

Current Uses of Surgery in the Treatment of Genital Pain

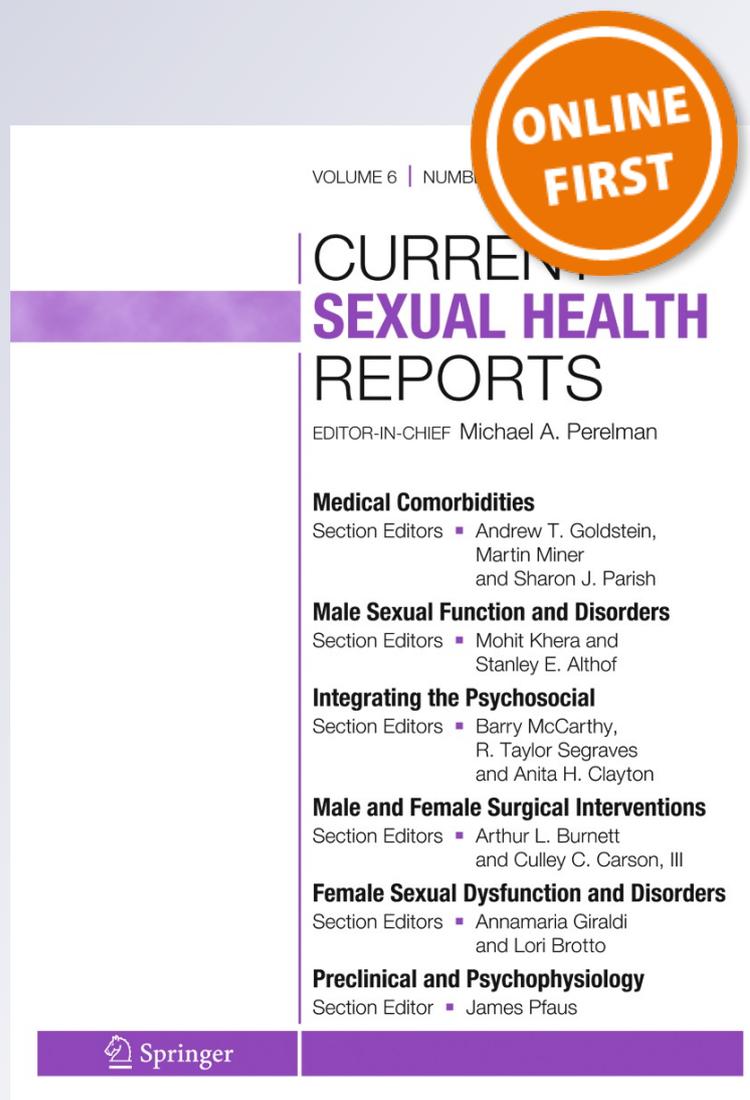
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Current Uses of Surgery in the Treatment of Genital Pain

Michelle King · Rachel Rubin · Andrew T. Goldstein

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Abstract Genital pain frequently causes sexual dysfunction and psychological distress that can impact every aspect of a woman's life. When conservative medical treatments do not adequately treat the genital pain, surgical procedures have the potential to significantly decrease the pain. The following review examines surgical procedures currently being used to treat pudendal neuralgia, scarring from lichen sclerosus, and provoked vestibulodynia. In addition, a diagnostic algorithm is included that can be used to identify specific causes of genital pain and to determining when surgery is an appropriate treatment option.

Keywords Pudendal neuromodulation · Pudendal neuralgia · Lysis of vulvar adhesions · Vestibulodynia · Vulvar vestibulectomy · Perineoplasty · Genital surgery · Dyspareunia

Introduction

Millions of women around the world are afflicted with sexual pain disorders that interfere with their daily lives and frequently result in sexual dysfunction that has the ability to cause significant psychological, physiological, and marital distress. The results of studies examining the prevalence of dyspareunia, or pain that occurs

during sexual intercourse, vary greatly. However, it has been suggested that as many as 17–19 % of women have a lifetime prevalence of dyspareunia [1]. Due to the fact that the mechanisms behind these sexual pain disorders have only recently been explored through evidence-based research, few medical providers have adequate training on the evaluation and management of dyspareunia [2]. Women experiencing sexual pain disorders frequently visit multiple health care providers and undergo multiple treatments prior to learning the true cause of their symptoms and identifying appropriate treatment [3]. As the different etiologies of dyspareunia are further clarified, diagnostic and treatment algorithms are being developed.

This review will examine a diagnostic algorithm for the evaluation of vulvodynia and vestibulodynia and the current surgical treatments being utilized in the treatment of genital pain of multiple etiologies, including pudendal neuralgia, complications of lichen sclerosus, and neuroproliferative vestibulodynia. The vulvar vestibule is defined as the tissue between Hart's line and the hymen. Hart's line is the lateral borders of the vestibule and marks the transition between the non-keratinized squamous endothelium of the vestibule to the keratinized epithelium of the labia minora. The vulvar vestibule extends from the frenulum of the clitoris anteriorly to the posterior fourchette. Vulvodynia is defined as pain that of the anatomical structures of the vulva, including the labia majora, labia minora, clitoris, prepuce, vestibule, mons pubis, and urethra. Vestibulodynia is pain specifically localized to the vestibule of the vulva.

The aim of this review is to provide health care professionals and patients diagnosed with sexual pain disorders with a general overview of the surgical approaches utilized in these procedures and examples of their success rates as published in the literature.

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Diagnostic Algorithm

One algorithm for the evaluation of vulvar pain was developed at The Center for Vulvovaginal Disorders in Washington, DC, USA (Fig. 1). This diagnostic algorithm has been derived from research conducted in the last 15 years that has helped to identify the underlying pathophysiology of different causes of vulvovaginal pain (vulvodynia). Specifically, this research has help to recognize myofascial, hormonal, neuroproliferative, inflammatory, and neuropathic causes of vulvar pain [4–8]. This algorithm helps practitioners to differentiate between different causes of vulvodynia/provoked vestibulodynia and, in so doing, allows them to select appropriate treatments. This has helped to improve the success of conservative treatments and to limit unnecessary surgical intervention. For example, a woman who is found to have the combination of provoked vestibulodynia confined to only the posterior vestibule and hypertonic pelvic floor muscles will be offered treatments to correct the hypertonus. The patient will be offered treatments such as pelvic floor physical therapy, intravaginal diazepam suppositories, and levator ani botulinum toxin injections, but she would not be offered a surgical intervention such as vulvar vestibulectomy because she does not have an intrinsic pathology within the vestibular endothelium [9–11]. Conversely, a woman who has the combination of provoked vestibulodynia throughout the entire vulvar vestibule and umbilical hypersensitivity (which is evidence of a congenital neuronal hyperplasia within the tissue derived from the urogenital sinus) would be diagnosed with congenital neuroproliferative provoked vestibulodynia (CNPVD) [12]. This patient would be offered surgical removal of the tissue

of the vestibule, a vulvar vestibulectomy with vaginal advancement, to remove the affected abnormal endoderm.

Surgery for Genital Pain Caused by Pudendal Neuralgia

The term pudendal neuralgia refers to a neuropathic pain disorder involving the pudendal nerve dermatome [13]. Originating from the S2–4 sacral nerve roots, the pudendal nerve carries sensory, motor, and autonomic fibers in both efferent and afferent pathways [14]. The nerve travels inferiorly and posteriorly in a fixed space between the sacrospinous and sacrotuberous ligaments. It then exits the pelvis through the obturatorinternus muscle membrane termed Alcock’s canal [15]. There are classically three terminal branches described in the literature including the dorsal nerve of the clitoris/penis, the perineal nerve, and the inferior rectal nerve [15, 16]. Damage or entrapment of the nerve causes refractory pain in the pudendal dermatome that is worsened by sitting and often progressive throughout the day. The neuropathic pain is usually described as burning, tingling, and numbing and can affect the clitoris, vulva, vagina, or rectum in a unilateral or bilateral fashion [17].

Patients who fail initial treatment with medications (such as pregabalin and gabapentin) and pelvic floor physical therapy can be offered a number of procedural and surgical options.

Initial therapy routinely begins with pudendal nerve blocks. These injections are classically used for both diagnosis and therapeutic relief. Various techniques have been described in the literature including transvaginal [17], transperineal [18], and transgluteal. The transgluteal approach is often used

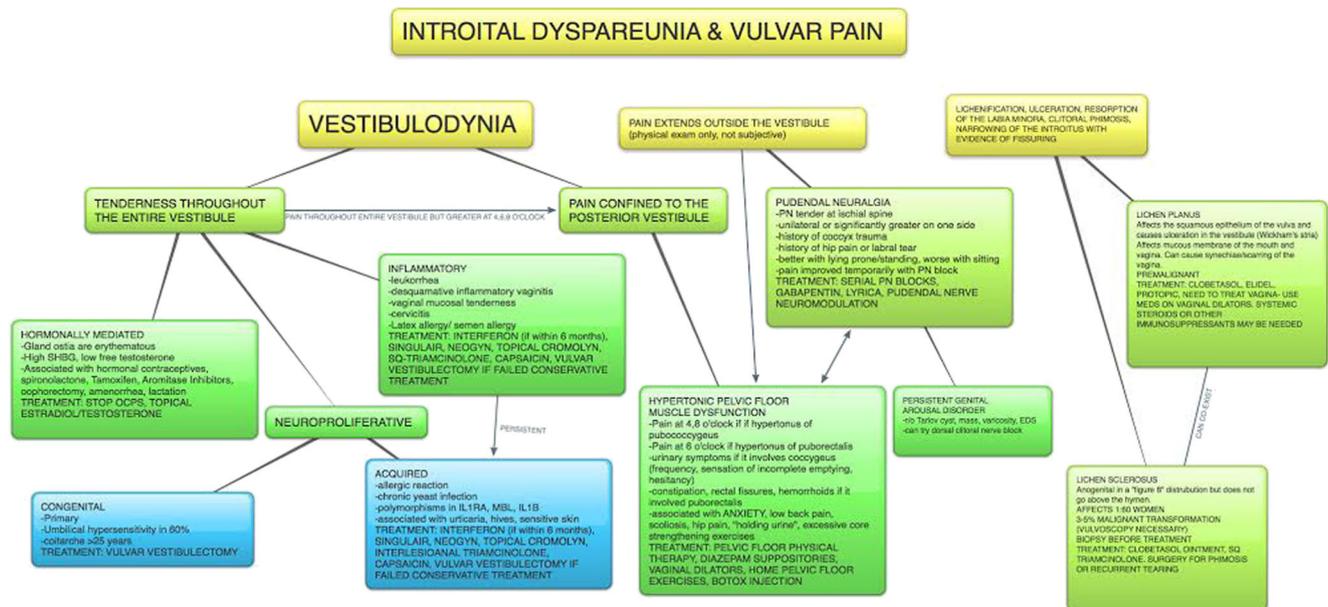


Fig. 1 Algorithm for diagnosis and management of vulvar pain

because it allows for a blockade at the ischial spine and directly in Alcock's canal [8]. Various imaging techniques are used to ensure accuracy of the injection. Hibner describes his technique of injecting a series of three unilateral or bilateral CT-guided injections into Alcock's canal done 3 weeks apart. Injections consist of 10 cc of 0.5 % bupivacaine and triamcinolone [8].

Robert and colleagues conducted a randomized control trial that showed a reduction in pain in two thirds of patients with two to three blocks each over a period of 6 months [19].

Patients who do not find sustained relief despite serial nerve blocks can be offered surgical decompression of the nerve. There are four described operations, including transchiorectal [20], perianal [15], laparoscopic, and transgluteal [21, 22]. The transgluteal approach has gained favor because it allows for better visualization of the pudendal nerve. The procedure takes place with the patient in a jack-knife prone position. The incision is made in the gluteal region just over the sacrotuberous ligament. Once the gluteus muscle fibers are separated, the sacrotuberous ligament is divided at its narrowest point. The nerve is then identified either visually or with the aid of a NIMS monitor (Nerve Integrity Monitoring System; Medtronic, Minneapolis, MN). The pudendal nerve is then decompressed along its entire length, from the piriformis muscle to Alcock's canal. Some centers describe encasing the nerve in nerve protective sheath and using platelet-rich plasma to promote healing and decrease scar formation [22, 23].

One randomized controlled trial looked at 32 patients separated into surgery versus conservative treatments. Fifty percent of the surgery group reported improvement in pain at 3 months versus 6.2 % of the non-surgery group ($p=0.0155$). At 12 months, 71.4 % of the surgery group were improved as compared to only 13.3 % of the non-surgery group ($p=0.0025$) [19].

Neuromodulation has shown success in the treatment of overactive bladder and certain chronic pain syndromes. More recently, it is being studied as a tool to treat pudendal neuralgia [24]. It is hypothesized to work by introducing exogenous electrical current to the affected region, which changes the native electrical signals of the nervous system, thereby changing the patient's perception of pain. Peters et al. describe implantation of a standard quadripolar timed lead adjacent to the pudendal nerve placed under fluoroscopic guidance while using needle electromyography (EMG) at the external anal sphincter to monitor the activity of the pudendal nerve. Once the lead is confirmed to be in place, intraoperative programming is performed to determine optimal device settings and the leads are connected to an external stimulator. The patient will feel tapping or vibration in the distribution of the pudendal nerve. If there is at least a 50 % reduction in pain within the 2 weeks following placement of the lead, then a permanent pulse generator is implanted. Patients without response to the

system have their leads removed [18]. While initial outcomes of neurostimulation used to treat pudendal neuropathy and other chronic pelvic pain conditions are promising, small sample sizes and lack of randomized control trials make it difficult to make broad conclusions [24, 25].

Surgery for Genital Pain Caused by Complications of Vulvar Lichen Sclerosus

Lichen sclerosus is a chronic inflammatory disease of the anogenitalepithelium that causes symptoms of vulvar itching, irritation, and burning [26, 27]. It has been reported that vulvar lichen sclerosus affects 1 in 660 British women and approximately 1 in 70 women in a general gynecology private practice in the USA [28, 29]. Left untreated, vulvar lichen sclerosus can lead to significant vulvar scarring, including labial resorption, clitoral phimosis, introital stenosis, and recurrent tearing of the anterior and posterior aspects of the introitus (vulvar granuloma fissuratum) [26, 27].

Clitoral phimosis, scarring of the clitoral prepuce and/or labia majora such that the glans clitoridis is partially or completely "buried," is a complication of vulvar lichen sclerosus that can cause significant morbidities including loss of clitoral sensitivity and anorgasmia [27, 30]. In addition, clitoral phimosis may cause significant emotional trauma due to the distortion of the vulvar architecture and perceived diminution of sexuality and femininity. In addition, it can become necessary to surgically correct the clitoral phimosis because in some women, smegma accumulates in the space between the prepuce and clitoris (a smegmatic pseudocyst), which may become inflamed or infected (a smegmatic pseudocyst abscess) [31].

One approach to clitoral phimosis repair described by Goldstein and Burrows includes insertion of a lacrimal duct probe between the clitoris and prepuce, which is used to bluntly lyse any adhesions [27]. A small dorsal incision may then be made in the prepuce using Iris scissors to allow lysis of additional adhesions with the lacrimal probe. The edges of the prepuce are then trimmed to prevent recurrent adhesions and then silver nitrate can be used to obtain hemostasis. Alternatively, the prepuce may be oversewn to obtain hemostasis and to prevent recurrent adhesions [32]. Postoperatively, patients apply clobetasol 0.05 % ointment daily to the surgical site to prevent Koebnerization, and patients decrease the frequency of clobetasol application to twice weekly after the surgical site heals [33•]. Kroft and Shier reported a case series of 23 women with phimosis in which a CO₂ laser was used to lyse the vulvar adhesions and to obtain hemostasis [34•].

In a study examining the surgical outcomes of eight patients who had surgery to correct clitoral phimosis seven of the eight (88 %) reported that they were "very satisfied" with the results of their surgery. The additional patient reported that she

was “satisfied,” and all eight patients reported that they would recommend surgery to other women with similar symptoms. Of the four women who reported decreased clitoral sensitivity prior to surgery, all experienced increased clitoral sensitivity and orgasm following the procedure [27].

An additional complication that many women with vulvar lichen sclerosus develop is introital stenosis [26]. For many women, narrowing of the introitus causes significant dyspareunia, sexual dysfunction, and recurrent tearing of the vestibular endothelium and perineal skin-vulvar granuloma fissuratum. Women with introital stenosis should be initially treated with conservative treatment consisting of topical ultrapotent corticosteroids and manual dilation with graduated vaginal dilators. If conservative treatment fails, it may be necessary to perform a surgical procedure known as a perineoplasty to correct the narrowing of the introitus. The scarred endothelium of the posterior fourchette and the scarred epithelium of the perineum are excised (Fig. 2a) and a vaginal advancement flap is then used to close the defect (Fig. 2b) [31, 35].

Rouzier and colleagues reported the functional outcomes following perineoplasty for introital stenosis in a cohort of 64 women. Of the 50 women who completed a postprocedure questionnaire and complied with follow-up, 92 % reported relief of introital dyspareunia after the perineoplasty, and 86 % reported improvement in the quality of sexual intercourse [31, 36]. Only 1 of 64 had recurrence of introital dyspareunia. Average healing time for the procedure was 6 weeks, and complications of the perineoplasty included dehiscence of the vaginal advancement flap in 4 of 64 patients [31].

Surgery for Genital Pain Caused by Neuroproliferative Vestibulodynia

Neuroproliferative vestibulodynia is a condition in which women have an increase in the density of C-afferent

nociceptors in the epithelium of the vestibular mucosa, as identified by Bohm-Starke and colleagues, who used PGP 9.5 immunohistochemistry to demonstrate proliferation of intraepithelial nerve endings in the vulvar vestibule [37]. Bornstein and colleagues confirmed these results and showed that patients with neuroproliferative vestibulodynia (previously known as vulvar vestibulitis syndrome) had up to ten times the density of nerve fibers as compared to normal controls [34]. Due to the increased density of C-afferent nociceptors, women with neuroproliferative vestibulodynia experience an allodynia and hyperpathic sensation at the vulvar vestibule frequently described as “cutting, rawness, or burning.” As described by Friedrich in 1987, there are three main criteria to identify neuroproliferative vestibulodynia (vulvar vestibulitis): exquisite tenderness when the vestibule is palpated with a cotton swab; vestibular erythema (often at the ostia of the major and minor glands); and severe pain with attempted vaginal entry of a penis, speculum, tampon, etc. [38].

In women who fail conservative treatments such as oral montelukast, topical capsaicin, or topical gabapentin cream, vulvar vestibulectomy with vaginal advancement can be performed to remove the abnormal vestibular mucosa [39–41]. In 1983, Woodruff and Parmley were the first authors to describe vulvar vestibulectomy [42]. Their procedure consisted of the excision of a semicircular segment of perineal skin, the mucosa of the posterior vulvar vestibule, and the posterior hymeneal ring. Three centimeters of the vaginal mucosa was then undermined and approximated to the perineum.

Several variations of the procedure have been described to help decrease complications such as dehiscence of the vaginal advancement flap as well as to improve operative success [30]. A complete vulvar vestibulectomy with vaginal includes the excision of the mucosa of the entire vulvar vestibule including the mucosa adjacent to the urethra, while a modified vestibulectomy limits excision of the mucosa to the posterior vestibule [43]. Tommola and colleagues did a systematic review of the success rates and complication rates of the several variations of this procedure and concluded “there is no straightforward recommendation of the best technique. Certainly the surgeon’s experience plays a critical role. As with all surgeries, the procedure should be extensive enough to remove all painful areas but also to avoid unnecessary risks” [43].

The procedure for a complete vulvar vestibulectomy with vaginal advancement is as follows [44]: The labia minora are grasped using Allis clamps and are separated laterally to reveal the entire vulvar vestibule. The vulvar vestibule is outlined using a skin marking pen by making parallel lines on both sides of the urethra and carrying these lines superiorly to Hart’s line then inferiorly following Hart lines meeting approximately 0.7 cm on the perineum. Marcaine 0.05 % with epinephrine is used to infiltrate the vulvar vestibular mucosa

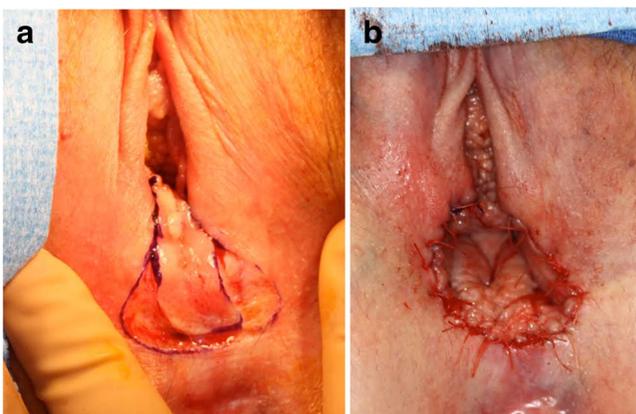


Fig. 2 a, b Excision of vestibular endothelium and perineal epithelium

for intraoperative hemostasis and postoperative pain control. A scalpel is used to excise the entire vulvar vestibular mucosa approximately 3 mm deep and 5 mm past the hymenal ring, thus removing the entire hymenal ring. Some physicians advocate excision of the Bartholin's glands at this point, to prevent postoperative Bartholin cyst formation [39]. Other authors suggest that the risk of postoperative Bartholin cyst formation is low and removal of the Bartholin gland increases operative time, and intra-operative blood loss, and increases the risk of pudendal nerve injury [45]. The vaginal mucosa is then grasped with two wide Allis clamps, and 2 cm of vaginal mucosa are gently dissected off the recto-vaginal fascia to create a vaginal advancement flap. This flap will be used to cover the defect in the posterior vestibule. After the vaginal mucosa has been separated from the recto-vaginal fascia, it is anchored in an advanced position using two rows of mattress stitches of 3-0 Vicryl (Ethicon, Somerville, NJ). These mattress stitches go through the vaginal mucosa and are then "back-handed" through the recto-vaginal fascia, to go back through the vaginal mucosa. These mattress stitches are placed in an anterior-posterior direction to avoid narrowing the diameter of the introitus, and when tying these stitches, an assistant applies gentle downward traction on the advancement flap to ensure that the mucosa will be secured in an advanced position. The mattress sutures ensure that there will not be significant tension on the suture line when the advancement flap is approximated to the perineum, thereby decreasing the risk of dehiscence of the advancement flap. In addition, these mattress sutures prevent the advancement flap from curling and prevent hematoma formation under the advancement flap (Fig. 3). The defects in the anterior vestibule are closed with running interlocked 4-0 Vicryl suture. Meticulous



Fig. 3 Mattress sutures and vaginal advancement flap

attention to detail is necessary as to ensure that the urethral meatus is not injured when closing these anterior defects to prevent a postoperative hematoma that can lead to urinary retention. The advancement flap is then approximated to the medial aspect of the labia minora and to the perineum using approximately 20 interrupted stitches of 4-0 Vicryl suture to complete the procedure. The urethra should be catheterized to demonstrate that it was not compromised when closing defects in the anterior vestibule and a digital rectal examination should be performed to confirm that the mattress stitches did not go through the rectum.

In the immediate postoperative period, liberal use of ice packs prevents swelling and helps with pain. Sitz baths starting several days after the surgery may help prevent infection. Physical activity should be limited for the first 4–6 weeks to allow the surgical site to heal and to help prevent wound dehiscence. Pyrex or plastic vaginal dilators are often used to stretch the introitus after the surgical site has completely healed. Treatment by a qualified woman's health physical therapist may then be utilized to address postoperative hypertonus of the levator ani muscles.

Patients considering surgery need to be informed that complications of vestibulectomy and perineoplasty do occur, though they are infrequent. Specifically, complications include bleeding, infection, increased pain, hematoma, wound dehiscence, scar tissue formation, and Bartholin cyst formation [46]. The risk of these complications can be reduced if appropriate surgical techniques are utilized. Since the risks of complication are low, they should not be overemphasized when counseling patients about surgical treatment for neuroproliferative vestibulodynia [37, 47].

As of 2010, there were 33 studies that addressed improvement in dyspareunia as a measure of surgical success for patients who had a partial or complete vulvar vestibulectomy [43]. Seventeen out of 33 of those studies based improvement in dyspareunia solely on a patients' self-report on improvement of dyspareunia, alleviation of symptoms, or reduction in pain. Overall, these retrospective studies reveal that operative treatment provided significant long-term relief in 78.5 % of patients, some relief in 88.8 % of patients, and no relief in 12.2 %. In nine studies that reported improvement in sexual function as a measure of success, all nine studies reported significant improvement in sexual function following vestibulectomy. Another recent study by Swanson and colleagues reported an 84.1 % moderate-substantial improvement in dyspareunia following a modified vestibulectomy [48]. These results suggest that vulvar vestibulectomy may be an appropriate first-line treatment option rather than a "last resort." However, in a recent study by Tommlola and colleagues, 66 women diagnosed with severe vulvar vestibulitis were followed long term from the time of diagnosis until after successful treatment. Thirty-nine women did not respond to conservative treatment and

were given posterior vestibulectomies, while 27 were managed conservatively with medication [49]. Overall, dyspareunia decreased significantly in both groups, with 66.7 % of women in the surgery group reporting improvement in dyspareunia, and 78.1 % in the conservative treatment group reporting improvement in dyspareunia. Long-term sexual well-being did not differ between the two groups, and 89 % of women in both groups were satisfied with their overall treatment. This suggests that conservative treatment may be preferred in patients who exhibit some improvement with conservative treatments in order to avoid the inconveniences and long recovery period of the vestibulectomy.

These studies are difficult to compare [38]. Techniques and terminology used to describe the various procedures varied significantly. Different authors referred to widely different surgeries characterized by the same name. Often, there are changes to the techniques employed even within the same series of patients. The outcome criteria for “surgical success” are often poorly defined and rarely are standard procedures employed to assess success. The evaluation of success is always non-blind, rendering it highly subjective. Patient selection criteria are usually not mentioned or are variable within a given series. Diagnostic algorithms such as the one described earlier in this chapter to differentiate between patients with different types of vestibulodynia have only been utilized in one study [50]. Most studies did not distinguish between various forms of vestibulodynia (primary or secondary, provoked pain only or spontaneous pain). There is a great degree of variability in the length of follow-up even within a given series, and follow-up is not always long term. Therefore, determining the rate of recurrence of vestibulodynia after surgery is very difficult to assess.

Conclusion

Evidence-based research has allowed us to identify and develop surgical techniques that have shown promise in the treatment of genital pain and vulvar disorders. However, limitations to these studies include small sample sizes, lack of randomized-controlled trials, and lack of standardized terminology and diagnostic procedures.

Compliance with Ethics Guidelines

Conflict of Interest Michelle King, Rachel Rubin, and Andrew T. Goldstein declare no conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

1. Moynihan R. The making of a disease: female sexual dysfunction. *BMJ*. 2003;326(7379):45–7.
2. Rosen R, Kountz D, Post-Zwicker T, et al. Sexual communication skills in residency training: the Robert Wood Johnson Model. *J Sex Med*. 2006;3(1):27–46.
3. Harlow BL, Stewart EG. A population-based assessment of chronic unexplained vulvar pain: have we underestimated the prevalence of vulvodynia? *J Am Med Women's Assoc*. 2003;58(2):82–8.
4. Morin M, Bergeron S, Khalife S, Mayrand MH, Binik YM. Morphometry of the pelvic floor muscles in women with and without provoked vestibulodynia using 4D ultrasound. *J Sex Med*. 2014;11:776–85.
5. Burrows L, Goldstein AT. The treatment of vestibulodynia with topical estradiol and testosterone. *Sex Med*. 2013;1:30–3.
6. Bornstein J, Goldschmid N, Sabo E. Hyperinnervation and mast cell activation may be used as histopathologic diagnostic criteria for vulvar vestibulitis. *Gynecol Obstet Investig*. 2004;58:171–8.
7. Wesselmann U, Bonham A, Foster D. Vulvodynia: current state of the biological science. *Pain*. 2014;155:1696–701.
8. Hibner M, Desai N, Robertson LJ, Nour M. Pudendal neuralgia. *J Minim Invasive Gynecol*. 2010;17(2):148–53.
9. Hartmann D. Chronic vulvar pain from a physical therapy perspective. *Dermatol Ther*. 2010;23:505–13.
10. Rogalski MJ, Kellogg-Spadt S, Hoffmann AR, Fariello JY, Whitmore KE. Retrospective chart review of vaginal diazepam suppository use in high-tone pelvic floor dysfunction. *Int Urogynecol J*. 2010;21:895–9.
11. Adelowo A, Hacker MR, Shapiro A, Modest AM, Elkadry E. Botulinum toxin type A (BOTOX) for refractory myofascial pelvic pain. *Female Pelvic Med Reconstr Surg*. 2013;19:288–92.
12. Burrows LJ, Klingman D, Pukall CF, Goldstein AT. Umbilical hypersensitivity in women with primary vestibulodynia. *J Reprod Med*. 2008;53:413–6.
13. Robert R, Prat-Pradal D, Labat JJ, et al. Anatomic basis of chronic perineal pain: role of the pudendal nerve. *Surg Radiol Anat*. 1998;20:93–8.
14. Gray H, Williams PL, Bannister LH. Gray's anatomy: the anatomical basis of medicine and surgery. 38th ed. New York: Churchill Livingstone; 1995.
15. Shafik A, el-Sherif M, Youssef A, Olfat ES. Surgical anatomy of the pudendal nerve and its clinical implications. *Clin Anat*. 1995;8:110–5.
16. Schraffordt SE, Tjandra JJ, Eizenberg N, Dwyer PL. Anatomy of the pudendal nerve and its terminal branches: a cadaver study. *ANZ J Surg*. 2004;74(1–2):23–6.
17. Goldstein A, Pukall C, Goldstein I (Eds). *Female sexual pain disorders: evaluation and management*. John Wiley & Sons; 2011.
18. Naja Z, Ziade MF, Lönnqvist PA. Nerve stimulator guided pudendal nerve block decreases posthemorrhoidectomy pain. *Can J Anesth*. 2005;52(1):62–8.
19. Robert R, Labat JJ, Bensignor M, Glemain P, Deschamps C, Raoul S, et al. Decompression and transposition of the pudendal nerve in pudendal neuralgia: a randomized controlled trial and long-term evaluation. *Europeanurology*. 2005;47(3):403–8.
20. Bautrant E, de Bisschop E, Vaini-Elies V, et al. Modern algorithm for treating pudendal neuralgia: 212 cases and 104 decompressions. *J Gynecol Obstet Biol Reprod (Paris)*. 2003;32(Pt 1):705–12.

21. Robert R, Labat JJ, Riant T, et al. Neurosurgical treatment of perineal neuralgias. *Adv Tech Stand Neurosurg.* 2007;32: 41–59.
22. Peltier J. Anatomical basis of transgluteal approach for pudendal neuralgia and operative technique. *Surg Radiol Anat.* 2013;35(7): 609–14.
23. Hibner M, Castellanos ME, Drachman D, et al. Repeat operation for treatment of persistent pudendal nerve entrapment after pudendalneurolysis. *J Minim Invasive Gynecol.* 1999;19(3):325–30.
24. Yang CC. Neuromodulation in male chronic pelvic pain syndrome: rationale and practice. *World J Urol.* 2013;31(4):767–72.
25. Peters KM, Feber KM, Bennett RC. A prospective, single-blind, randomized crossover trial of sacral vs pudendal nerve stimulation for interstitial cystitis. *BJU Int.* 2007;100(4):835–9.
26. Powell JJ, Wojnarowska F. Lichen sclerosis. *Lancet.* 1999;53: 1777–83.
27. Goldstein AT, Burrows LJ. Surgical treatment of clitoral phimosis caused by lichen sclerosis. *Am J Obstet Gynecol.* 2007;196(2): 126.e1–4.
28. Smith YR, Haefner HK. Vulvar lichen sclerosis: pathophysiology and treatment. *Am J Clin Dermatol.* 2004;5:105–25.
29. Goldstein AT, Marinoff SC, Christopher K, et al. Prevalence of vulvar lichen sclerosis in a general gynecology practice. *J Reprod Med.* 2005;50:477–80.
30. Dalziel KL. Effect of lichen sclerosis on sexual function and parturition. *J Reprod Med.* 2005;40:351–4.
31. Paniel BJ. Surgical procedures in benign vulvar disease. In: Ridley CM, Neill SM, editors. *The vulva.* 2nd ed. Oxford: Blackwell Science; 1999. p. 288–9.
32. Goldstein I. Dorsal slit surgery for clitoral phimosis. *J Sex Med.* 2008;5(11):2485–8.
33. Peters KM. Pudendal neuromodulation for sexual dysfunction. *J Sex Med.* 2013;10(4):908–11. *Reviews current treatment options for pudendal neuralgia, including the use and success of pudendalneuromodulation.*
34. Kroft J, Shier M. A novel approach to the surgical management of clitoral phimosis. *J Obstet Gynecol Can.* 2012;34(5): 465–71. *This reference provides an alternative approach with case reports using a CO2 laser in the surgical treatment of clitoral phimosis.*
35. Goldstein A. Perineoplasty and vaginal advancement flap for vulvar granuloma fissuratum. *J Sex Med.* 2011;8(11):2984–7.
36. Rouzier R, Haddad B, Deyrolle C, et al. Perineoplasty for the treatment of introital stenosis related to vulvar lichen sclerosis. *Am J Obstet Gynecol.* 2002;186:49–52.
37. Bohm-Starke N, Hilliges M, Falconer C, et al. Neurochemical characterization of the vestibular nerves in women with vulvar vestibulitis. *Gynecol Obstet Invest.* 1999;48(4):270–5.
38. Friedrich Jr EG. Vulvar vestibulitis syndrome. *J Reprod Med.* 1987;32(2):110–4.
39. Kamdar N, Fisher L, MacNeill C. Improvement in vulvar vestibulitis with montelukast. *J Reprod Med.* 2007;52(10):912–6.
40. Steinberg AC, Oyama IA, Rejba AE, et al. Capsaicin for the treatment of vulvar vestibulitis. *Am J Obstet Gynecol.* 2005;192(5):1549–53.
41. Boardman LA, Cooper AS, Blais LR, et al. Topical gabapentin in the treatment of localized and generalized vestibulodynia. *Obstet Gynecol.* 2008;112(3):579–85.
42. Wooddruff JD, Genadry R, Poliakoff S. Treatment of dyspareunia and vaginal outlet distortion by perineoplasty. *Obstet Gynecol.* 1981;57(6):750–4.
43. Tommola P, Unkila-Kallio L, Paavonen J. Surgical treatment of vulvar vestibulitis: a review. *ACTA Obstet Gynecol Scand.* 2010;89(11):1385–95.
44. Goldstein A. Surgery techniques: surgery for vulvar vestibulitis. *J Sex Med.* 2006;3:559–62.
45. Goetsch MF. Incidence of Bartholin's duct occlusion after superficial localized vestibulectomy. *Am J Obstet Gynecol.* 2009;200(6): 688.e1–6.
46. Marinoff SC, Turner ML. Vulvar vestibulitis syndrome: an overview. *Am J ObstetGynecol.* 1991;165(4 pt 2):1228–33.
47. Marinoff SC. Surgical treatment of vulvar vestibulitis. In: *Vulvodinia Workshop: current knowledge and future direction.* 1997; p. 28–32.
48. Swanson CL, Rueter JA, Olson JE, et al. Localized provoked vestibulodynia: outcomes after modified vestibulectomy. *J Reprod Med.* 2014;59(3–4):121–6. *Discusses recent success rates of a modern modified approach to the vulvar vestibulectomy to provide evidence of the success of the procedure.*
49. Tommola P, Unkila-Kallio L, Paavonen J. Long-term well-being after surgical or conservative treatment of severe vestibulitis. *Acta Obstet Gynecol Scand.* 2012;91(9):1086–93.
50. Goldstein AT, Klingman D, Christopher K, Johnson C, Marinoff SC. Outcome assessment of vulvar vestibulectomy with vaginal advancement for vulvar vestibulitis syndrome: results of a post-operative questionnaire survey. *J Sex Med.* 2006;3(5):923–31.