ANAL PAIN CAUSED BY ENTRAPMENT OF NERVUS PUDENDUS

- Etiology and Treatment -

Ahmed Shafik, MD, PhD, Professor and Chairman, Department of Surgery and Experimental Research, Faculty of Medicine, Cairo University, Cairo

PUDENDAL CANAL SYNDROME

Pudendal canal syndrome (PCS) comprises entrapment of the pudendal nerve (PN) in the pudendal canal (PC). It presents with the sensory and / or motor manifestations of PN notably pelvic pain and fecal or urinary incontinence. In this communication, we present our study of the PCS.

Pudendal canal. The anatomy of the PC was studied in 26 cadavers: 10 stillborn and 16 adults (mean age 48.2 years)[1]. Two approaches were used to expose the PC: 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. It started at a mean distance of 0.8 cm from the ischial spine in the stillborn and of 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. The PC contained the PN and vessels embedded in loose areolar tissue. The 3 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 arranged in a criss-cross plywood pattern which appears to have a pumping action on the pudendal vessels in the PC. The obturator fascia consists of collagen only. The PC seems to act as a “pulley” for the PN and vessels as they pass from the pelvic cavity to the ischiorectal fossa .

Pudendal nerve. The PN is an important motor and sensory nerve to the pelvic organs and perineum. It provides supply for the anal and urethral sphincters, Levator ani as well as cutaneous and skeletal motor innervation of the penis and clitoris[2,3]. Pudendal neuropathy or nerve injury leads to

pathologic changes of these structures. PN compression in the PC causes the PCS (an entrapment syndrome)[4-11]

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 intraabdominal pressure caused by straining at stool is borne by the levator plate and in particular by its rectococcygeal raphe which is its most dependant and durable part[12]. The levator hiatus being plugged by the viscera which are firmly fixed to the levator plate by the hiatal ligament, is immune against increased intraabdominal pressure. Moreover, the hiatal ligament attachment to the anorectal junction firmly seals the pelvic floor and prevents intraabdominal pressure leak to the infralevator structures. This mechanism is maintained so long as the increased intraabdominal pressure is within its physiological limit. An increase beyond this limit, as in chronic straining conditions, (chronic constipation at prolonged 2nd stage of labor) would tend to throw its load on the rectococcygeal raphe, hiatal ligament and the levator hiatus. The rectococcygeal raphe and hiatal ligament, both tendinous, become overtretched and subluxated. Consequently, the levator plate saggs down leading to suspensory sling subluxation[12,13] (fig.1). Being continuously exposed to increased intraabdominal pressure, the sagging levator plate acquires a vertically oblique position; the levator hiatus, consequently, is overwidened and lowered so that most of the anal canal, urethra and vagina lie above it (fig.1).

These changes in the levator plate and its ligaments would interfere with the normal evacuation (defecation / urination) mechanism, with the ultimate development of the 'levator dysfunction syndrome'[12,13] . Thus, on straining at stool or urination, the contraction of both the sagging levator plate and the

subluxated suspensory sling is too weak to effect anal canal or vesical neck opening. 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 the intraabdominal pressure. Thus, on straining at defecation and urination, the increased intraabdominal pressure is transmitted through the abnormally wide levator hiatus to the anal canal and urethra leading to their obstruction[12,13]. It seems that the direct anal canal compression by the increased intraabdominal pressure effects the high anal pressure detected during straining in all patients of the levator dysfunction syndrome.

The ultimate result of levator dysfunction is impairment of the urination and defecation mechanism so that straining at stool or micturition would tend to close rather than open the anal canal and vesical neck. This is essentially attributable to: (a) atrophy and 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 (b) leak of the high intraabdominal pressure through the widened levator hiatus to the anal canal and urethra, obstructing them[12,13].

Causes of levator dysfunction. Conditions leading to chronic straining at stool or micturition or prolonged second stage of labour or forceps application would disturb the levator function and ultimately result in levator dysfunction syndrome[13]. In view of the effect which chronic straining evidently has, first on the levator plate and subsequently on the whole of the defecation or urination mechanism, this (straining) is to be considered the first stage in the developing levator dysfunction syndrome.

It seems there is an individual susceptibility to straining which could depend on the different levator crural and rectococcygeal raphe patterns. 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[12]. This, in consequence, would necessitate an 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 these cases, despite the extra-straining.

Also considered, however, should be other possible causes of chronic straining as occupational straining, severe loss of muscle tone from a debilitating disease or senility, or the increased intraabdominal pressure from a fat-laden viscera in obese stout-built subjects.

Role of levator ani in idiopathic pelvic pain (proctolgia vulvodynia, scrotalgia and prostatodynia) and the pudendal canal syndrome. Levator dysfunction might occur as a result of straining at stool or prolonged second stage of labor or forceps application with a resulting pudendal canal syndrome. The mechanism is described later on. The patients with prostatodynia, scrotalgia. vulvodynia and proctalgia have common clinical presentation and investigative findings of PCS[4-11]. These comprise the neurogenic nature of the pain, the motor and sensory changes being localized to pudendal nerve distribution, and the increased PNTML which indicates the involvement of the distal part of the nerve[4-11]. The diagnosis of PCS is further ascertained by the temporary disappearance of the scrotal pain following bilateral PN block.

Mechanism of pudendal nerve entrapment. The PN is a mixed nerve. Arising from the sacral plexus, it leaves the pelvic cavity through the greater sciatic foramen. It passes over the sacrospinous ligament close to the ischial spine to enter the perineum through the lesser sciatic foramen. The nerve then passes forward in the pudendal canal. Its branches comprise the inferior rectal, perineal and dorsal nerve 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[2,3]. The perineal nerve supplies the external 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.

An increase of the intraabdominal pressure beyond the normal physiologic limits, as occurs in chronic straining at stool, urination or prolonged 2nd stage of labor, would eventually result in subluxation and sagging of levator ani[13]. The latter muscle lies at a lower level than normal and consequently pulls on the pudendal nerve (fig.2). 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. Continuous nerve stretch leads to neurapraxia or axonotmesis[13]. Being entrapped in the PC and subjected to continuous stretch, the PN may undergo edema with subsequent nerve compression inside the canal, leading to nerve ischemia which would add to the nerve damage. Eventually “entrapment neuropathy” occurs.

It seems that pudendal neuropathy affects the branches of the PN in different grades. The main brunt of neuropathy may involve one of the 3 branches and, to a lesser extent, the other 2 branches[4-11]. This is evident from the sensory and motor affection. The patients present clinically with fecal or stress urinary incontinence, and pelvic or perineal pain which takes the form of scrotalgia, proctalgia, prostatodynia or vulvodynia. They may complain of erectile dysfunction[14]. These presentations occur singularly or incombination. Pudendal canal fasciotomy decompresses the nerve and sets it free within the ischiorectal fossa. The nerve lies loosely free in the ischiorectal fossa, thus escaping being stretched by the contracting and sagging levator ani muscle.

Pudendal nerve decompression was effective in the treatment of the PCS[4-12]. The scrotal, anal, vulvar and perineal pain disappeared in most of the cases. There was improvement in the sensory and motor affection of the PN as evident from improvement of anal and stress urinary incontinence, anal reflex, EMG activity of the external urethral and anal sphincters, and PNTML. Erctile dysfunction improved in some patients. Although the scrotal and perineal hypoesthesia as well as the anal reflex did not improve in all the patients alike, the initially increased PNTML showed significant reduction. Failures might be due to incomplete pudendal canal decompression, an irreversible PN damage or faulty diagnoses.

Technique of PCD

Three approaches were conducted in the treatment of PCS:

1. Anterior approach
2. Posterior approach
3. Laparascopic approach

Briefly, the anterior approach[4,6-12] comprises a paraanal incision; the ischiorectal fossa is entered and the inferior rectal nerve is identified and followed to the PC which is slit open (fig.3) For the posterior approach[5], a parasacral incision was done and the gluteus maximus divided and reflected laterally (fig.4). The PN is identified and followed to the PC. We prefer the anterior approach because it is simple, easy, direct approach and we can deal with anal pathology. The posterior approach is reserved for the recurrent cases. PC decompression was performed laparoscopically[15].

REFERENCES

1. Shafik A, Doss S. Pudendal canal. Surgical anatomy and clinical implication. *Am Surg* 1999; 65:176-180
2. Shafik A, El Sherif M, Youssef A, El-Sibai O. Surgical anatomy of the pudendal nerve and its clinical implications. *Clin Anat* 1995; 8: 110-115.
3. Shafik A, Doss S. Surgical anatomy of the somatic terminal innervation to the anal and urethral sphincters: Role in anal and urethral surgery. *J Urol* 1999; 161: 85-89.
4. Shafik A. Pudendal canal syndrome. Description of a new syndrome and its treatment. Report of 7 cases. *Coloproctology* 1991; 13:102-109.
5. Shafik A. The posterior approach in the treatment of pudendal canal syndrome. *Coloproctology* 1992; 14: 310-315.
6. Shafik A. Role of pudendal canal syndrome in the etiology of fecal incontinence in rectal prolapse. *Digestion* 1997; 58: 489-493.
7. Shafik A. Pudendal canal decompression for the treatment of fecal incontinence in complete rectal prolapse. *Amer Surg* 1996; 62: 339-343.
8. Shafik A. Pudendal canal decompression in the treatment of urinary stress incontinence. *Int Urogynecol* J 1994; 5: 215-220.
9. Shafik A. Pudendal canal syndrome: A new etiological factor in prostatodynia and its treatment by pudendal canal decompression. *Pain Digest* 1998; 8: 32-36.
10. Shafik A. Chronic scrotalgia. Report of 4 cases with successful treatment. *Pain Digest* 1993; 3: 252-256.
11. Shafik A. Pudendal canal syndrome as a cause of vulvodynia and its treatment by pudendal nerve decompression. *Eur J Obstet Gyn Reprod Biol* 1998; 80: 215-220
12. Shafik A. A new concept of the anatomy of the anal sphincter mechanism and the physiology of defecation. VIII. Levator hiatus and tunnel. Anatomy and function. Dis Colon Rectum 1979; 22:539-549
13. Shafik A. A new concept of the anatomy of the anal sphincter mechanism and the physiology of defecation. XVIII. The levator dysfunction syndrome. A new syndrome with report of 7 cases. *Coloproctology* 1983; 5: 158-165
14. Shafik A. Pudendal canal decompression in the treatment of erectile dysfunction. *Arch Androl* 1994; 32:141-149

15. Shafik A. Endoscopic pudendal canal decompression for the treatment of fecal incontinence due to pudendal canal syndrome. *J Laparoendosc & Adv Surg Techn* 1997; 7(4): 227-234

ILLUSTRATIONS

Fig. 1: The mechanism of defecation in levator dysfunction syndrome. (A) Normal findings. (B) Pathological findings in levator dysfunction syndrome at rest; atrophy and 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 rectal neck to the intraabdminal pressure. (C) Levator dysfunction syndrome at defecation. On straining at stool, contraction of both the sagging atrophic levator plate and the subluxated suspensory sling is too weak to effect rectal neck opening in front of the descending faecal mass. The increased intraabdominal pressure leaks, through the abnormally wide levator hiatus, to the rectal neck closing it, with a resulting faecal obstruction.

Fig. 2: Mechanism of pudendal nerve stretch; A) At rest: levator muscle is relaxed (cone shaped); B) On contraction at stool: Levator muscle is elevated and flattened; C) Difficult deliveries chronic straining at stool cause levator subluxation and sagging with a resulting pudendal nerve stretch

Fig. 3: Steps of the pudendal nerve decompression operation. (A,B) Incision. (C) Inferior rectal nerve crossing ischiorectal fossa. (D) Inferior rectal nerve hooked with index finger. (E) Inferior rectal nerve followed to pudendal nerve. Inset showing the pudendal nerve in and outside the pudendal canal.

Fig. 4: Pudendal nerve decompression: posterior approach. (A) Parasacral incision. (B) Division of gluteus maximus. (C) Triangle of identification. It consists of piriformis muscle, sacrotuberous ligament and greater and lesser sciatic notches. At the floor of the triangle there is the ischial spine and the sacrospinous ligament with the overlying pudendal nerve and vessels. Inset show the anatomical structures in the area of the parasacral incision

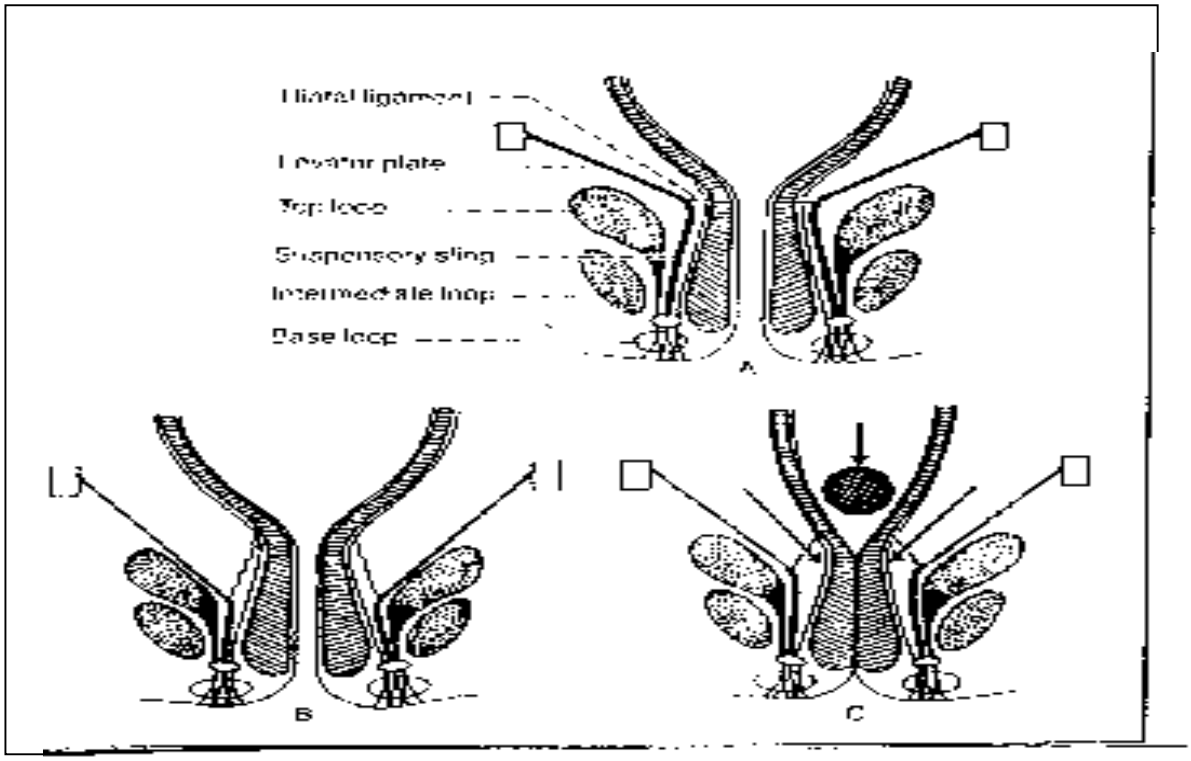

Fig. 1: The mechanism of defecation in levator dysfunction syndrome. (A) Normal findings. (B) Pathological findings in levator dysfunction syndrome at rest; atrophy and sagging of the levator plate as well as hiatal ligament and suspensory sling subluxation. Levator hiatus is widened and lowered so as to expose the rectal neck to the intraabdminal pressure. (C) Levator dysfunction syndrome at defecation. On straining at stool, contraction of both the sagging atrophic levator plate and the subluxated suspensory sling is too weak to effect rectal neck opening in front of the descending faecal mass. The increased intraabdominal pressure leaks, through the abnormally wide levator hiatus, to the rectal neck closing it, with a resulting faecal obstruction.

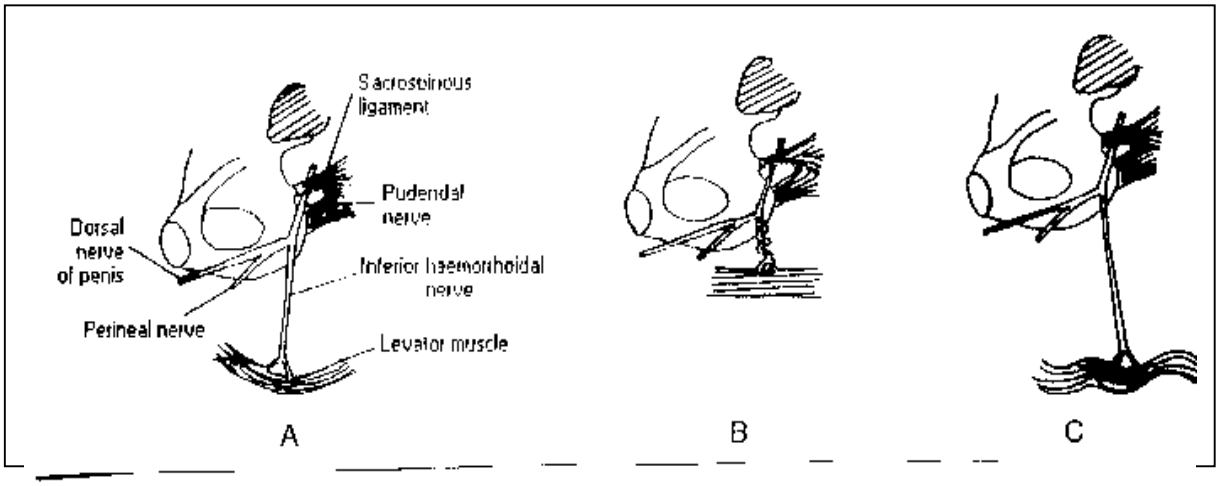

Fig. 2: Mechanism of pudendal nerve stretch; (A) At rest: levator muscle is relaxed (cone shaped); (B) On contraction at stool: Levator muscle is elevated and flattened; (C) Difficult deliveries chronic straining at stool cause levator subluxation and sagging with a resulting pudendal nerve stretch

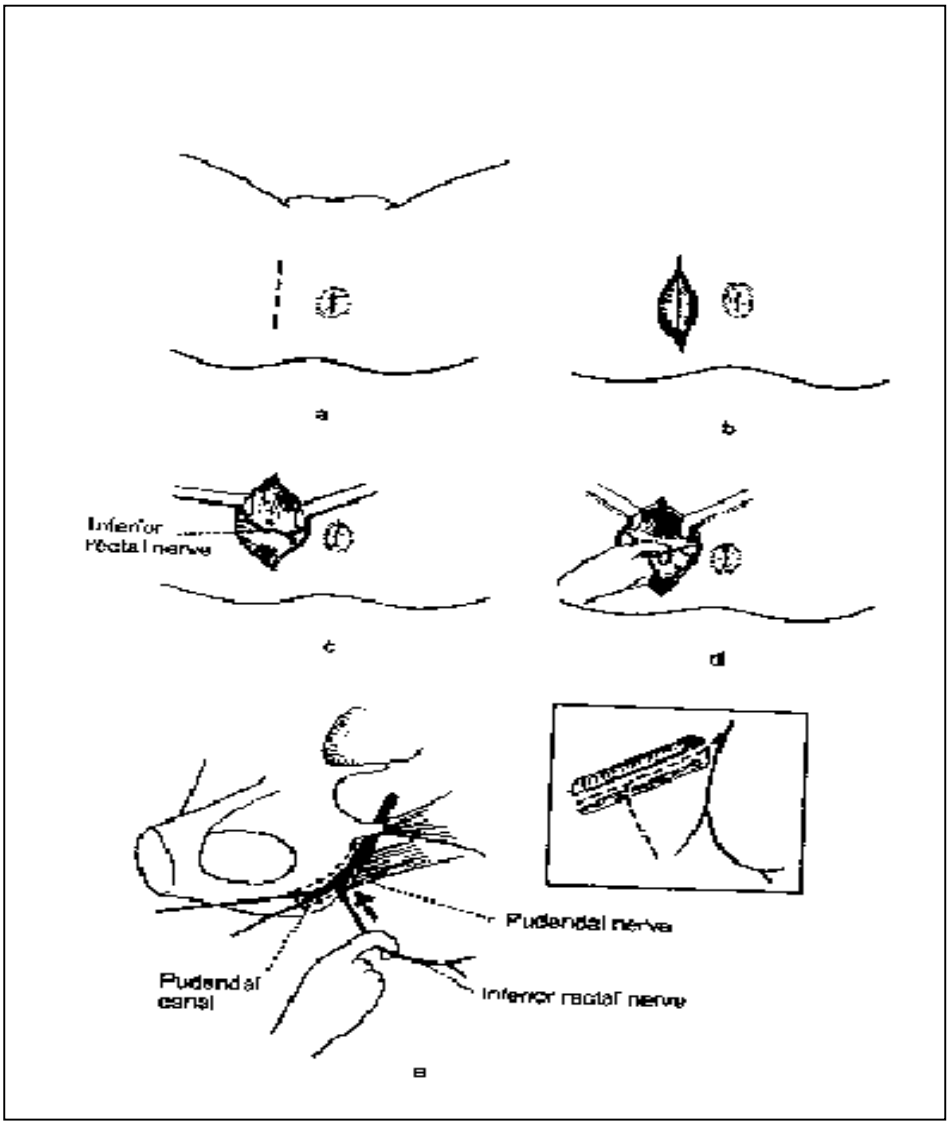

Fig. 3: Steps of the pudendal nerve decompression operation. (A,B) Incision. (C) Inferior rectal nerve crossing ischiorectal fossa. (D) Inferior rectal nerve hooked with index finger. (E) Inferior rectal nerve followed to pudendal nerve. Inset showing the pudendal nerve in and outside the pudendal canal.

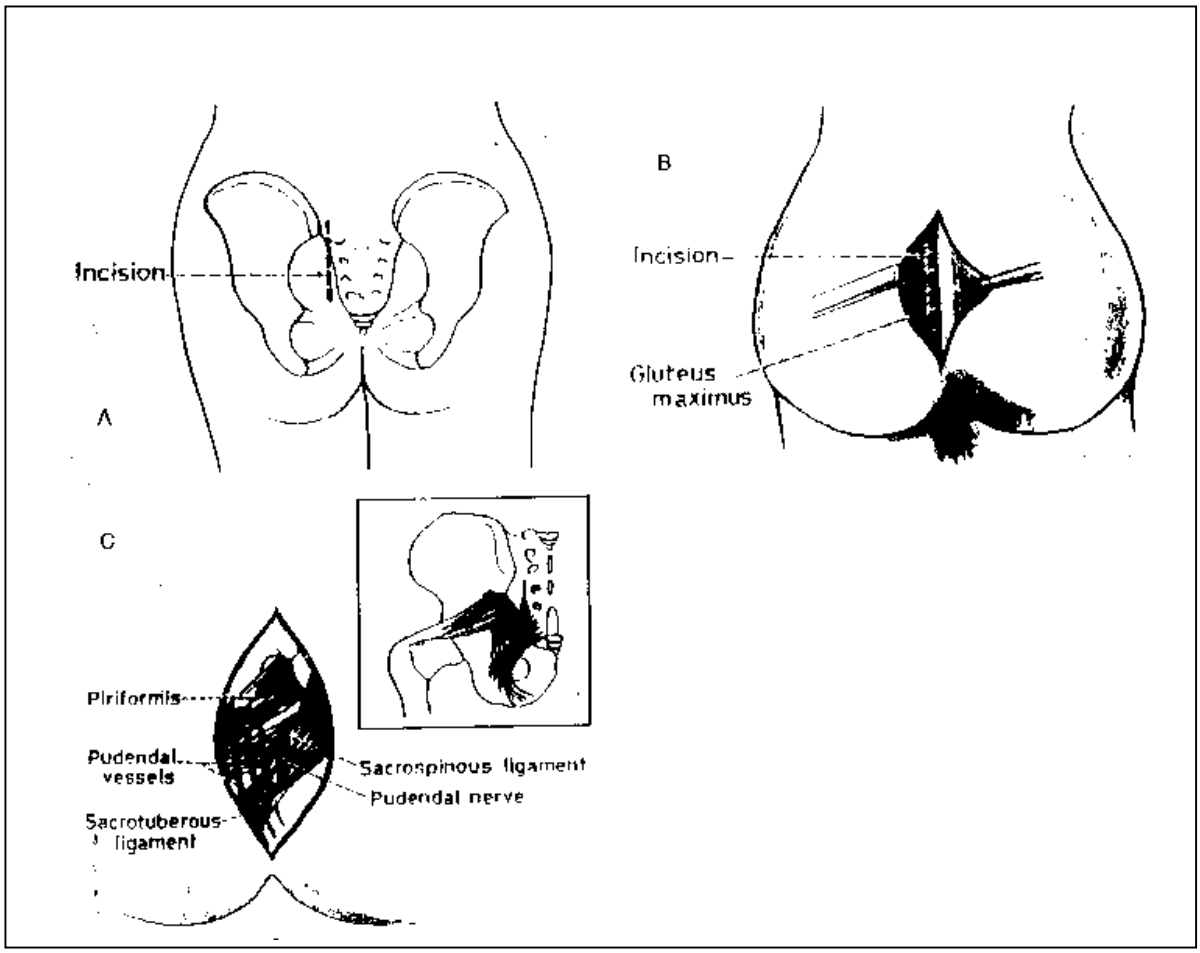

Fig. 4: Pudendal nerve decompression: posterior approach. (A) Parasacral incision. (B) Division of gluteus maximus. (C) Triangle of identification. It consists of piriformis muscle, sacrotuberous ligament and greater and lesser sciatic notches. At the floor of the triangle there is the ischial spine and the sacrospinous ligament with the overlying pudendal nerve and vessels. Inset show the anatomical structures in the area of the parasacral incision