# CHRONICAL PERINEAL PAIN:
# WHAT IS IT ABOUT?

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**An overview, collaboration between the Association for Rehabilitation in Pelviperineology and Montlouis Perineal Pain Surgery**

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## INTRODUCTION

Over the past three decades, there have been significant developments in the treatment of pelviperineal pain. It has been shown that a multidisciplinary approach is essential. It involves the work of pain specialists, various organ specialists, physiotherapists and osteopaths. A better understanding of the influence of the psyche on the mechanisms of pain now makes it possible to utilise numerous complementary therapies such as sophrology, psychology, and hypnosis. Treatment has gone from having a highly focused approach, to a regional and finally a comprehensive approach.

### What we do

By chronic pelviperineal pain, we mean any pain from the pelvic or perineal regions that lasts 6 months or more, whether or not it is related to an underlying injury. In this definition we include pains that persist despite the treatment of their original cause.
This definition includes:
- **pain that can be described as injury-induced.** Injury-induced pain arises from lesion (damage) in an organ, nerve damage (pudendal or cluneal neuralgia etc.), muscle damage (piriformis syndrome internal obturator syndrome, psoas sign, etc.), or damage to the bone structure (coccyx, bone fracture or fissure).
-**These tissues can also be involved in painful phenomena without lesions being present**. This is then referred to as **dysfunction**. Pain is often experienced through an organ without it having any visible anatomical damage. This can take the form of vulvodynia or vulvar discomfort, prostatodynia, proctalgia, cystalgia, or even irritable bowel syndrome. Often, we find that these patients have experienced local irritation in the past, be it chronic, traumatic, inflammatory or infectious (recurring urinary and/or vaginal infections, endometriosis, anal fissures, recurrent haemorrhoids), which causes occasional and recurring moderate pain. Sometimes, we find that the pain only occurred once, but in a notably intense episode. Migraines, anxiety and depression, post-traumatic stress, or fibromyalgia can sometimes form part of the larger picture, but **not systematically so**.
On the pathophysiological level, these 2 mechanisms (injury and dysfunction) may be linked.

These injuries and/or dysfunctions can develop over 6 months or more due to problems with diagnosis and treatment. As time passes, they may continue to affect only one part of the body, or they may induce a reaction in a wider region or across the whole nervous system and its effectors (muscles, viscera, bones). This is known as "hypersensitization". This reaction is a response to the intensity and / or the chronic nature of the pain. The changes induced are real. They lead to impairment of physiological mechanisms of the peripheral and central levels, the brainstem and cortex. They result in an exaggerated reaction, or one that spreads through the body, responding to a stimulus which may be painful or otherwise. This can also have an impact on functions such as urination or defecation. It is no longer appropriate to think in terms of one particular nerve or muscle. The reaction can become self-sustaining, and continue whether or not the initial damage is still present. The initial damage is only a trigger or a catalyst, and patients are sometimes (but not always) predisposed, as we have outlined above (local fibromyalgia, anxiety, neurosis, other chronic pain, ...).

This situation can lead to pain syndromes in the limbs, which were formerly known as algoneurodystrophy. In English, the term "reflex sympathetic dystrophy" (RSD) was also used where autonomic signs were present. However, their inconsistent nature has led to the adoption of the term complex regional pain syndrome (CRPS). Pain may be expressed through a limb or an organ, or a region of the body.

In the perineum, this corresponds to what are referred to above as prostatodynia (or abacterial prostatitis), cystalgia, vulvodynia (without infection) or functional colopathy. They are referred to collectively as complex regional pain syndrome (CRPS). These symptoms may be isolated or linked.

To simplify, we can say that

CRPS = hypersensitization, which explains the great diversity and polymorphism of symptoms observed.

**Our approach:**

Our approach is inspired by this equation. The symptoms are listed and analysed. We refer to this as deconstructing the pattern of pain. Our goal is to identify the initial lesion (if any) - it must be treated if it is still present (nerve compression, etc.) - and to differentiate it from reactive pain, which indicates secondary hypersensitization.

Using this algorithm requires a knowledge of the descriptive anatomy of the region, its autonomic and vegetative nerve structure.

**The autonomic nervous system (ANS) and the somatic nervous system**

The whole surface of our bodies is served by autonomic and somatic nerve fibres. The same is true of our striated muscles and our joints.

***The ANS***

Anatomically, the parasympathetic system guides the nerves, while the sympathetic nervous system guides the blood vessels. The autonomic nervous system participates in the pathways of nociception mainly through the sympathetic system. However, there is a constant balance between the function of the 2 systems. Therefore, a decrease in parasympathetic tone, alongside sympathetic hyper-activity (which, like the pain, is diffuse), may help to explain some kinds of dysfunction (dysuria, erectile dysfunction, etc.) )

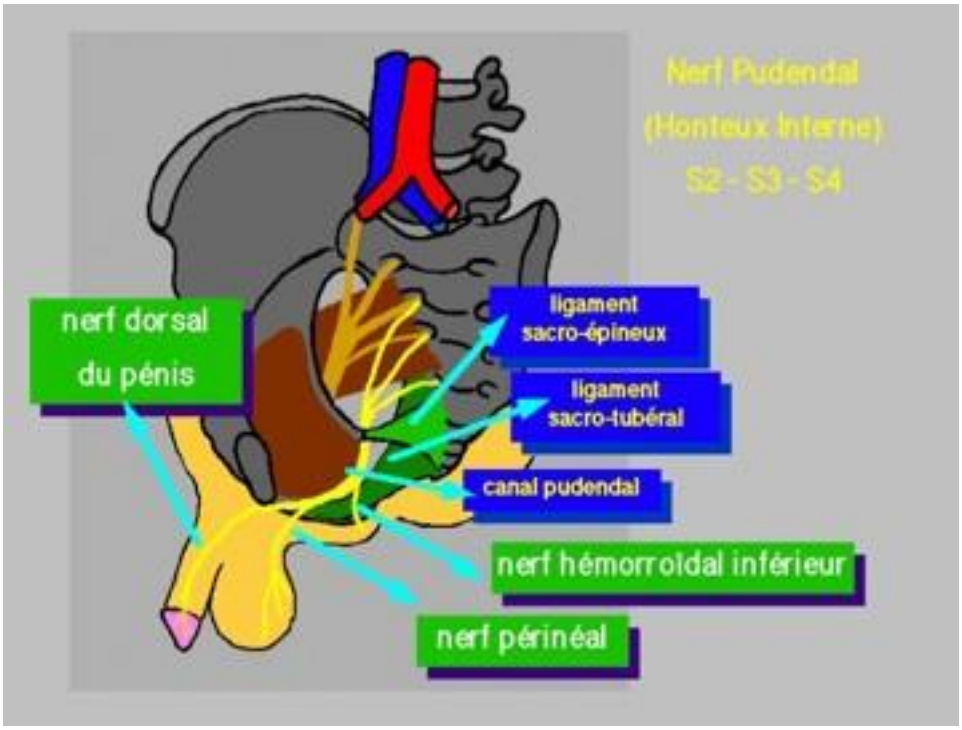
This is system is organized segmentally, like that of the somatic (voluntary) nervous system, albeit understood with less precision. Pain of the autonomic system can be

identified by its diffuse nature, described as a sensation of cold, or a sensation of awkwardness, or pain that feels like a foreign body in an orifice, or simply "unease".
This way this system is organized is partly unknown. If we start with the spinal cord, segmentally, the autonomic fibres come out from the intermediate horns, leaving the spinal cord and joining a para-spinal ganglion structure. These ganglion structures are layered, and linked by an inter-gangliar branch. They contain afferent fibres involved in nociception. They take the opposite route. From the organ , they run through a plexus (e.g. the hypogastric plexus) or a paravertebral ganglion, and then reach the spinal cord through the connecting branches. They are projected to the posterior horn. The ganglia are interlinked and located symmetrically on either side of the spine. Where they reach the sacrococcygeal region, this chain ends with a single ganglion, the impar ganglion.
Anaesthetic block tests, performed on this ganglion, show how it is involved in painful sensitivity of the skin and viscera of the whole of the pleviperineal region. Its role in the functioning of the viscera is still being explored. Destroying this ganglion chemically is effective in treating sweat disorders and cancer-related pain in the region. It automatically receives input from the para-spinal ganglion chain, but also from the pudendal nerve. We also find outgoing signals towards the lowest sacral nerve roots (S4, S5), the sacrococcygeal plexus and the coccygeal nerve.
The results of anaesthetic blocks performed in painful situations suggest that the impar ganglion is part of the nerve supply of all the surfaces of the perineum, the bladder, the urethra, the penis, glans penis, anus, rectum, and also of the sphincters of the region. Its involvement in functional disorders, hypersensitivity and pain means that we often provide a block test of the impar ganglion as part of the deconstruction and treatment of pain components.

**The somatic nervous system:**
**Figure 1: the links between somatic nerves and muscles. Canal Structures according to NETTER**

The somatic nervous system contributes to perineal pain that is sometimes caused by nerve damage or compression in the canal structures (pudendal neuralgia, cluneal neuralgia, obturator neuralgia, sub-piriformis sciatic compressions).
In the context of painful hypersensitivity, associated myofascial pain syndrome can in turn lead to irritation or compression of the surrounding nerve fibres. A knowledge of the anatomy of the nerves and nerve roots is essential, from the thoracolumbar junction to the perineum. Knowing the paths where these nerves pass anatomically, their proximity to certain muscles, and the areas for which they provide innervation, allows us to understand the mechanisms involved, as well as the extent of areas affected by pain.
In order to treat patients suffering from perineal pain, a knowledge of certain neuralgias is essential, namely pudendal neuralgia and cluneal neuralgia.

**Pudendal Neuralgia:**
**Figure 2: The conflict zones for the pudendal nerve**




It is caused by the compression of the pudendal nerve where it leaves the endopelvic zone. 3 conflict zones are identified: under the piriformis muscle, where the sacrotuberous and sacrospinous ligaments can cause clamping, and in the Alcock canal. Other elements can also complicate the path of the pudendal nerve, but for the sake of simplicity, we will limit ourselves to the 3 zones of conflict described above.
In a sitting position, these canal structures shrink and exert pressure on the pudendal nerve. On a soft seat, the fatty tissue adjoining the rectum is pushed up inwards and out to the sides. This increases the pressure around the pudendal nerve significantly. When in sitting position, these patients suffer from pain in the area served by the pudendal nerve (medial zone running from the genitals to the anus). The occurrence of pain is determined mechanically, subsiding when the patient stands or when sat on a toilet seat. The pain does not wake the patient up . Clinical assessment does not identify any objective neurological deficiency. Spinal and pelvic imaging are normal. When these 4 clinical elements are present, the diagnosis will be confirmed by performing a block (using anaesthesia) on the function of the pudendal nerve in the conflict zones (ligament clamping of the sacrospinous ligament and Alcock canal). A temporary reduction in pain of at least 50% in sitting position confirms the diagnosis. In order to avoid false negatives or false positives, these tests must be performed using a scanner, with a contrast medium, by a team trained to perform and assess such tests.
In addition to these criteria, essential for diagnosis (the Nantes criteria), there are many symptoms fostered by the chronic nature of the pain and hypersensitivity.
Medical treatment alone is sometimes able to relieve the pain, but often in a partial and transient manner. It can call for a decompression routine. No correlation has been found between the duration of compression time and the prognosis for recovery. The prognosis is probably linked to a factor which is difficult to assess: the extent of damage to the nerve. Although pudendal Injections are indispensable for diagnosis, they cannot be considered treatments (13% of patients show improvement). The use of corticosteroids has been shown not to have any additional benefit, and is no longer recommended.

Currently, the only approved treatment–is surgical decompression of the nerve, as part of a framework of comprehensive care:

- Treating the various elements of hypersensitization (muscles, using medication for blocking of pain pathways, possibly blocking the impar ganglion, and psychological treatment,)
- Treatment of the nerve injury produced by chronic compression (neuropathic pain) using medication, and stimulation of the skin.

One year after the intervention, stimulation of the spinal cord may help with residual pain, with promising results in the long term. When such stimulation is performed immediately however, there is either a transient improvement that does not last, or no improvement at all.

Figure 3: sensitive areas: A/ ilioinguinal nerve, hypogastric nerve, B/ pudendal nerve, C and D/ cluneal nerve.

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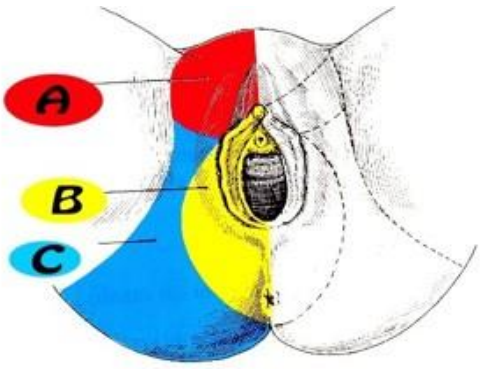
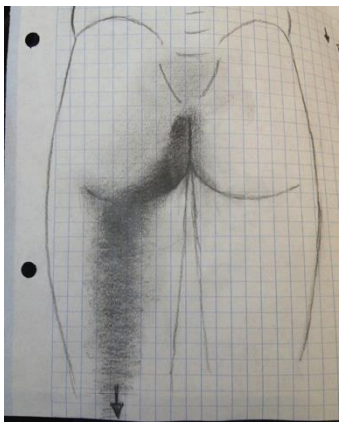


**Cluneal neuralgia**

It is the result of compression of the cutaneous posterior nerve of the buttock, or the cluneal branches, or sometimes where they pass under the lateral face of the sacrotuberous ligament, or in a canal structure where they pass along the posterior surface of the ischium.
The pain depends on position in this case too, but tends to be triggered by hard seats, and relieved with toilet seats. Patients have the sensation of sitting on their bones. The pain is observed in an area to the side, extending to the ischium, laterally to the intergluteal cleft and the posterior surface of the thigh, centrally to the region of the labia or the scrotum, or the genitofemoral groove. Pain within the ischium is reported in 80% of cases.
When these criteria are present, the diagnosis is confirmed by an anaesthetic block test along the path of cutaneous posterior nerve of the thigh. If this test is to be usable, the conditions under which it is performed and assessed are non-negotiable.
The treatment generally involves surgical decompression, as part of a framework of comprehensive care, similar to that required for pudendal neuralgia.

**Bone pain:**

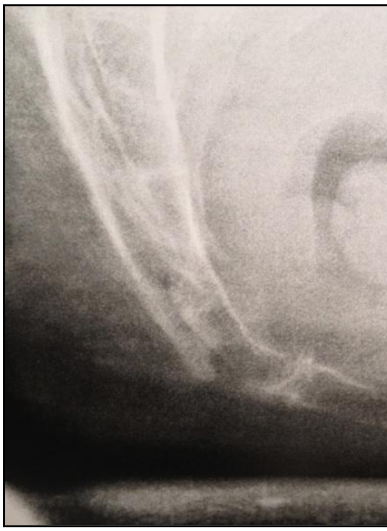
Pain arising from the bone structure is common in perineal pain. It is likely to be the result of locoregional hypersensitization. The diffuse nature of the pain indicates that the cause may be in the autonomous nervous system. In this case, a desensitization test approach can start by considering the impar ganglion.

**Coccydynia**

We will deal more specifically with the nosological framework of coccydynia.
The functional complaint is pain of the coccyx, provoked by a sitting position and more particularly by pressure on the coccyx. We also find that many patients' pain is exacerbated when they get up from a sitting position. More rarely, pain is also found in standing position and when walking. These latter characteristics result from a myofascial component involving the muscles attached to the coccyx (the coccygeus head of the levator ani, the gluteus maximus and the anal sphincter).
In 60% of chronic cases (over 3 months), the pain is the result of bone spurs, anterior or posterior dislocation, or of hypermobility in the flexion of the segments of the coccyx. Bone spurs are congenital and often appear following weight loss. Dislocations can be triggered by repetitive micro strain of the coccyx due to a loss of sagittal rotation of the pelvis. Obesity or loss of mobility in the spine (arthrodesis) are contributing factors. The other 40 % of cases include 15% incipient true dislocation or hypermobility, and around 25% reflective "referred" pain arising from the pelvis, perineum or spine. More rarely, there are microcrystalline pathologies, and true interbody ankylosing spondylitis.
Again, a comprehensive approach to treatment is essential, as taking the psychological impact of these injuries into consideration. The myofascial component should be identified and treated. Injection is beneficial for 60% to 80% of patients. It is repeated if the improvement lasts over a month.
**In our practice, we combine it with an injection of the impar ganglion. By looking for locoregional desensitization, it is possible to reduce the number of non-responders to injection and to increase the effectiveness of the treatment. Increasingly, radiofrequency treatment is proposed before moving on to surgery**.
Surgical intervention can include the resection of the element identified as being the

source of the pain (spine, coccyx). It lasts around 20 minutes. It allows a 50% or better reduction in pain in 80% of cases. The main risk, infection, is now kept under control by a specific antibiotic prophylaxis procedure.

Figure 4: hyper-mobility of flexion of the coccyx: standing (left) sitting (right)



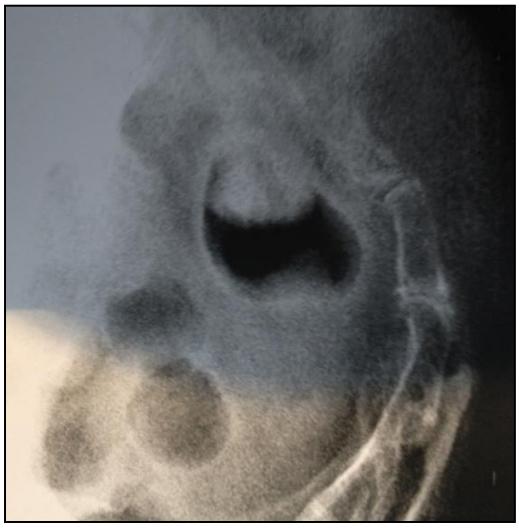
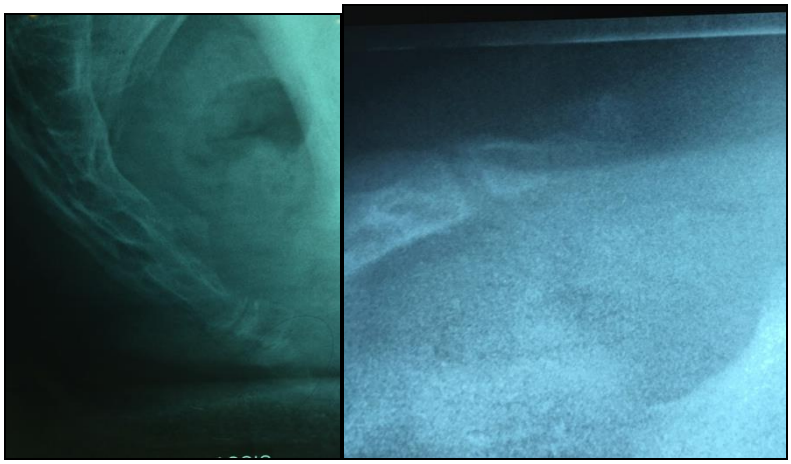


Figure 5: x-ray in sitting position: posterior dislocation (left), bone spur on the coccyx (right)



**Complex pelvic pain syndrome:**

The definition was set out at the beginning of this presentation. Roughly speaking, it includes any chronic pelvic pain that is not restricted to one organ or neurologically coherent area.

Associated autonomic signals are prevalent, (here: sensation of a foreign body in an orifice, cold buttocks, pain following defecation, urination, ejaculation or coitus). The pain is often neuropathic. Absence of an apparent or treatable cause leads us to a treatment strategy that is aimed at modulating nociception (pain pathways). This can be performed at the peripheral level or on the spine.

A medical examination performed by specialists of the relevant organ must be part of the patient's treatment path. Reference imaging of the spine and area affected by pain may be performed depending on the context. Deconstructing the pattern of pain allows us to undertake the diagnostic process (imaging, injection tests, specialist consultations, ...) and treatment with the goal of local desensitization (topical injection, nerve blocks, using radiofrequency or scanner, outpatient external cutaneous stimulation, physiotherapy), general methods of desensitization (oral medication, ketamine injection, psychological treatment) sometimes made more effective by combining both in a short hospital stay.

Peripheral stimulation is frequently used with good results. The electrodes on the skin may be positioned on the inner side of the ankle, to modulate the perineal sensory afferent (cutaneous zone served by the S3 root), or at the site of the pain. Both methods should be tried.

In the absence of success, or when there is an effect, but the electrodes on the skin become too restrictive, we can consider epidural stimulation. Depending on the improvement observed, this may be followed by implanting a device. We believe firmly that this approach should be followed in patients:

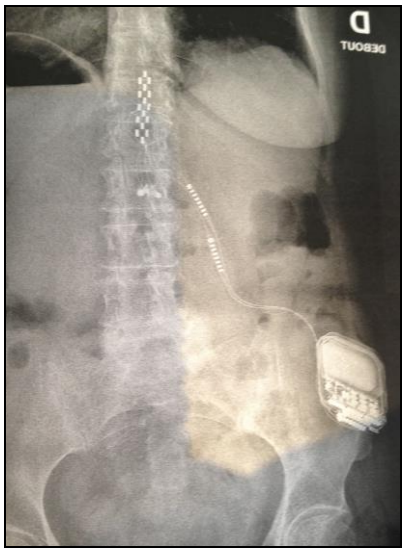
- with pain that is not sufficiently relieved by general treatments (medication) and local treatment (physiotherapy, topical treatment) performed over 1 year.
- with pain that can be classed as sequela pain.
  It serves no purpose and can be harmful if it is done instead of or before the functional treatment of an underlying causal injury.
  The development of anaesthetic techniques, and the low level of invasiveness of the routine, mean that this test can be performed with the patient's participation. Guided by the patient, the epidural electrode is then inserted, at a position that gives stimulation in the area affected by pain.

The patient is then assessed 10 days after the test in their everyday environment:

- covering at least 80% of the area affected by the pain
- pain reduction of at least 50%.

The most recent studies show that the length of time the pain has been present can be a factor in poor prognosis. This approach should therefore be at least discussed at the end of the first year after the pain starts to develop. An assessment, undertaken by a pain unit, should be performed to allow for early identification of patients who benefit from such treatment.

**Figure 6: stimulation of the conus medullaris for persistent perineal pain**



**CONCLUSION**

In current practice, comprehensive treatment is essential when dealing with chronic pain and particularly for pelvic pain. A deconstruction of the neurological, bone, and muscle components, and their integration into a nosological framework which includes the hypersensitization aspect, enables us to gain a better understanding of the pain reported by our patients. A large part of this success is the result of coordination (gynaecology / gastroenterology/coloproctology/ visceral surgery/ physiotherapy of the perineum, psychology, ...), something which continues to develop in our activities.