W22: Pudendal Neuralgia and Other Intrapelvic Peripheral Nerve Entrapment - A Neglected Cause of Pain and Pelvic Floor Dysfunction

Workshop Chair: Nucelio Lemos, Brazil
07 October 2015 16:00 - 17:30

Start	End	Topic	Speakers
16:00	16:10	Pelvic neuroanatomy and neurophysiology	Nucelio Lemos
16:10	16:20	Clinical aspects of nerve entrapment syndromes	Michael Hibner
16:20	16:30	Tissue, cellular and molecular aspects of peripheral nerve entrapment	Margot Damaser
16:30	16:45	Discussion	All
16:45	16:55	Principles and Rationale of the Treatment of Neurpathic Pain – from clinical to surgical	Michael Hibner
16:55	17:05	Laparoscopic approach to intrapelvic nerve entrapments	Nucelio Lemos
17:05	17:15	Future therapeutic perspectives	Margot Damaser
17:15	17:30	Discussion	All

Aims of course/workshop

Intrapelvic nerve entrapment is a probably underestimated source of pelvic and perineal pain. This workshop is directed to both clinicians and basic scientists interested at understanding the pathophysiology, clinical features and the therapeutic options of intrapelvic nerve entrapments, as well as discussing possible areas for research and perspectives.
The program starts with a review of the normal pelvic neuroanatomy through real surgery laparoscopic dissections and the neurophysiological aspects of these structures. After this introduction, the clinical features of nerve entrapment syndromes will be explained, followed by the cellular and molecular basis for these symptoms.
On the second half of the workshop, medical treatment guidel

Learning Objectives

1. Identify and intrapelvic radiculopathy and to differentiate it from other causes of pelvic and perineal pain

2. Initiate medical treatment of intrapelvic radiculoptahies and understand the principles of surgeries for nerve detrapment

3. Understand the cellular mechanisms related to nerve entrapment syndrome and the future perspectives for this clinical entity

Pelvic Neuroanatomy & Neurphysiology

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Doctorate in Gynecology by FCM Santa Casa SP
Fellowship in Neuropelveology by the International School of Neuropelveology, Klinik Hirslanden, Zurich
Post-Doctorate Researcher of the Pelvic Neurodysfunctions Clinic of the Department of Gynecology of the Federal University of São Paulo
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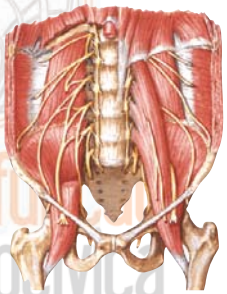
Financial Disclosures

Nucelio Lemos, MD PhD

- Speaker/Proctor
  - Medtronic®
- Research Grants
  - Medtronic®
  - Laborie®

Lumbar Nerves

- Iliohypogastric N.
- Ilioinguinalis N.
- Genitofemoralis N.
- Femoral N.
- Obturator N.



Sacral & Coccigeal Nerves

- Superior Gluteal N.
- Inferior Gluteal N.
- Post. Cutaneous Femoralis N.
- Sciatic N.
- Pudendal N.
- Nn. to the Levator Ani Mm.

Somatic Nerves of the Pelvis

Video showing the dissection of the nerves of the obrturator space

Sensitive Innervation

Motoric Innervation

- L2/L3 - Hip flexors (ilipsoas)
- L3 - Hip adductors
- L3/L4 - Knee extensors (Quadriceps)
- L5 - ankle dorsiflexion, eversion and inversion + hip abductors
- S1 - ankle plantar flexion + hip extensors
- S2-S4 - External anal and urethral sphincters

Autonomic Nerves

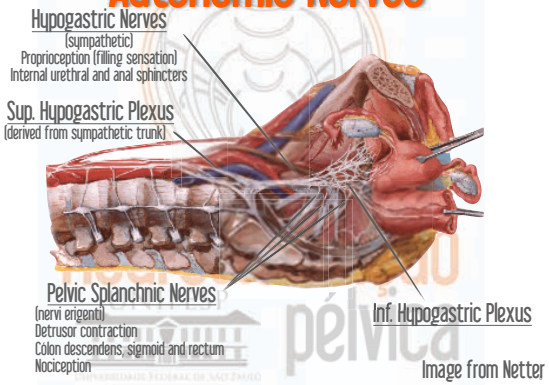
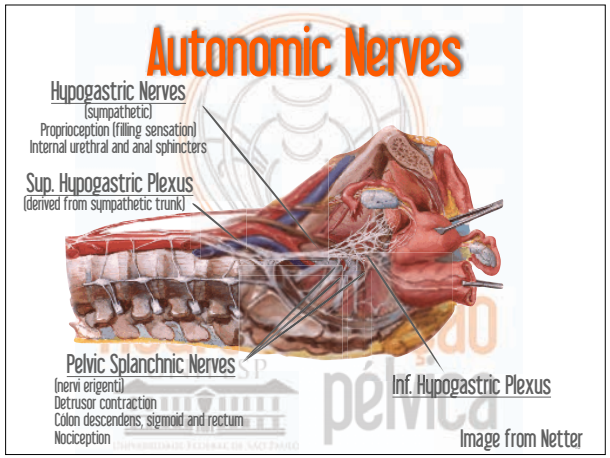


Image from Netter

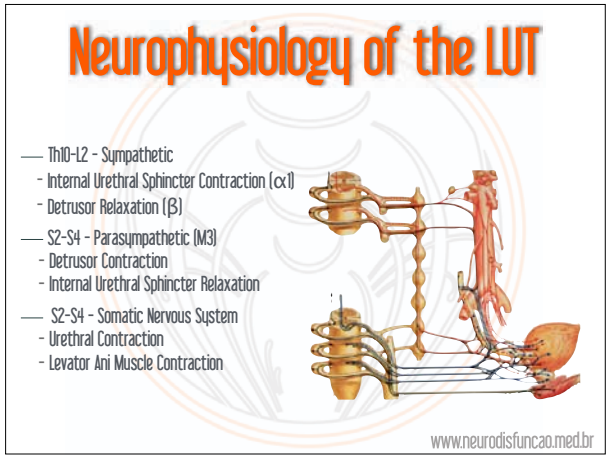
Hypogastric Nerve

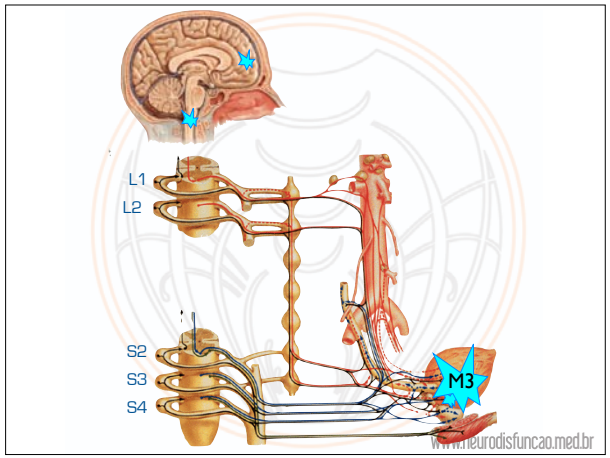
www.neurodisfuncao.med.br

Video demonstrating the anatomy of the hypogastric nerves



Video showing the anatomy of the sacral nerve roots and the pelvic splanchnic nerves





Animation summarizing the pathways of bladder sensation and the micturition reflex

In Conclusion...

In Conclusion...

The abdominopelvic retroperineal spaces house a large number of nerves

In Conclusion...

The somatic ones are those of the lumbosacral plexus, which control the striated muscles of the perineum, pelvic floor and inferior limbs

In Conclusion...

The autonomic ones are those of the hypogastric plexuses, which control the urinary, bowel and sexual functions and carry sensitive impulses from the pelvic viscerae

In Conclusion...

The laparoscopic approach offers optimal lighting and visualization of these nerves, while the pneumoperitoneum reduces the risk of capilary bleeding and facilitates dissection.


Thank You!
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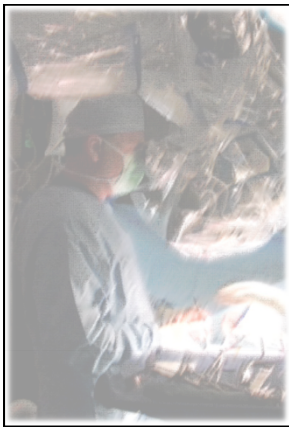

Arizona Center for Chronic Pelvic Pain

THE UNIVERSITY OF ARIZONA COLLEGE OF MEDICINE PHOENIX

Creighton UNIVERSITY School of Medicine

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Principles and Rationale of the Treatment of Neuropathic Pain from clinical to surgical

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Neuropathic pain

- Pain caused by a lesion or disease of somatosensory system
- Affects 8% of population

Pathophysiology of nerve injury

- Compression
- Transection
- Contusion
- Stretch
- Crush

Etiology pelvis

- **Blunt Trauma**
  - Falls
  - Car accidents
  - Pelvic fractures
- **Obstetrical**
  - Prolonged second stage of labor
  - Positioning
  - Traumatic Delivery
- **Surgery**
  - Incisions
  - Compression from retractors
  - Stretching from positioning
  - Mesh
- **Radiation Therapy**
  - Fibrosis
- **Medical conditions**

Mechanisms

- Ectopic activity
- Peripheral sensitization
- Central sensitization
- Impaired inhibitory modulation
- Activation of microglia

Diagnosis and treatment

- Diagnosis and treatment of neuropathic pain has to be done in timely fashion to minimize central and peripheral sensitization

History

- Description of injury
- Description of distribution of motor, sensory and autonomic changes
- Pain quality
- Additional pain generators
- Palliative and provocative positions

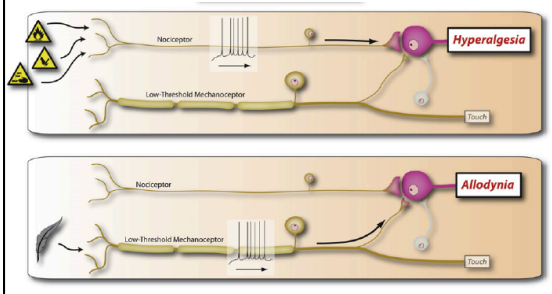
Symptoms

- Tingling (“pins and needles” or “prickling”)
- Burning (“hot”)
- Shooting (“electrical shocks”)

Signs

- Hypoesthesia (abnormally reduced sensation to touch or cold)
- Hypoalgesia (abnormally reduced pain sensation to noxious stimulus)
- Hyperalgesia (abnormally increased sensation to noxious stimulus)
- Allodynia (pain sensation to a nonnoxious stimulus)

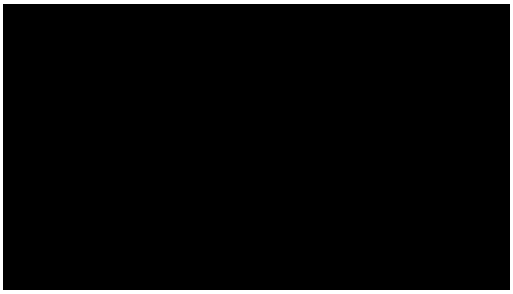
Mechanism of pain



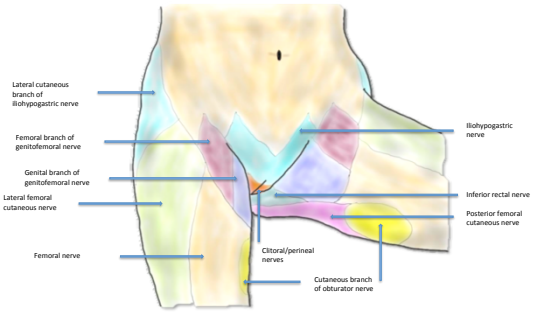
Exam – fiber type

- Aβ(touch) – finger, cotton swab, brush
- Aδ(fast pain) – metal pin or sharp stick
- C(slow) – warm 40°C object

Cotton swab test



Pelvic nerves



Exam

- Detailed neurological exam testing sensory, motor and autonomic fibers
- Hypo and hyper sensitivity, allodynia
- Tinel's sign – percussion tenderness over affected nerve – distal migration of axonal cone

Additional studies

- MRI
- EMG
- Nerve conduction studies/PNMTL
- Nerve blocks

Basic principles of nerve blocks

- Pain must be present
  - Patients must have pain at the time of the injection
- Evaluated for technical success
  - Anatomical position, diffusion of solution, and achieved analgesia
- Interpretation
  - Relief of symptoms, the specificity of the block, and the possibility of placebo effect

Materials for nerve blocks

- Local anesthetic
  - Lidocaine 1 – 2% with epinephrine
  - Bupivacaine 0.5% with epinephrine

- Sodium Bicarbonate 8.4% (10:1 ratio)
- Three to five milliliters, to minimize spread
- Image guidance (ultrasound, CT, etc…)

Treatment – non surgical

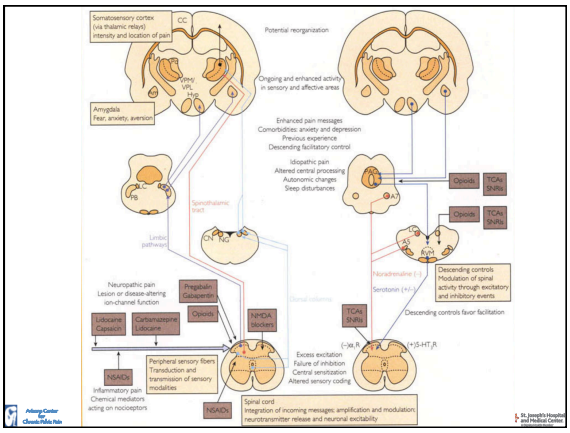
- Multidisciplinary approach
- Avoidance of offending factors
- Pharmacotherapy
- Physical therapy
- Holistic treatments – acupuncture
- Psychological – counseling, biofeedback
- Botox and steroid injections

Drug therapies

- Antidepressants
- Anticonvulsants
- Local anesthetics
- BMDA receptor antagonists
- Opioids
- Cannabinoids
- Botulinum toxin
- Topical capsacin

Drug therapies

Drug	Total daily dose and dose regimen	Recommendations
Strong recommendations for use		
Gabapentin	1200-3600 mg, in 3 divided doses	First line
Gabapentin extended release or enacarbil	1200-3600 mg, in 2 divided doses	First line
Pregabalin	300-600 mg, in 2 divided doses	First line
Serotonin-norepinephrine reuptake inhibitors duloxetine or venlafaxine[b]	60-120 mg, once a day (duloxetine); 150-225 mg, once a day (venlafaxine extended release)	First line
Tricyclic antidepressants	25-150 mg, once a day or in 2 divided doses	First line[c]
Weak recommendations for use		
Capsaicin 8% patches	One to 4 patches to the painful area for 30-60 min every 3 mo	Second line (peripheral neuropathic pain)[d]
Lidocaine patches	One to 3 patches to the region of pain once a day for up to 12 h	Second line (peripheral neuropathic pain)
Tramadol	200-400 mg, in 2 (tramadol extended release) or 3 divided doses	Second line
Botulinum toxin A (subcutaneously)	50-200 units to the painful area every 3 mo	Third line; specialist use (peripheral neuropathic pain)
Strong opioids	Individual titration	Third line[e]



Treatment - surgical

- Neurolysis
- Neurectomy
- Nerve repair/reconstruction

Mechanism of injury

- Compressive injuries occur when nerves are subjected to repetitive low impact forces leading to structural or functional damage

Pathophysiology

- Interruption of microvascular flow leading to ischemia and edema
- Alteration of axonal transport aggravating nerve dysfunction

Points of entrapment

- Static
  - Within rigid fibro-osseus tunnel
  - Between ligaments
  - Scarring in or apart of the tunnel
- Dynamic
  - Narrowing of the nerve from muscle contractions
  - Angulation during positioning

Decompression/Neurolysis

- Freeing the nerve at the point of compression or modification of the environment around the nerve may improve nerve function and decrease pain symptoms

Decompression/Neurolysis

- Freeing the nerve
- Unroofing the nerve from compressive ligament
- Dissection preformed outside the epineurium

Decompression/Neurolysis

- Obtain proximal and distal control of normal nerve
- Identify and release the nerve from any exterior scar, points of tethering or abnormalities
- Ensure good vascularized bed with vasularized flaps(fat flap)
- Nerve wraps and amniotic membrane*
- Promote early movement to ensure gliding

Neurectomy

- For patients who have pain of purely sensory nerves
- Numbness is permanent but diminishes over 1 year as surrounding nerves take over
- Risk of stump neuroma (tangle of regenerating axons and Schwann cells without end destination)
- Implantation into the muscle may diminish the risk of neuroma formation

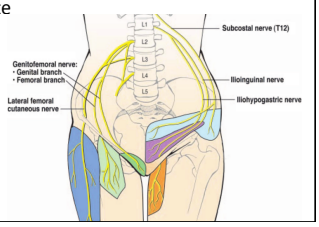
Outcomes

- Neurolysis – 70-88%
- Neurectomy – 64-75%

SELECTED NERVES

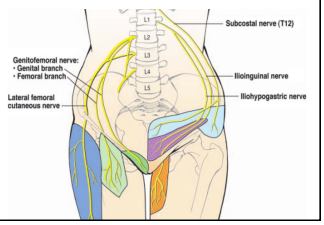
Iliohypogastric nerve

- Originates from L1 ventral ramus
- Sensory:
  - Posterior lateral glute
  - Suprapubic skin
- Motor:
  - Transverse abd.
  - Internal oblique



Genitofemoral nerve

- Originates from L1-L2 ventral nerve roots
- Divides into genital and femoral branches
- Sensory:
  - Labium majus
  - Anteromedial thigh
- Motor:
  - Cremaster

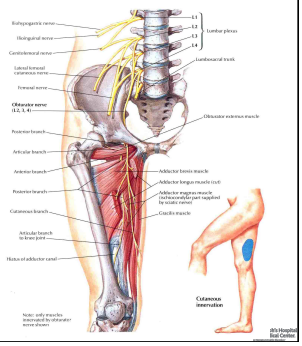


Posterior cutaneous femoral nerve

- Emerges from S1-4
- Sensory:
  - Inferior buttocks
  - Lateral perineum
  - Proximal medial thigh
  - Labia majora
  - Clitoris
- Often confused with the pudendal nerve

Obturator nerve

- Emerges form L2-4
- Sensory
  - Medial thigh
- Motor
  - Pectineus
  - Abductor longus/brevis
  - Gracillis



Tissue, Cellular and Molecular Aspects of Peripheral Nerve Entrapment

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Take Home Points:

- There is very little research on pelvic nerve entrapment, and none on basic science aspects
- Research in other fields can elucidate the major points
- The most common nerve entrapment syndrome is carpal tunnel syndrome so this is used as an example
- Animal models have been developed based on these mechanistic theories and are used to investigate the detailed molecular mechanism of injury and potential pathways to facilitate recovery
- Molecular mechanisms of nerve dysfunction in nerve entrapment syndromes appear to be distinct from those in acute nerve injuries, such as crush or transection, and therefore development of therapeutic pathways should be different as well.

Neuroanatomy

Most nerves, including the pelvic and pudendal nerves, consist of hundreds or thousands of axons, both myelinated and unmyelinated, both sensory and motor. The axons are surrounded by the endoneurium and are then grouped together in fascicles, each of which is surrounded by the perineurium. The entire nerve is then embedded in the epineurium (1) Small blood vessels supply the nerve from the surrounding tissues and do not tether the nerve since they are coiled to enable

additional flexible with joint movement and bending (2). Myelinated fibers are surrounded by the myelin sheath which is created by Schwann cells. A single Schwann cells will associate with a single segment of a myelinated axon and will extend cytoplasmic processes around it to create the internode and the myelin sheath. Interrupting the internodes at regular intervals are the nodes of Ranvier, areas of the axon devoid of myelin and rich in voltage-gated sodium channels (3). Between each internode and its adjacent nodes of Ranvier lie the paranodes and juxtaparonodal areas of the axons. Juxtaparanodes are high in voltage-gated potassium channels, which in concert with the voltage-gated sodium channels enable action potentials to propagate down the length of the axon via salutatory conduction (4).

Hypothesized Mechanism of Injury in Nerve Entrapment

The leading hypotheses to explain the mechanism of injury in nerve entrapment syndromes is that localized pressure or compression leads to ischemia and nerve damage. Pressures of 20-30 mmHg interfere with venous return flow while pressures of 35-50 mmHg reduce capillary flow. Pressures exceeding 70 mmHg cause complete ischemia (5). A 4 hour period fo only 30 mmHg pressure on a nerve begins to show disease process with increased vascular permeability and edema lasting 24 hours after removal of the compression. Increasing either the pressure or its duration increases the resultant edema and other pathophysiologies in a dose-response fashion. This injury can be compounded with time by tethering of the nerve due to scar tissue that develops from the inflammatory response to the ischemia. Tethering of the nerve can add additional ischemic injury and nerve damage to the entrapped nerve (2).

The mechanism of nerve dysfunction due to entrapment or compression injury can be detailed further by investigating the changes to myelin in the course of the disease process. Compression or entrapment nerve injuries are different from acute, severe crush or transection injuries, in that they are slowly forming. Their pathophysiology is significantly different also since nerve dysfunction due to compression injury is from gradual demyelination of myelinated nerves followed by subsequent remyelination of the compressed nerve (3). In an ongoing disease

process, both of demyelination and remyelination are ongoing simultaneously, potentially in different portions of the axon or nerve.

Demyelination begins with Schwann cell proliferation at the periphery of the nerve approximately 2 weeks after the compression injury, prior to measurement of nerve dysfunction, although onset and duration of the injury process is dose-dependent in terms of the pressure applied to create the nerve compression (3). Demyelination begins at the paranode and progresses toward the internode where chronically, regions of thin myelin can be found interspersed between regions of normal myelin, resulting in mylinated regions, or internodes, of shorter length (6). Schwann cells downregulate myelin-associated protein (MAG), enabling axonal sprouting which is also observed after compression injury (7). GAP-43 is upregulated in sensory nociceptive neurons, which begins the process of pain development from nerve entrapment. This process begins early in the injury development, prior to abnormalities to motor neurons (8). This slow process of Schwann cell proliferation with demyelination and remyelination is likely initiated by the direct response of Schwann cells to mechanical shear stress via integrin signaling and is distinct from the injury process of acute nerve injuries which is driven by a massive wave of macrophage recruitment to the injured area (3).

Animal Models of Nerve Entrapment

Acute compression injuries have been studied using the rat sciatic nerve as a model since it is relatively large and easily dissected. Inflatable cuffs placed around the nerve have been used with pressures up to 80 mmHg and induce inflammation and fibrin deposits within hours with marked fibrosis within a month. Demyelination and axonal degeneration occurred 1 week after compression and proceeded in a dose-dependent fashion based on the pressure used to induce the injury and the duration of the imposed pressure (2).

Chronic compression models have been created using silicon tubes secured around the rat sciatic nerve to induce slow compression injuries from fibrosis occurring around the tube. Pain and histological changes occur after 1-3 months in this model. Other models have used a balloon catheter placed in the carpal tunnel of a rabbit and then inflated to 40-80 mmHg (2).

Lessons from Animal Models

Mechanisms of injury are difficult to impossible to determine in human studies due to the difficulty of obtaining tissue and conducting controlled repeatable experiments. This is particularly true in nerve entrapment studies in which removal of a piece of the nerve for study is usually considered unethical as it will detrimentally affect the course of recovery. Therefore animal models are key to determining the cell and molecular basis for pathophysiology and also for proposing and testing potential pathophysiologically-based therapies. From the animal models on nerve compression it was determined that the injury process leading to nerve dysfunction and pain in compression injuries differs from that in acute injury and begins with Schwann cell proliferation and demyelination in the paranodal regions of myelinated axons. Novel therapies could be designed specifically to interrupt these pathophysiological processes and regenerate normal nerve.

References

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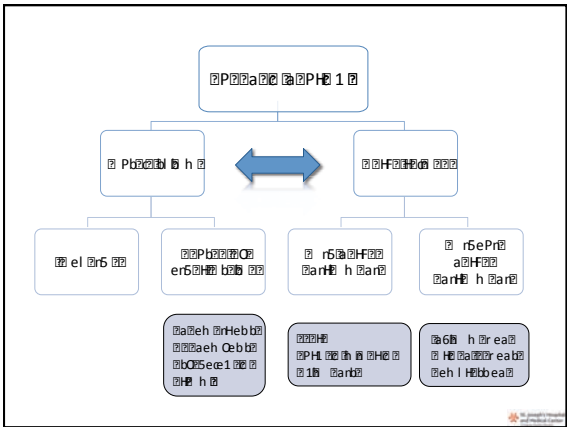
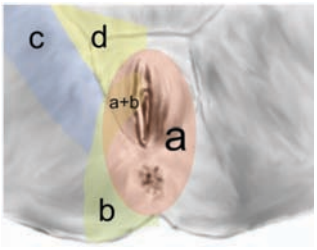


Pudendal neuralgia

- Painful condition in the area of innervation of the pudendal nerve
- Pudendal nerve entrapment (PNE) is compression of the pudendal nerve by scar, ligaments or surgical material **causing** pudendal neuralgia



a- pudendal n. b- inferior cluneal n.
c- obturator n. d- genitofemoral n.

Symptoms

- Pain in the area of innervation of the pudendal nerve
- Pain is neuropathic in nature
  - Paresthesia – burning, tingling, prickling, numbness sensation
  - Allodynia – pain in response to non painful stimulus
  - Hyperalgesia – pain out of proportion to the stimulus
- Pain is more severe with sitting
- Pain absent or significantly less when lying down
- Pain less when sitting on the toilet vs. chair
- Sensation of foreign body in the rectum or vagina (allotriesthesia)*

Symptoms

- Urinary symptoms – frequency, urgency, hesitancy
- Dyschesia
- Dyspareunia
- Pain with orgasm
- Pain with sexual arousal
- Persistent sexual arousal

Numbers

- Incidence 1/100,000 (tipna.org)
- 4% of patients with pelvic pain (orpha.net)
- 70/30 women/men ratio
- 70% unilateral

Causes

- Caused by injury to the pelvic floor by:
  - Surgery
    - Direct mesh injury
    - Indirect – hysterectomy, cystocele repair, prolapse repair
  - Vaginal childbirth
  - Trauma
    - Falls
    - Cycling
    - Intense lower extremity exercise (abductor machine)
    - Excessive masturbation
    - Excessive use of anal vibrators

Diagnosis

- History
  - Make sure that onset of pain coincides with traumatic events
  - If no traumatic event PFTM more likely
  - If bilateral pain PFTM more likely
  - In addition patient may have pain outside the distribution of the pudendal nerve (lower back, anterior and posterior thighs)
- Exam
  - Significant tenderness to palpation along the course of Alcock□s canal (vaginal)
  - Palpation of the course of the nerve reproduces symptoms (Tinel□s sign)

Diagnosis - exam

THE INTERNATIONAL PELVIC PAIN SOCIETY

http://www.pelvicpain.org

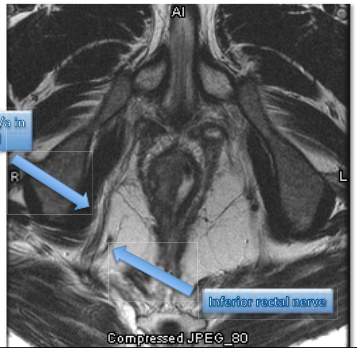
Section on Women's Health APTA

http://www.womenshealthapta.org

Diagnosis

- Pudendal nerve motor terminal latency (PNMTL)
  - Unreliable in multiparous patients
  - High interobserver and intraobserver variability
- Sensory threshold testing
  - Warm detection threshold testing
  - Two point discrimination testing
- MRI
  - Anatomical MRI
  - Functional MRI (MR neurography)
- Diagnostic CT guided pudendal nerve block
  - Patients must have at least temporary relief of pudendal neuralgia (part of Nantes criteria)

PN MRI



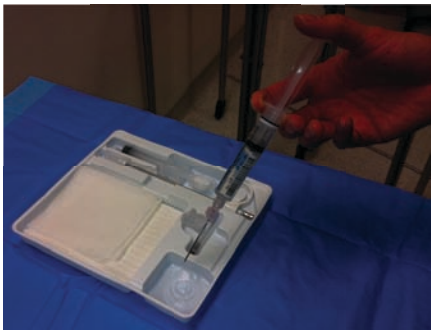
Treatment

- Self care – avoidance of pain, use of sitting support
- Pelvic floor physical therapy
- Oral medications
  - Gabapentin (Neurontin) up to 2400 mg/day
  - Pregabalin (Lyrica) start at 75 mg BID up to 600 mg daily
  - Amitryptiline 25-50 mg/day
  - Duloxetine (Cymbalta)
  - Appropriate pain management (narcotics)
- Suppositories
  - Belladonna and Opium 16.2/30 mg rectal suppository BID
  - Diazepam 5 mg/Baclofen 4 mg vaginal suppository BID

Treatment

- Botulinum toxin A (Botox)
  - Done under anesthesia/sedation
  - Examine patient prior to sedation to identify most tender areas
  - After sedation do pudendal nerve block with 0.5% Bupivacaine with epinephrine
  - Dilute 200 units of Botulinum toxin in 20 ml of NS
  - Inject using pudendal nerve block needle at volumes 1 ml per injection deep into levator and obturator muscles
  - Usually patients start feeling relief from Botox about a week after the injection. If no relief and muscles feel relaxed pain is most likely due to nerve injury, not muscle spasm

Treatment



Treatment

Treatment

- Therapeutic CT guided pudendal nerve block
  - Bupivacaine 0.5% with epinephrine
  - Triamcinolone (Kenalog) 80 mg (40 mg per side if bilateral)
  - Injections repeated every 6 weeks (3 total)

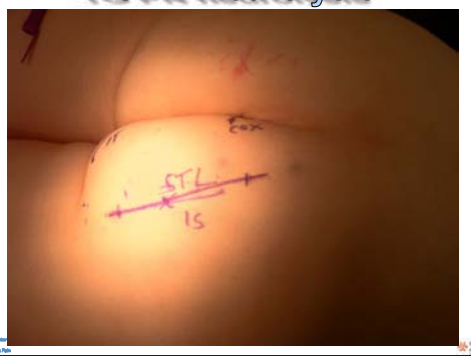
CT guided block

Treatment

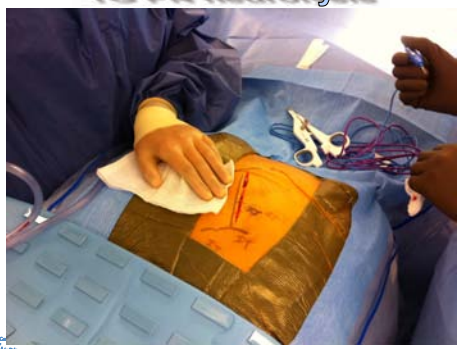
- When all the conservative measures have failed, surgery is the only other option
- Prior to offering surgery it is important to:
  - Make sure this pain is not caused by muscle spasm
  - Determine location of injury (intrapelvic vs. extrapelvic)

TG PN neurolysis

TG PN neurolysis



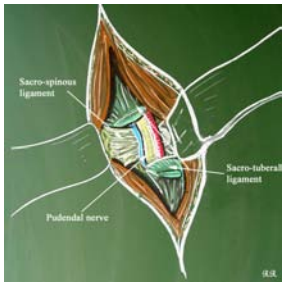
TG PN neurolysis



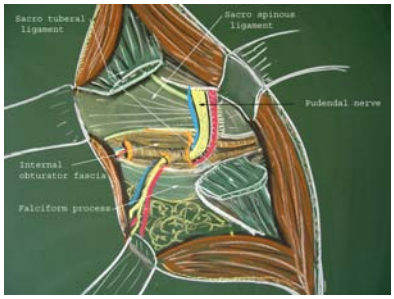
TG PN neurolysis

TG PN neurolysis

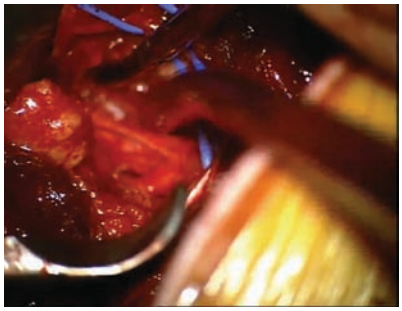
TG PN neurolysis



TG PN neurolysis



TG PN neurolysis



TG PN neurolysis
(Phoenix modification)

- D 5h an e HenP HePb 1h an
  - F H LH cc ba ea LH
  - r5eP n5 LH u l c bnO
- b e af a l Ph l e bnel H F cOg A i by
- PH a a H-F l He n ne Hg h a er h h H a y
- r Fa l a c n H-5 l d h
- ePa F ea ce b bi a

Postoperative care

- Avoid activities causing pain
  - No prolonged sitting
  - No squatting
- Continue physical therapy
- Continue medications
- Some patients will benefit from additional injections of Botulinum toxin A or nerve blocks

Outcomes

- Hon h l HeF h an a l a z h ear5b
- h Ph h l HeF h an a l a pl h ear5b
- bPonb A eHb a PH 1 E pJ O Hb

Outcomes 2009-2012
Do and Redo surgery combined

pJJ [illegible]

- [illegible] mxN
- [illegible] xLN

If surgery fails

- [illegible]
  - [illegible]
- [illegible]
  - [illegible]
  - [illegible]
- [illegible]
  - [illegible]

Why does surgery fail?

- [illegible]
- [illegible]
- [illegible]
- [illegible]
  - [illegible]
  - [illegible]
- [illegible]

The Laparoscopic Approach to Intrapelvic Neuropathies

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Doctorate in Gynecology by FCM Santa Casa SP
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Post-Doctorate Researcher of the Pelvic Neurodysfunctions Clinic of the Department of Gynecology of the Federal University of São Paulo
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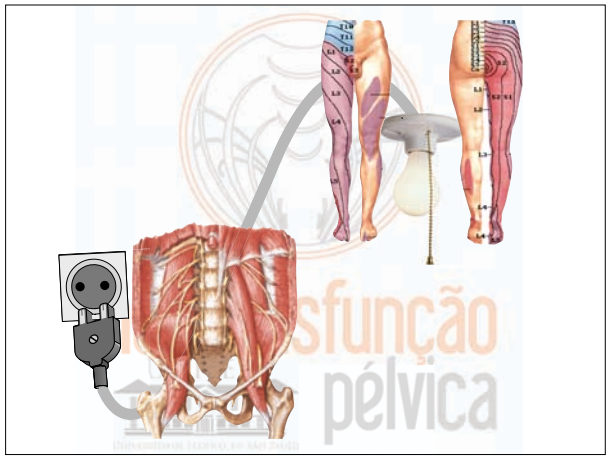
Financial Disclosures

Nucelio Lemos, MD PhD

- Speaker/Proctor
  - Medtronic®
- Research Grants
  - Medtronic®
  - Laborie®

Syndromic Diagnosis

Nerve entrapment syndrome, or compression neuropathy, is a clinical condition caused by compression on a single nerve or nerve root. Its symptoms include **pain**, **tingling**, **numbness**, and **muscle weakness** on the **affected nerve's dermatome**.



Animation explaining the rationale of dermatomes on topographic diangosis

Symptoms

- Gluteal/Perineal/Lower Limb Pain/Alodynia
- Vaginal/Retal Foreign Body Sensation
- Refractory Urinary Urgency
- Dischezia
- Proctalgia
- Vesical/Retal Tenesmus

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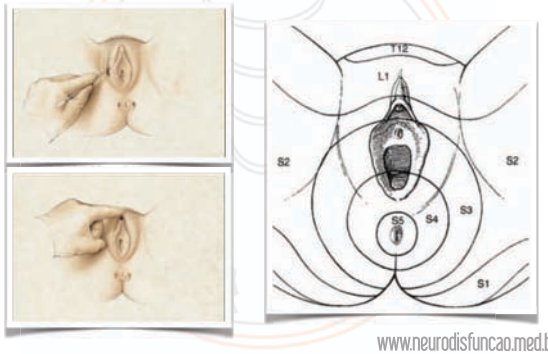
Signs



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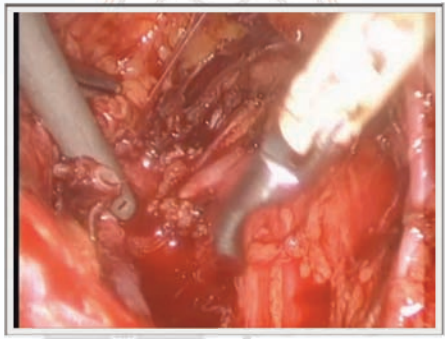
Endometriosis



Video demonstrating the treatment of an endometrioma in S3

Endometriosis

- Symptoms
  - Cyclic Pain – continuous or perimenstrual
  - Motoric Deficit ± (Foot Drop)

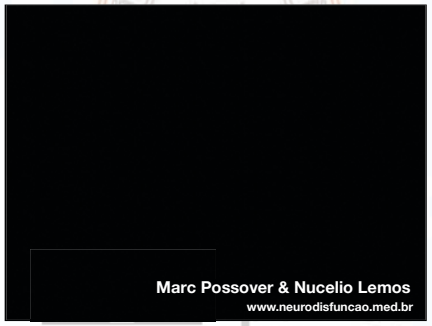
Fibrosis

- Surgical manipulation
- Delivery
- Hematomas
- PID
- Sutures
- Grafts
- Anorectal Abscess
- Continued Trauma (i.e. cycling)
- Endometriosis

Fibrosis

Video - removal of endometriosis-induced fibrotic tissue

Fibrosis



Video - removal of hematoma-induced fibrotic tissue

Grafts/Sutures

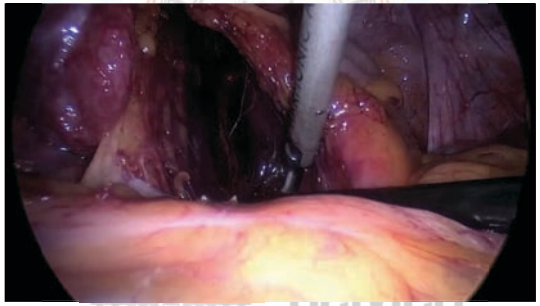
Video - removal of a McCall suture from S2 nerve root

Fibrosis

Symptoms

- Continuous Pain
- Varying Intensity
- Allodynia
- Trigger Point

Vascular Entrapment



Video - removal of varicose entrapping S2-S4 nerve-roots

Vascular Entrapment

- Symptoms
  - Cyclyc ♀ / Continuous ♂
  - May worsen on exercise
  - LUT and anorectal symptoms

Muscular Entrapment

Video - removal of pyriformis muscle fibers entrapping S2-S3 nerve-roots

Muscular Entrapment

Symptoms

- Symptoms are triggered by a specific movement
- Pyriformis Syndrome – hip adduction and flexion

Nerve Transection

- Laparotomy
  (iliohypogastric, ilioinguinalis)
  - Incision
  - Retractors
- Laparoscopy
  (iliohypogastric, ilioinguinalis)
- Episiotomy
  (pudendal nerve rami)
- Lower Limb Amputation
  (cSciatic e femoralis)

Neuroma

Nerve Transection

Symptoms

- Continuous intense pain
- Anesthesia and muscular atrophy on the nerve territory
- Phantom pain

Diagnosis

- Proximal anesthetic block

Nerve Transection

Autonomic Nerve

- Bladder Hypo/Atonia
- Intestinal Hypo/Atonia

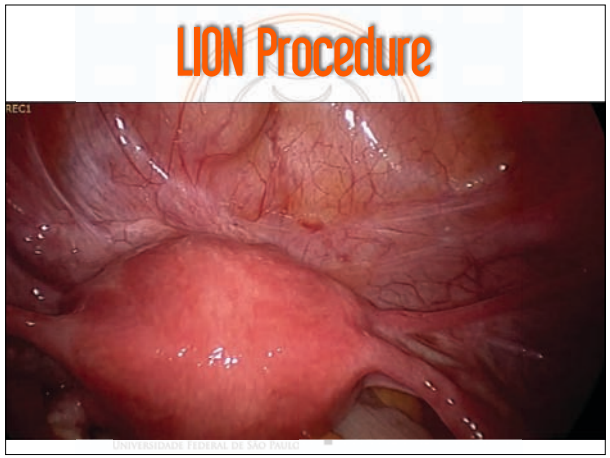
Diagnosis

- Urodynamics
- Rectal Manometry
- Electrophysiologic Study of the Pelvic Floor

Nerve Transection

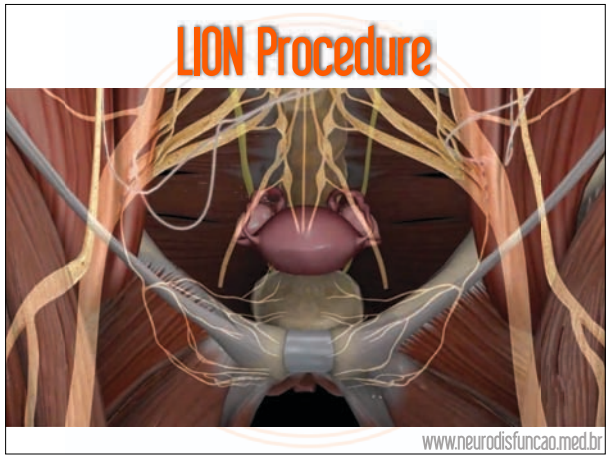
Treatment

- Botulinum toxin
- Neuromodulation



Video demonstrating the laparoscopic implantation of neuromodulation electrodes

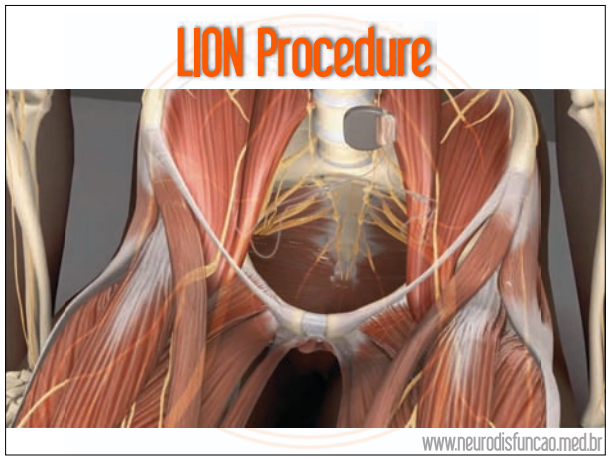
Animation demonstrating the position of the LION electrodes



Animation demonstrating pudendal nerve stimulation

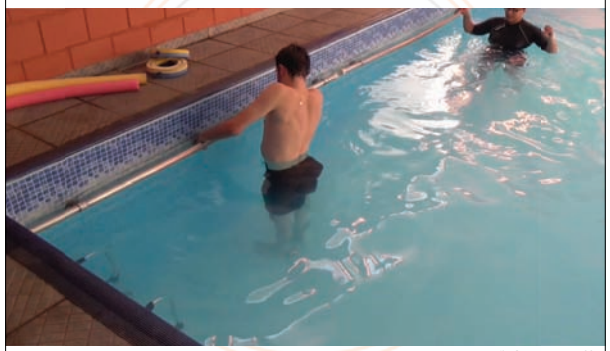
Animation demonstrating femoral nerve stimulation

Video demonstrating femoral nerve stimulation in a SCI patient



Animation demonstrating sciatic nerve stimulation

LION Procedure



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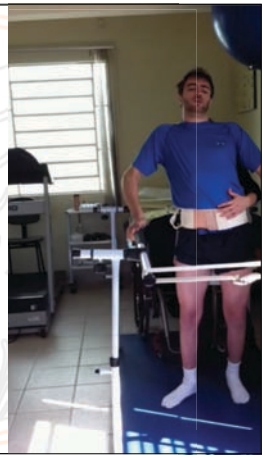
Videos demonstrating orthostatic training in SCI patients

LION Procedure



Videos demonstrating orthostatic training in SCI patients

LION Procedure

INDICATIONS

- Neuropathic Pain
  - Phantom limb pain
  - Post-herniorhaphy inguinidynia
  - Chronic Pelvic Pain
  - Perineal Pain
  - Ciatica
  - Post-decompression pain
- Anal Incontinence
- Detrusor Overactivity
- Rehabilitation

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Thank You!
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Notes