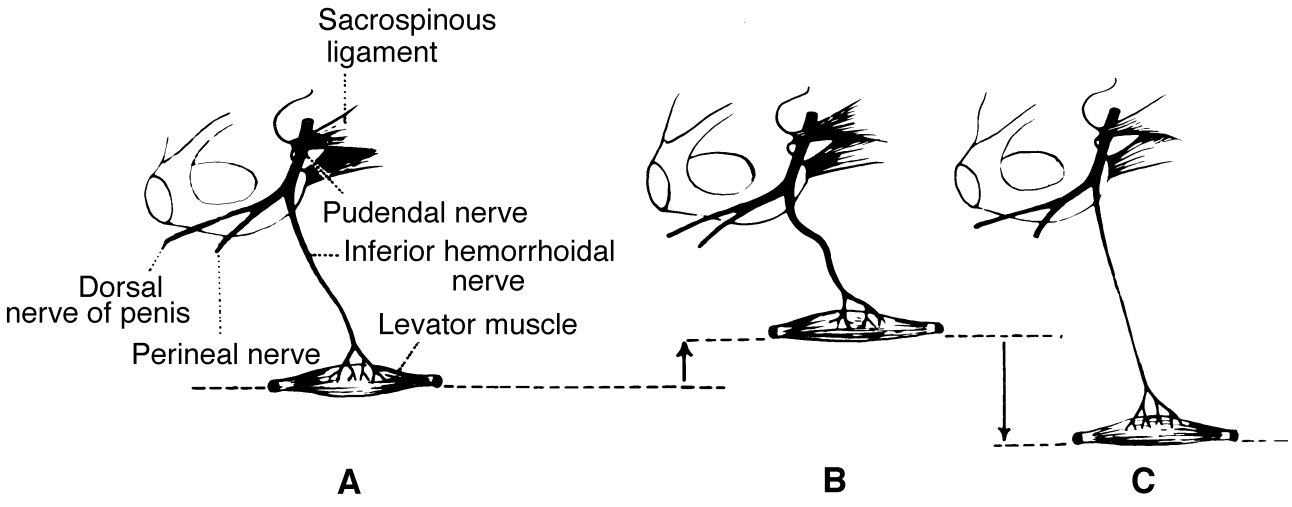

Fig. 11. Mechanism of pudendal nerve stretch. (A) At rest: levator muscle is relaxed; (B) on contraction at evacuation: levator muscle is elevated; (C) difficult deliveries or chronic straining at evacuation cause levator subluxation and sagging, with a resulting pudendal nerve stretch (from Shafik [49]).

As mentioned above, an increase in intra-abdominal pressure beyond the normal physiologic limits, as occurs in chronic straining at stool or urination, during a prolonged second stage of labor or forceps application, would eventually result in subluxation and sagging of the levator ani. The latter lies at a lower level than normal and consequently pulls on the pudendal nerve (Fig. 11). The stretching affects the distal portion of the nerve which extends from the ischial spine to the muscles. The winding of the nerve around the ischial spine fixes the nerve at this point and exposes only the distal portion to stretch [46,49]. Continuous nerve stretch leads to neurapraxia or axonotmesis. Being entrapped in the pudendal canal and subjected to continuous stretch, the pudendal nerve may undergo edema with subsequent compression inside the canal, leading to nerve ischemia which would add to the damage. Eventually 'entrapment neuropathy' occurs.

It seems that pudendal neuropathy affects the branches of the pudendal nerve to different degrees. The main brunt of neuropathy may involve one of the three branches and, to a lesser extent, the other two. This is evident from the sensory and motor affection. The patients present clinically with fecal or stress urinary incontinence [50,60], and pelvic or perineal pain which takes the form of scrotalgia, proctalgia, prostatodynia or vulvodynia [49,63–65]. These presentations occur singularly or in combination. Pudendal canal fasciotomy decompresses the nerve and sets it free within the ischiorectal fossa [49,50,57,59,60,63–65]; it thus escapes being stretched by the contracting and sagging levator ani.

Pudendal nerve decompression was effective in the treatment of the pudendal canal syndrome [49,50,58–60,63–65], the scrotal, anal, vulvar and perineal pain disappearing in most cases. There was improvement in fecal and urinary incontinence and in the sensory and motor affection of the pudendal nerve, as is evident from improvement of perineal sensation, anal reflex, EMG activity of the striated urethral and anal sphincters, and PNTML. Although the scrotal and perineal hypoesthesia, as well as the anal reflex, did not improve in all patients alike, the initially increased PNTML showed a significant reduction. Failures might be due to faulty diagnosis, incomplete pudendal canal decompression or irreversible pudendal nerve damage.

Role of Levator Ani in Stress Urinary Incontinence

Vesical and Urethral Descent and Levator Muscle Subluxation

The cause of the vesical and urethral descent with loss of the urethrovesical angle that occurs in stress urinary incontinence (SUI) is controversial [4–7,66–74]. The levator ani is attached to the urethrovesical angle by the hiatal ligament, the anterior part of which is the pubovesical ligament [1,3]. On straining and increased intra-abdominal pressure there is reflex levator muscle contraction induced by the straining–levator reflex [17]. The cone-shaped levator plate becomes elevated and laterally retracted, and is transformed from a cone to a plate shape (Fig. 12). It pulls on the hiatal ligament, which pulls upward and opens the urethrovesical angle. Levator contraction thus not only opens but also slings up the urethrovesical angle during the increased intra-abdominal pressure, thereby preventing it from sagging [75].

With chronic straining as occurs in chronic idiopathic constipation, repeated and difficult deliveries or with acute straining in the second stage of labor, which may necessitate the use of forceps, the levator plate, and especially its fibrous anococcygeal raphe, are overstretched, leading to levator subluxation and sagging [1,3]. The hiatal ligament also subluxates. The urinary bladder and the urethrovesical angle, lacking levator support, sag under the effect of straining, with a resulting obliteration of the angle [75].

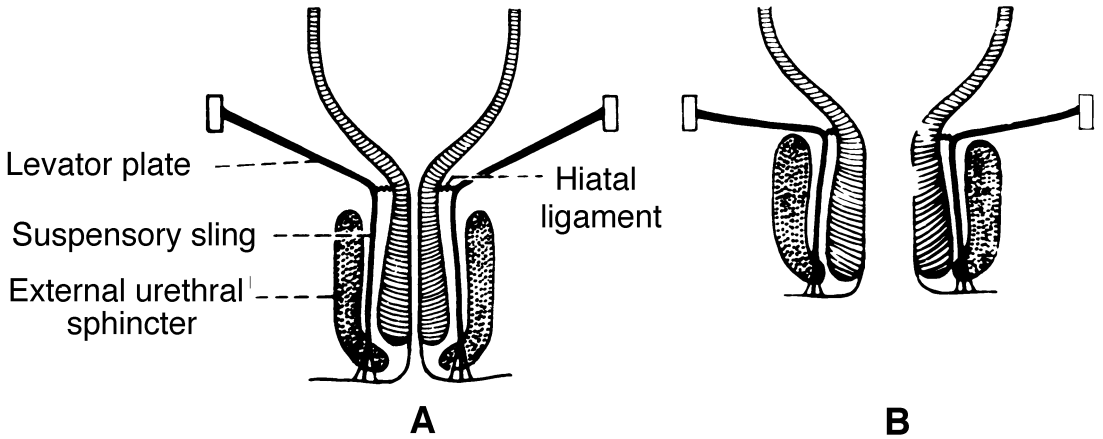

Fig. 12. The levator ani action. (A) At rest; (B) on contraction: levator plate becomes elevated and laterally retracted, pulling on the hiatal ligament which pulls open the urethrovesical junction (from Shafik [75]).

Stress Urinary Incontinence and Pudendal Canal Syndrome

The cause of diminished striated urethral sphincter activity and prolongation of both the latency of the straining–urethral reflex [76] and PNTML in SUI patients is not known. However, evidence is in favor of a neurogenic etiology, possibly pudendal nerve compression in the pudendal canal [75]. Evidence includes: (a) the weak striated urethral sphincter; (b) the prolonged latency of the straining–urethral reflex; (c) prolonged PNTML; (d) concomitant idiopathic fecal incontinence; and (e) the high incidence of SUI in multiparous females with a history of difficult deliveries. In a previous study [75], one-third of SUI patients also had fecal incontinence. Pudendal nerve entrapment may affect both striated urethral and anal sphincters, as it supplies both muscles. However, these patients complained mainly of SUI being more frequent and distressing than the partial fecal incontinence for flatus and/or fluid stools.

Twelve women with SUI due to pudendal canal syndrome (PCS) were treated by pudendal canal decompression [60]. The investigations comprised determination of the EMG activity of the striated urethral sphincter as well as the straining–urethral reflex latency and PNTML. We defined three scores. In score 1, the patient became dry (6 patients); in score 2, the patient improved (5 patients) and in score 3 no change was noted (1 patient). In scores 1 and 2 there was improvement in labia majora sensation and EMG activity of the striated urethral sphincter, as well as a decrease in the straining–urethral reflex latency and PNTML.

Pudendal canal decompression seems to correct a basic cause of SUI, in contrast to other procedures which deal with the effect rather than the cause of the condition. The technique is simple and without complications, and can be performed on an outpatient basis [60].

Role of Levator Ani in Fecal Incontinence

Levator ani subluxation and sagging, with resulting pudendal neuropathy, may present with fecal incontinence [50]. Investigations demonstrated an effect on the sensory and motor elements of the pudendal nerve, manifesting as perianal hypo- or anesthesia, diminished EMG activity of the external anal sphincter and levator ani muscle, and prolonged PNTML; anal pressure was also diminished. In a previous study of 11 patients with fecal incontinence due to pudendal canal syndrome, 7 had additional urinary stress incontinence [50]. Pudendal canal decompression resulted in cure of the fecal and urinary incontinence in 8 of the 11; follow up was 7–10 years.

The cause of the fecal incontinence that accompanies complete rectal prolapse in 43%–66% of the cases is not known [77,78]. Many theories have been proposed but none was entirely satisfactory [79,80]. Recently, we investigated the cause of fecal incontinence in a group of patients with complete rectal prolapse [58]. Manifestations of pudendal neuropathy were demonstrated, due probably to pudendal nerve entrapment in the pudendal canal with a resulting pudendal canal syndrome. Pudendal canal decompression improved fecal continence in these patients [59].

Role of Levator Ani in Erectile Dysfunction

An earlier study demonstrated that levator dysfunction and pudendal canal syndrome could present with erectile dysfunction [81]. The main brunt of pudendal neuropathy involved the penile branch of the pudendal nerve. The patients presented with the manifestations of pudendal neuropathy and penile, perineal and scrotal hypo- or anesthesia. There were increased bulbocavernosus and PNTML latencies. Pudendal canal decompression improved erectile dysfunction in 6 of 7 patients [81].

Acknowledgement. Waltraut Reichelt and Margot Yehia assisted in preparing this manuscript.

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