

## **PUDENDAL NERVE ENTRAPMENT**

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PNE, an abbreviation for pudendal nerve entrapment may also represent an abbreviation for a diagnosis Probably Not Entertained. The reason that it is not commonly diagnosed is that it is the mother of all masqueraders. I believe that this diagnostic deception, in the eyes most medical practitioners, stems from two causes. The first is that it is a relatively new diagnosis that was first described by Amarenco in 1987 as the Alcock's Canal or Cyclists Syndrome, and therefore is a relatively unfamiliar entity. And secondly, the symptoms that it produces fall into the domain of many medical specialists who falsely recognize them as more familiar problems and therefore give them a knee jerk diagnosis-treatment response.

The uninformed are easily deceived because PNE throws out a camouflage screen operating on two levels. The primary level represents the

pain created by the nerve compression itself and the second levels are the symptoms created by the muscles dysfunction brought about by the compressed and damaged nerve. And it is this secondary muscle dysfunction that adds to the source of the diagnostic confusion. Since the pudendal nerve innervates the muscles of the pelvic floor which includes the urinary and anal sphincters, nerve compression not only causes pain, but also causes increased muscle tension which is transferred to the penetrating organs i.e. the urethra, vagina, and rectum. This increased muscle tension causes symptoms in the organs, as well as the muscles themselves. When this muscle tension exceeds a certain threshold, the muscles develop myofascial trigger points. Complicating the matter even more is that these local trigger points refer the pain to more distant areas.

Now you can begin to understand the practitioner's dilemma. They are presented with the same symptom complexes as seen with myofascial dysfunction in its disguise as visceral disease i.e. urinary hesitancy, urgency and frequency, interstitial cystitis, prostatodynia, anal pain, scrotal pain, orgasmic pain, and pain with sitting. However, even if the disguised muscle component is recognized, the underlying and driving force is different than that usually seen with myofascial trigger points.

Fortunately, this dilemma can easily be solved by asking some simple questions. Is the pain initiated or worsened with sitting and lying on the affected side? Is the pain improved with standing and when sitting on a toilet seat?

However when these questions are not asked and the diagnosis is not entertained patients begin to receive the “standard treatments’ for (a) nonbacterial /bacterial prostatitis, prostatodynia, or orchialgia with long term antibiotics, anti-inflammatory medication or surgery. (b) Vulvar pain with antifungals, laser surgery, laproscopy to rule out endometriosis, or a hysterectomy. (c) Or for a diagnosis of levator ani syndrome, coccygodynia and proctalgia a treatment course of high voltage galvanic stimulation, hemmorhoidectomy, anal sphincterotomy, coccygectomy, or Botox injections.

Therefore, it is no wonder that the time to the diagnosis of PNE is a mean of 4 years (1-15 yrs); have seen 10-30 M.D.s; and have suffered innumerable invasive and noninvasive procedures; and have developed significant central and peripheral sensitization with the concomitant allodynia, hyperalgesia ; and have developed an attitude of hopelessness and have become markedly depressed to the point of committing suicide.

In my practice I have in excess of seventy patients with PNE, some of whom I initially misdiagnosed as having only pelvic floor myofascial trigger points and hypertonus. It is for these suffering patients who have essentially no place to turn for help or have been made worse by treatment that I am on a crusade to both increase awareness and to develop more effective conservative therapy for PNE.

Why does PNE occur? In general, it is the result of our pelvic floor anatomy in which the pudendal nerve both follows a tortuous course that leaves it exposed to damage by ligaments, fascia, and muscles, and has a position inside of the ischial tuberosities that leave it vulnerable to direct trauma. However, there must be more specific reasons otherwise every bicycle rider would develop PNE. Possibilities that increase the likelihood of developing PNE are (1) the wider pelvis seen in women who have a 3:1 incidence over men. (2) genetic variations in the nerve course and pelvic muscle structure (3) or developmental changes in the muscles and pelvic bones.

Dr. Stanley Antolak attributes many cases of entrapment, especially in athletes, to the remodeling of the ischial spine. Flexion exercises of the hip from such activities as cycling and squats in early years can result in a pull on the ischial spine by the hypertrophied pelvic floor muscles. These forces

will cause a rotation of the sacrospinous ligament causing it to override the sacrotuberous ligament, thus acting like a “lobster claw” which pinches the traversing pudendal nerve. The remodeling also causes the pudendal nerve to traverse a longer course making it more vulnerable to squats, sitting, or standing from a seated position. This results in an additional site for repetitive micro trauma of the pudendal nerve as it curls around the ischial spine.

A review of the pelvic floor anatomy is important in understanding the mechanisms of pudendal nerve injury, the symptoms it produces, and the formulation of effective treatment.

The pudendal nerve arises most often from the S3 nerve root with additional fibers from S2 and S4. After formation the intrapelvic trunk leaves the pelvic region through the greater sciatic notch, passing between the piriformis muscle and the sacrospinous ligament. It is very important to note that the nerve does not always turn directly behind the ischial spine, but may take a variable course more medial. This is important in regards to x-ray guided blocks which I will discuss later. At this level the nerve is adherent to the posterior portion of the sacrotuberous ligament, and lies under the pudendal artery in the gluteal region.

It then re-enters the pelvis and describes a bow that is concave medially. At this point it becomes flattened anterior-posterior against the aponeurosis of the obturator internus muscle which creates a sliding space (Alcocks canal) by doubling itself with a second fine layer. In this position the nerve rides on the very sharp superior concave expansion of the sacrotuberous ligament, the falciform process.

At this level of contact, the nerve trunk is behind and the terminal branches are in front. At the distal position the nerve gives off the inferior rectal branch prior to dividing into two terminal branches, the perineal nerve and the dorsal nerve of the penis/clitoris. The inferior rectal branch crosses the ischioanal fossa to innervate the external anal sphincter and the skin around the anus. The perineal branch divides to one that supplies the skin of the scrotum/labia and one that supplies the muscles-transverse superficial perineal, bulbocavernosus, ischiocavernosus, urethral sphincter, corpora cavernosum of the urethra, and the urethral mucosa. The dorsal nerve of the penis/ clitoris runs along the ischial ramus between the superior and inferior layers of the fascia of the urogenital diaphragm. It pierces this layer and goes forward with the dorsal artery of the penis/clitoris between the suspensory ligament to the glans penis or clitoris.

The visceral branches arise from S3 and S4 and occasionally S2 and go to the bladder, rectum, and vagina and communicate with the sympathetic plexus. Muscle branches S4 supply the levator ani, coccygeus, and external anal sphincter.

### **WHERE ARE THE ANATOMIC SITES OF PNE?**

David Butler, the author of *MOBILIZATION OF THE NERVOUS SYSTEM and THE SENSITIVE NERVOUS SYSTEM*, describes the anatomic sites that predispose nerves to injury. They are (1) soft tissue, osseous, or fibroosseous tunnels. (2) Areas of nerve branching, especially when it branches at an acute angle. (3) When the nerve is relatively fixed (such as the ischial spine). (4) And when the nerve is subjected to friction because it is in close proximity to an unyielding interface.

Butler's descriptions of anatomic sites that predispose to nerve injury are those that are seen in pudendal nerve entrapment syndromes. The sacrospinous and sacrotuberous ligaments, including the falciform process, form two sides of a tunnel which is closed by the obturator internus muscle. If the obturator internus shortens and widens because of injury, the tunnel narrows and can be a source of entrapment. These ligaments are also sites of friction when the nerve moves, and represent the most common sites of

nerve injury. In Robert's surgical patients, the source of the entrapment was at the sacrospinous ligament in 58%; the sacrotuberous in 69%; and the falciform process in 42%. The tunnel formed by the obturator fascia, the Alcock's canal, houses a branching segment of the pudendal nerve is the site of the injury in 48% of the patients. The ischial spine, a point of nerve fixation, is the source of damage in 11% of Robert's cases. Hypertrophy of the piriformis muscle can narrow the sciatic notch and compress the pudendal nerve against the posterior edge of the sacrospinous ligament (17%). Therefore, this site serves both as a tunnel and a point of fixation.

In addition to the above, it is my opinion that there may be minor degrees of nerve fixation anywhere along the nerve course, not only in the above major points of fixation. For example, the obturator internus muscle, which attaches to the greater trochanter of the hip, supports a large portion of the nerve in Alcock's canal. Therefore, injuries to the hip, knee, or back or abnormal or stressful movements that result in spasm and widening of this muscle can compress the nerve and lead to minor areas of restriction.

### MECHANISMS OF ENTRAPMENT

The above sites represent potential areas of nerve damage, but what are the mechanisms that activate the damaging process.

1. Direct trauma such as from bicycling (pedal pumping increases perineal pressure), sitting on a hard chair, or as result of gynecologic, urologic, or orthopedic surgery. During sitting there is an ascent of the ischiorectal fat which pushes laterally against the falciform process of the sacrotuberous ligament. This pressure is transmitted to the nerve trunk contained in the aponeurosis of the obturator internus. If the nerve is fixed between the ligaments or between the piriformis muscle and the sacrospinous ligament, it cannot escape superiorly.

2. Nerve stretching. Dr. Ahmed Shafik(1990) described the Pudendal Canal Syndrome as a new clinical entity related to straining at stool or with difficult deliveries. He postulated that chronic straining resulted in a sagging of the levator muscle which in turn pulled on the pudendal nerve and artery. Since these structures are fixed around the sacrospinous ligament and entrapped in the pudendal canal, it exposes them to stretching. When exposed to continuous stretching, the pudendal artery can develop arteritis and erectile dysfunction and the nerve, a neuropathy.

3. A combination of entrapment and stretching as seen above.

4. Sacroiliac joint instability may be another source of nerve injury. Since the sacrospinous and sacrotuberous ligaments attach to the sacrum, sacral movement will cause traction, movement and possibly inflammation of these ligaments. Therefore this very narrow tunnel through which the pudendal nerve passes may be compromised; thereby compressing the nerve. We have one such patient with a PNML test of 5 and 6 msec that is now symptom free after stabilization of the SI joints with proliferative therapy. This is another reason that a thorough biomechanical evaluation is crucial in the treatment plan of PNE.

Dr. John Bascom recently working with Dr. Benson has developed a new paradigm for the source of pain in PNE. He believes that fixation of the nerve at the level of the ischial spine or the falciform process at one end and its adherence, by trauma, to the levator muscle on the other end is the set up for the pain syndrome. Instead of compression of the nerve with sitting he believes that the loose muscle between the anus and ischial spine, with its attached nerve, moves superiorly creating a stretch of the nerve. When the patient lies on the painful side, the anus moves toward the ischial tuberosity, and the viscera falls away from the levators. The resultant slack muscle with its attached nerve moves toward the head, and the nerve which is fixed at the opposite end to the ischial spine receives a painful tug.

### PATHOPHYSIOLOGY OF PUDENDAL NERVE PAIN

These theories explain how the nerve becomes entrapped on a macro level, but what happens microscopically to cause and perpetuate the symptoms? From the standpoint of clinical treatment it is important to have a basic understanding of neuroanatomy and pathophysiology. The nerve axons are surrounded by multiple layers of connective tissue; the endo, peri, epi, and mesoneurium. In general at least 50% of a peripheral nerve is connective tissue. This supporting connective tissue is highly reactive and when it is injured it can trigger a train of events that leads to varying degrees of nerve damage. Therefore, the pathophysiology of nerve entrapments involves ischemia of the vasa nervorum (see Ellis) The blood vessel damage from the nerve compression or trauma results in plasma and platelet extravasation which creates an endoneural edema. This endoneural fluid under significant pressure within a rigid nerve sheath causes a compartment syndrome. The intraneural edema sets up an inflammatory reaction with subsequent collagen deposition and fibrosis which further increases intraneural pressure and initiates a self-perpetuating cycle.

With compression and damage to a segment of the axon, the normal axoplasmic (cytoplasmic) flow is altered. The result is the so called ‘double crush’, whereby abnormalities occur in other areas of this axon. This process is seen in carpal tunnel or ulnar nerve entrapment syndromes in which a large percentage of neural lesions are found in the cervical portions of the nerve. These alterations make the nerve more vulnerable to friction injuries especially in tunnel areas.. As a result, the traumatized nerve is exposed to repeated bouts of ischemia in vasa nervorum which is precipitated by inappropriate movements or static positions that stretch the nerve. This vicious cycle of repeated micro-inflammation progressively involves more neural fascicles, thus spreading symptoms, and promotes nerve sprouting and an eventual perineural and endoneural fibrosis.

During this pathologic process portions of the myelin sheath may be lost by compression, thereby opening mechanosensitive ion channels. This can result in the formation of ***ABNORMAL IMPULSE GENERATING SITES (AIGS)*** (cite Butler) which can independently generate its own spontaneous activity and increase mechanosensitivity. Hence, active movements, palpation of the nerves, or neurodynamic testing may result in pain and/or dysesthesia. Unfortunately, the peripheral nerve is only the first level of a pain syndrome created by local pathology. The

second level occurs when the noxious input from the altered peripheral nerve to the central nervous system causes central sensitization.

### **DIAGNOSIS OF PNE**

Now that we have reviewed the pudendal nerve anatomy and the mechanisms and consequences of entrapment, let us turn to the clinical aspects of diagnosis and treatment. The diagnosis is made by the history of pain with sitting which improves with standing or sitting on a toilet seat, a pelvic examination which elicits tenderness on palpation of the pudendal nerve, and electrophysiologic testing that reveals a delayed conduction time. The most widely used method of electrophysiological testing of pudendal nerve function is that described by Kiff and Swash at St. Mark's Hospital in London. They used a rubber finger stall that has two stimulating electrodes at the tip and two surface electrodes for recording mounted three cm. proximally at its base. The index finger, mounted with the device, is inserted into the rectum and placed on the ischial spine. Electrical stimulation is then initiated and the latency of the response to the anal sphincter is recorded on surface or needle electrodes. The normal mean terminal latency is 2.0 +or – 0.3 msec.

It must be pointed out that the pudendal nerve terminal motor latency test (PNTML) is solely a motor study, and is of importance only if the study

is abnormal. In other words, the sensory nerve fiber component of the nerve more peripherally located can be compromised without involving the motor fibers. This anatomical situation can result in a patient with sensory fiber compression and pain having a negative PNTML test. In addition the test does not indicate the extent of injury or entrapment, but only if the nerve is responding abnormally.

A comprehensive examination should include sensory nerve tests; as well as testing of the components of motor function, and EMG of the pelvic floor. With this information one could ascertain the severity of the damage i.e., if there is axonal damage or focal demyelization, determined by the motor amplitude and EMG characteristics; if the process is of recent or longstanding; and if there is an attempt to regenerate (needle EMG).

Dr. Benson has developed a sensory testing method, based on the bulbo/clitorocavernosus reflex, in which a mild stimulus is applied to the glans penis or adjacent to the clitoris and the reflex conduction time to a pelvic floor muscle is measured.

In spite of all the testing confusion can arise if any of the sacral nerve roots more proximally are involved. Therefore if pudendal nerve blocks do not give any degree of pain relief, one must consider either severe entrapment damage or sacral nerve root compression. In the latter case, a

lumbosacral MRI would be required for the diagnosis. Another point of confusion in interpreting PNTML test is that prolonged or difficult childbirth and severe and longstanding constipation can cause permanent stretch injuries from pelvic floor descensus. Therefore, a past history of these complaints needs to be taken into consideration when formulating a treatment program.

### TREATMENT

In order to develop an effective conservative treatment plan one must revisit the pathophysiology of PNE. In essence, the nerve becomes fixed and inflamed. Therefore, the treatment should be directed toward nerve mobilization with manual therapy to decrease the nerve fixation and tension which can contribute to repetitive trauma; oral anti-inflammatory medication, nerve blocks with steroids or heparin; and the avoidance of prolonged sitting without a protective cushion.

### MANUAL THERAPY

Since the pudendal nerve can be entrapped by the sacrospinous and sacrotuberous ligaments or the obturator internus and piriformis muscles, attention is directed to correcting dysfunction in these areas. I palpate the nerve to assess the areas of maximal tenderness, and then check all of the pelvic floor muscles for spasm and trigger points. I generally begin with the piriformis since its release has a profound effect on reducing spasm in the other muscle groups. When this is done internally, I insert my finger in the rectum or vagina while the patient is in the lithotomy position and grasping and pulling the knee of the involved side towards the opposite shoulder.

The internal finger then sandwiches and compresses the muscle against the fingers of the opposite hand which is placed externally on the buttock. The compression continues intermittently and covers the entire muscle until it softens.

Once this is accomplished an internal active release technique is performed on the obturator internus. Active release technique, invented by Dr. Lahey, a chiropractor in Colorado Springs, is a method in which the patient actively moves a muscle group while the practitioner holds and stabilizes another. In the case of the obturator internus, the internal finger stabilizes the pudendal nerve in Alcock's canal while the patient grasps his or her ankle with the knee bent at a right angle. The lower leg is then used to

rotate the thigh internally, thus stretching and separating the obturator internus muscle from the nerve.

Similar techniques can be utilized externally, but it requires specialized training by Dr. Lahey. From phone conversations that Stephanie Prendergast has had with Dr. Lahey, and from the feedback of our patients, it appears that although ART can reduce pain levels, the effect may not be long-lasting and may need further refinement. It is my personal bias that successful conservative therapy will require the use of multiple therapeutic modalities, and that ART could be one of the cornerstones of treatment. At this time our clinic is integrating this approach into the one using pudendal nerve blocks.

Another important aspect of the physical therapy approach is to determine if muscle dysfunction created by the PNE is contributing to a significant portion of post surgical pain. Overlooking this possibility can lead to assumption of surgical failure. We have found that this surgical “failure” may not be a failure of surgery at all, but rather pain that has developed in muscles that have been subjected to long periods of nerve dysfunction. As a consequence, we have been able to help patients who have continued to have pain after surgery.

## PUDENDAL NERVE BLOCKS

Pudendal nerve blocks are performed for three reasons. (1) The first is to make the diagnosis of PNE. Labat et al, reporting the French experience, stated that anesthetic blocks, when used as a diagnostic test, were considered positive if there was total relief of pain when sitting within one hour of infiltration. (2) Secondly, for its prognostic value. Robert et al also reported that the longer the pain relief after a block, the better the surgical results. Mauillon et al confirmed that observation when he reported that in a study of twelve patients complete disappearance of pain for two weeks was the best predictor of surgical success. (3) Third, and the most important are for the therapeutic effect.

Guided pudendal nerve blocks, the first line of conservative treatment of PNE, comes in many flavors. They are guided by fluoroscopy with or without nerve stimulation, CT, or with the low tech finger. The method chosen appears to depend on the personal skills and experience of the practitioner. Internet debates on techniques relegate the finger to the caveman era. Perhaps, this is why fewer physicians aren't touching patients anymore.

Let us look at the difference between the use of x-ray and the finger. X-rays are used to identify the ischial spine, a landmark under which the

puddental nerve usually passes. The key word is usually, since as Shafik has pointed out, the pudental nerve can vary in its course under the sacrospinous ligament. The reason that electrical stimulation of the needle was introduced was to accurately identify the position of the pudental nerve by eliciting an anal contraction. Without electrical stimulation it is a guess as to whether the needle was placed in the optimal location. In order to overcome this problem, Dr. Bensignor states that he attempts to elicit paresthesias before injecting. In addition the x-ray gives no information regarding inflammation in the Alcock's canal. However for those who are not skilled in finger palpation, x-ray guidance is the best way to approach the treatment.

In order to understand the benefit of finger guidance, one must remember that with nerve entrapment there is inflammation, and the result of inflammation is tenderness. Therefore, when the finger comes in contact with areas of the nerve that is entrapped, tenderness is elicited. It is the tender areas that require the steroid injection, whether in the area of the ischial spine or Alcock's canal, not in an area predetermined by an unfeeling X-ray. X-ray guided blocks can give good results, but can the results be better? In my own personal experience, I have done blocks with CT and fluoroscopy with and without electrical stimulation and find those to give poorer results than finger guided blocks in the same patients.

What does the literature tell us about the results of pudendal blocks?

Amarenco et al reported that using CT guided blocks, 31% of 170 patients had pain relief that lasted an average of 107 days (range 1-900 days); whereas there was partial relief in 22% (37/170) and failure in 47% (80/170). His conclusion (Amarenco et al- 1996) was that there was a cure or near cure in 10.5% of the patients who had blocks in Alcock's canal and 15% of those who were blocked at the sacrospinous ligament. In the latter group partial relief was achieved in 42.5%.

It is somewhat confusing that Robert et al reported very inconsistent and divergent results in two papers. In the first paper, *Surgery* 119,535-39, 1993-94), they found after blocking 48 nerves, there was a benefit in 39 patients (81%) for up to 1 month; six patients (12.5%) had a positive effect for 3 months, and only one (2%) for more than three months. Yet in *Surgical Radiol Anat* 1998 20: 93-98, they stated that 65-70% were cured using X-ray or scanner guided injections. There is no mention of numbers, or length of time followed. To me, giving the range of 65-70% implies that there was no real study, but rather that these percentages were a "guestimate." three months. Labat (*Journal d' Urologie* 96(5) 239-44, 1990) reported that 60% had good results three months later. Bensignor (*Diagnosis and treatment of chronic pain in the perineal area*) studied 132 patients and

found that 95 (72%) had transitory flare of symptoms and that 90(68%) had good to excellent results. There was no mention about length of time the symptoms were improved.

In summary the studies indicate that pudendal blocks with CT or fluoroscopic guidance can effectively alleviate PNE pain for periods up to three months with only minimal improvement thereafter. Robert's claim of a 65-70% cure is not consistent with Amarenco's 10.5% cure or near cure results. The 10%-15% cure rate would seem the most plausible after reviewing my experience and the quality of the other studies.

The question remains, can we do better? Not only with the addition of physical therapy, but with the type of injectable medication. Two possibilities that I believe are worth considering are hyaluronidase and heparin. Hyaluronidase is an enzyme that can decrease the resistance of the collagenous ground substance, therefore making it more permeable to injectable agents such as steroids. It is commonly used to diffuse IV chemotherapeutic agents that accidentally infiltrate the subcutaneous tissues. It has also been used in the treatment of post surgical epidural fibrosis and arachnoiditis . In both of these conditions the entrapment of nerves within dense scar tissue is similar to that seen with PNE. My policy is to add the

hyaluronidase to my anesthetic/steroid combination for its theoretical benefit.

Ellis has recently reported that six weekly perineural infiltrations of buffered, unfractionated heparin gave durable pain relief in 83.8% of patients with ulnar nerve, carpal tunnel and brachial plexus entrapment syndromes. Ellis stated that the idea for this technique came from background information on heparin by Engelberg and a paper by Glantz et al in which they observed that patients who were anticoagulated with heparin had a reversal of radiation induced nerve injury. The underlying theory is that glycosaminoglycans, such as heparin, have many functions of which only 3% is anticoagulation. The major effects are involved in reducing collagen cross-linking, stabilizing vascular endothelium, modulating growth factor function ( binds cytokines), and significant neuroprotection( a nerve exposed to heparin is protected against nerve damaging factors). Therefore the actions of heparin are as an anti-inflammatory, an inhibitor of fibrosis, to stabilize negative ion charges( by surrounding and sequestering them which makes the nerve less excitable), and to inhibit nerve sprouting which leads to nerve sensitisation. It is these particular functions of heparin provide the therapeutic effect on the neural irritability, fibrosis, and microvascular changes that occur in peripheral nerve entrapment. He also pointed out that

the patients were subject to re-injury if they continued their traumatic activities, and therefore those with Thoracic outlet syndrome did not do as well as the other entrapment syndromes in his study. This underscores the need to prevent perineal trauma in patients with PNE.

At this time our clinic is implementing a study using a 10.0cc mixture of 4000 units (1000 units/cc) of unfractionated heparin, 4.0cc of 0.5% Bupivacaine, 1.0cc of 1% Lidocaine and 1.0cc of sodium bicarbonate. One-half of this combination is injected around the sensitive nerve at the ischial spine, and the other half around the painful nerve in Alcock's canal. This will be repeated every one to two weeks for a total of six as outlined in Dr. Ellis's protocol.

I believe that the combination of new physical therapy techniques and injectable perineural agents will decrease the percentage of PNE patients who will require surgery.

### **SURGERY**

For those patients who have failed a conservative approach, surgery, as pioneered by Prof. Roger Robert, a neurosurgeon in Nantes, France is the final treatment option. The surgical procedure was originally done through the perineal route, but later switched to the posterior transgluteal route because it offered better exposure. With the patient in the prone position, an

incision is made in the axis of fibers of the gluteus maximus from the coccyx to a point above the ischial spine. After the posterior aspect of the sacrotuberous ligament (ST) is stripped of its muscular attachments, a 2-3 cm window is made in the ligament. The pudendal neurovascular bundle is then seen crossing behind the sacrospinous ligament (SS). The latter is divided and the nerve transposed in front of the spine to increase its length. Prof. Robert denies that the cutting of these ligaments has created any biomechanical problems. The nerve is then followed into Alcock's canal where the obturator internus fascia is incised, and the nerve trunk and its branches are freed over a 3-4 cm. length. The falciform ligament is incised if it is a site of impingement. Robert et al reported that in 150 pudendal nerve surgeries 45% considered themselves cured; 22% improved; and 33% had change in symptoms.

They found that poor results occurred in those patients who were older (average 62); had symptoms longer than 5 years; had perianal or posterior perineal pain; had a distal latency of greater than 7 msec; had a neurotic or depressive reaction; and had normal appearing nerves at surgery. On the other hand those with good results had a median age of 55; had a shorter duration of symptoms (average 4 yrs.); had a distal latency of a distal

latency of 4-7msec.; had nerves that appeared flattened; and rarely had anal pain.

They believed that failures could be attributed to insufficient decompression which would be less likely with the posterior approach; or a persistent sympathetic reflex reaction. They believe that the latter theory was confirmed when some patients were improved with paravertebral or epidural blocks. Another option that was not addressed is that a nerve compression or lesion could exist proximally at the level of the sacral nerves. This would explain the normal nerve appearance in many of the failures

In summary, PNE is probably more common than expected, and therefore requires a heightened awareness among health care practitioners. Thousands of undiagnosed PNE sufferers could be salvaged and regain some quality of life, rather than be doomed to lifelong attempts at pain management or needless surgical procedures. And the ability to help requires only a thought.