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Spondylogenic diseases, back pain

Josef Bednařík

General neurology questions:

26. Spine examination

27. Segmental, pseudo-radicular and radicular (C6-8, L3-5, S1) syndromes

28. Epiconus, conus and cauda equina syndrome

Special neurology questions:

14. Back pain syndromes: acute, chronic, neck pain, thoracic pain, low back pain - diagnosis, symptomatology

15. Spondylogenic radiculopathies and myelopathies: diagnosis, symptomatology

16. Treatment of back pain syndromes (pharmacologic, physiotherapy, surgery)

Epidemiology

- 1st Most frequent cause of working disability in people younger than 45 years
- 2nd Most frequent cause to visit the doctor
- 3rd Most frequent cause of surgery
- 5th Most frequent cause of hospital admission

Epidemiology

- 1% of population is on sick leave
- 10-15% of sick leave days
- 1% of population is permanently disabled

How to classify spondylogenic diseases?

- According to the character of structural changes
 - Degenerative changes (spondylosis)
 - Non-degenerative structural changes (tumor, trauma, inflammation, osteoporosis, maldevelopment)
 - Non-structural „functional“ changes
- According to clinical manifestation
 - Back pain syndrome
 - Pseudoradicular syndrome
 - Radicular syndrome
 - Myelopathy
 - Cauda equina syndrome

How to classify spondylogenic diseases?

According to involved part of the spine

- cervical
- thoracic
- lumbosacral

According to aetiology

- developmental (congenital)
- trauma
- infection
- tumors
- metabolic (osteoporosis)
- physical overload (occupational, sports)

How to classify spondylogenic diseases?

It is not possible to establish reliably the etiology of s.c. simple back pain attacks in up to 85% of cases.

It seems to be useful and pragmatic to classify spondylogenic syndromes according to clinical manifestation a try to establish etiology.

Diagnostic algorithm in acute back pain: triage based on different prognosis and different management

There exists a dilemma, how on one side **not to burden** a lot of patients with otherwise benign and self-limited conditions with sometimes risky diagnostic procedures and not negligible side effects (and with respect to high frequency of these patients also not to increase economical burden of health care system), and on the other side **not to postpone** causal treatment in a small group of patients with potentially threatening disease that may lead to serious consequences.

Diagnostic algorithm in acute back pain: triage based on different prognosis and different management

Possible solution is an entry **„*triage*“** (assortment), which could be performed by a physician of the first contact (G.P.) in a patient suffering from acute back pain. The triage is based on taking a history, a basic neurological examination and on identification of „risk factors“ increasing probability of serious structural spine disease or damage - (**„*red flags*“**).

Diagnostic algorithm in acute back pain: triage based on different prognosis and different management

The triage could differentiate 3 big groups of acute back pain with different prognosis and necessity to differentiate diagnostic-therapeutic approach:

A. Up to 85% of acute back pain patients belongs to **non-specific, „simple“ back pain**, whose natural course is self-limited and who usually recovers spontaneously. It is, however, differentiate two other groups with more serious prognosis and requiring different diagnostic and therapeutic approach.

Diagnostic algorithm in acute back pain: triage based on different prognosis and different management

B. Patients with ***compressive neurological syndromes*** due to **spondylosis**, endangered by development of **neurological deficit: radicular syndromes** (discogenic or osteogenic), **neurogenic claudication syndrome** in multilevel lumbar stenosis and **cauda equina syndrome** (usually due to medial disc herniation). These compressive syndromes form about 8-10 % of patients with low back pain.

Diagnostic algorithm in acute back pain: triage based on different prognosis and different management

C. Patients with ***serious specific structural and usually progressive disease of the spine*** (tumor, infection, autoimmune inflammation, trauma, osteoporosis), that are in danger of development of neurological deficit, but pain could be the first symptom of serious, life-threatening, but potentially treatable disease (about 5 % of back pain patients). Identification of indicators (risk factors) of increased risk of such a disease (***„red flags“***) is considered as already verified strategy.

Diagnostic algorithm in acute back pain: triage based on different prognosis and different management

„RED FLAGS“:

- age >50 (55) yrs or <20 yrs (tumor); age >70 yrs (suspicion of trauma);
- presence of primary extravertebral tumor (increased OR from 0.7 to 9%), chronic inflammation (infection of kidney, skin, lungs), or other serious disease (diabetes – infection);
- long-term steroid treatment (trauma, infection); other immunosuppression (HIV, cytostatics – infection); intravenous administration of drugs (infection);

Diagnostic algorithm in acute back pain: triage based on different prognosis and different management

„RED FLAGS“:

- spine surgery or other invasive procedure (lumbar puncture, periradicular therapy, epidural catheter - infection);
- loss of weight, unexplained fever (tumor, infection);
- history of trauma;
- pain lasting >1 month (especially tumor);
- pain of extraordinary intensity or lasting >1 month without relief, resting, especially nocturnal pain (tumor, infection); pain provoked by stance and decreasing while sitting; localized in thoracic level; considerable local tenderness of vertebra

Diagnostic algorithm in acute back pain: G.P. vs. specialist?

All current clinical guidelines on the management of back pain agree on the attitude that patients with acute non-specific low back pain without red flags, extravertebral disease or neurological deficit should be managed by a doctor of the first contact, i.e. **general practitioner for approximately one month**. A specialist should be contacted in case of red flags, neurological deficit or if a patient does not respond to standard treatment for at least one month.

In all other cases patients should be managed by a specialist.

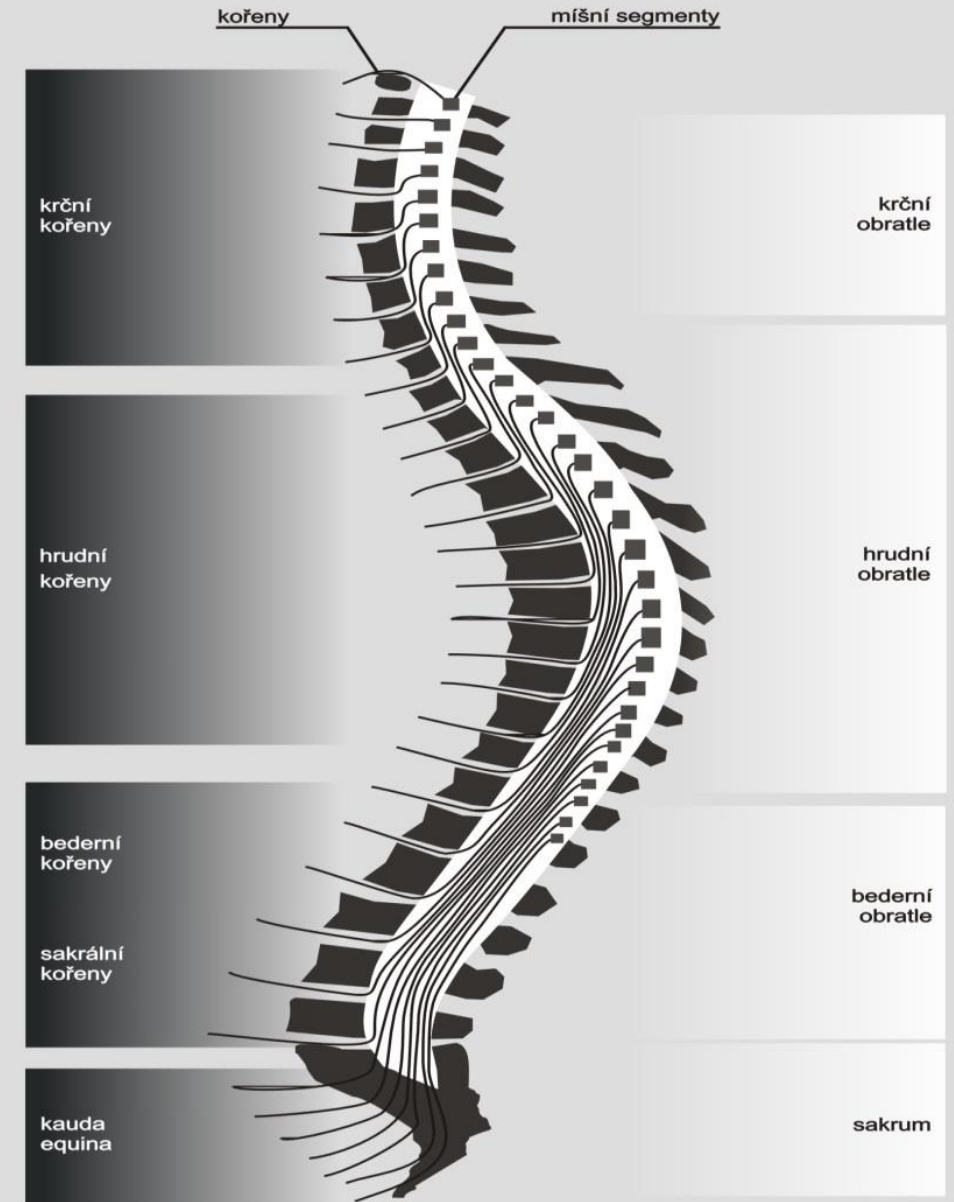
Whom will a patient with acute low back pain visit in the USA?

- GP: 58,6%
- Ortopedic surgeon: 36,9%
- Chiropractist: 30,8%
- Osteopathy specialist: 13,8%
- Internist: 7,6%
- Rheumatologist: 2,5%
- **Neurologist: 0!!!**

Deyo R, Tsui-Wu Y-Jo. Descriptive epidemiology of low-back pain and its related medical care in the United States. Spine 1987; 12:264-268.

Vertebromedullar topography

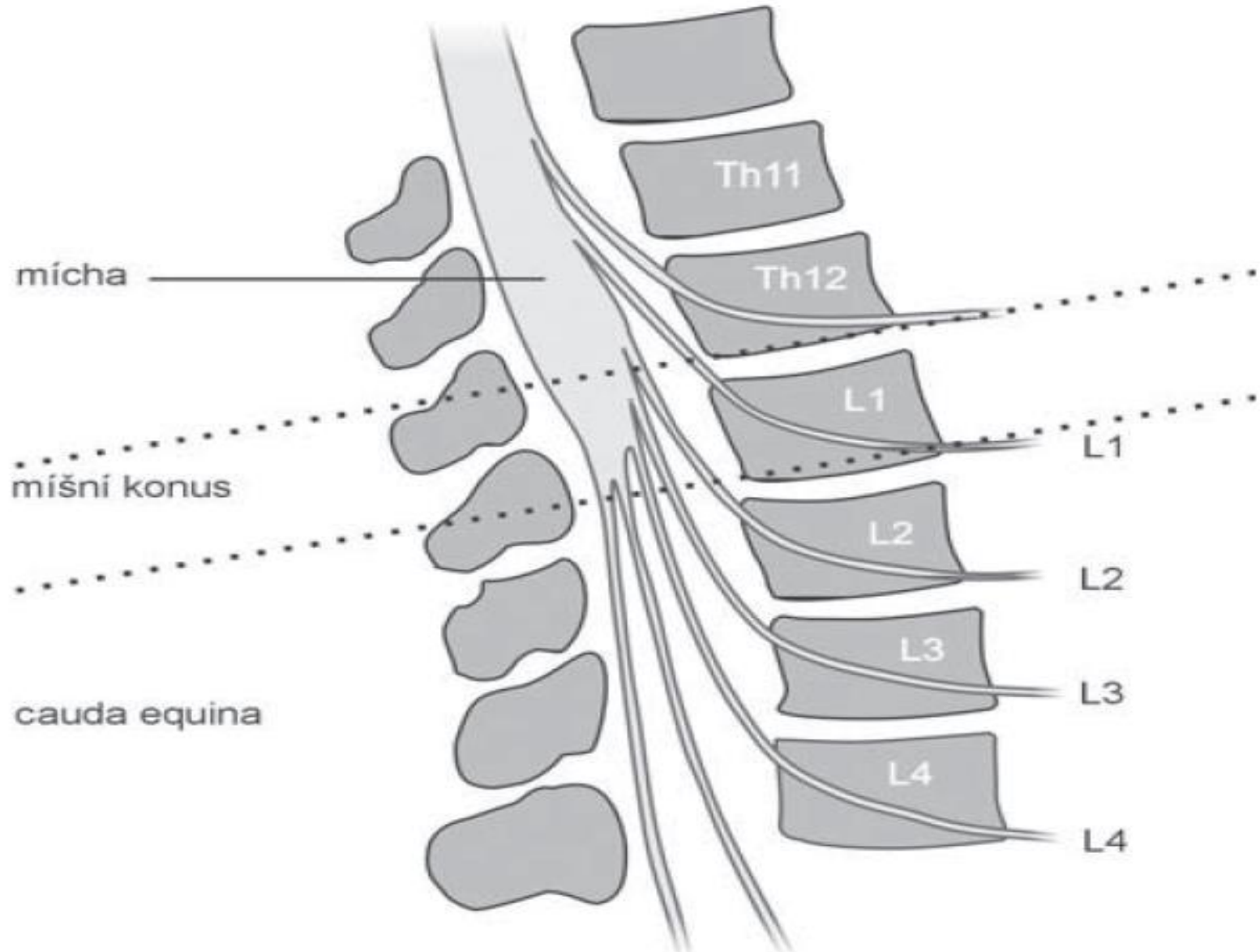
Vertebrae	Medullar segments and roots
C1-7	C1-8 (+1)
Th1-6	Th1-6 (+2)
Th7-10	Th7-12 (+3)
Th 11	L5
Th 12	S2
L1-2	S3-5 (conus medullaris)



Bednařík et al. 2010

Vertebromedullar topography

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Bednařík et al. 2010

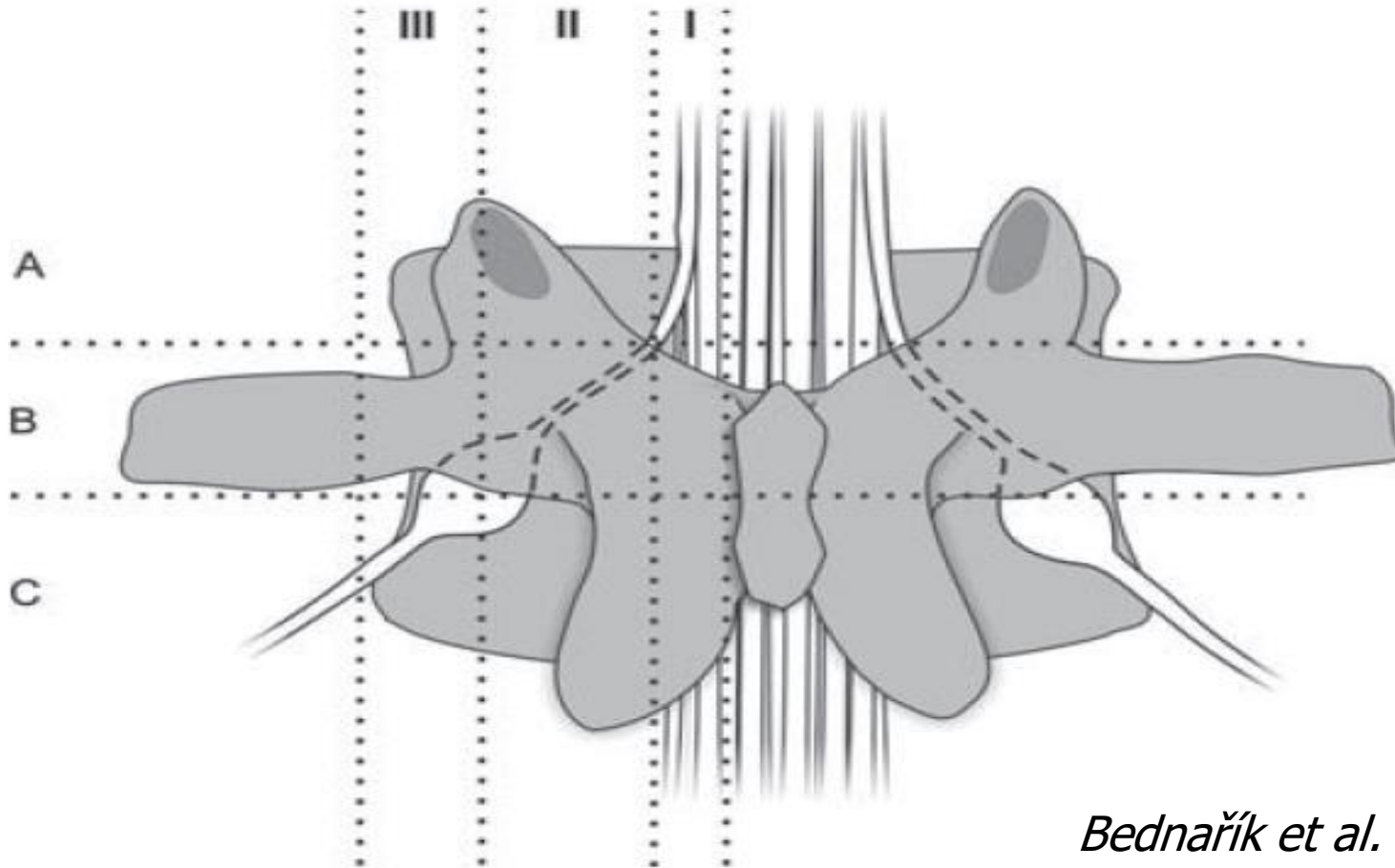
Úseky kořenového kanálu ve frontální rovině

Mediolaterální aspekt

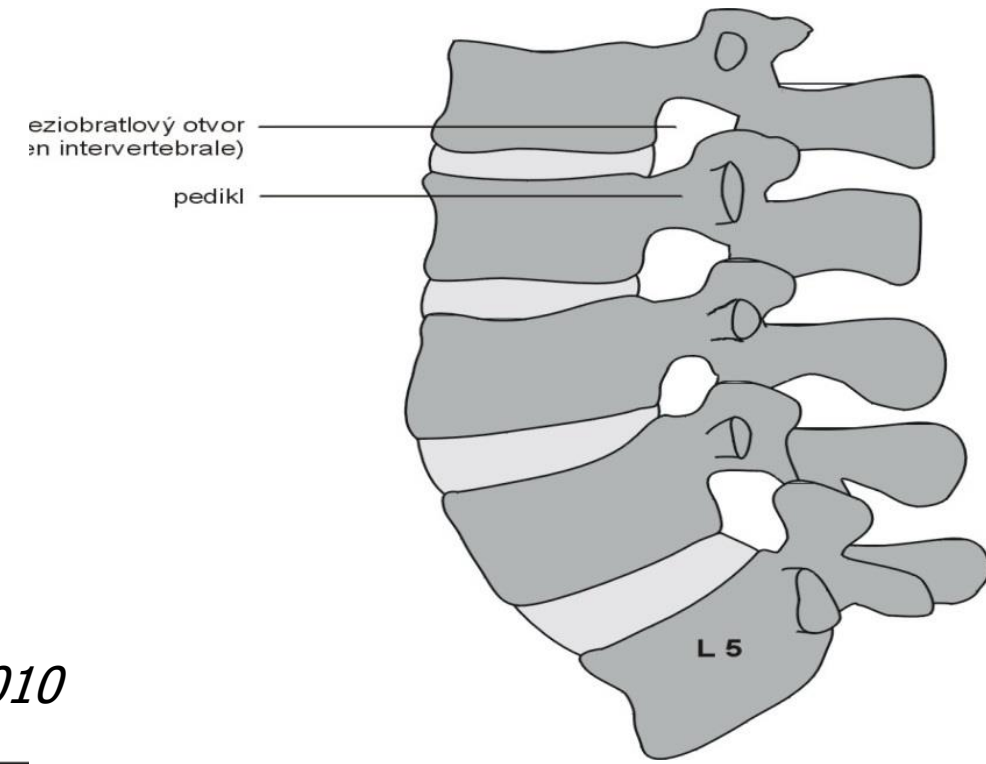
- I zóna laterálního recesu
- II zóna foraminální
- III zóna extraforaminální

Kraniokaudální aspekt

- A parapedikulární úroveň
- B foraminální úroveň
- C extraforaminální úroveň



Lumbar root canal



Bednařík et al. 2010

Causes of radiculopathies

A. Compressive radiculopathies

1. Degenerative

Discopathy: herniations (+fragments)

Osteophytes - mostly uncovertebral (anterior part of the upper recessus articularis)

Disc collapse

2. Non-degenerative: tumors, trauma, osteoporosis, developmental...

B. Non-compressive radiculopathies:

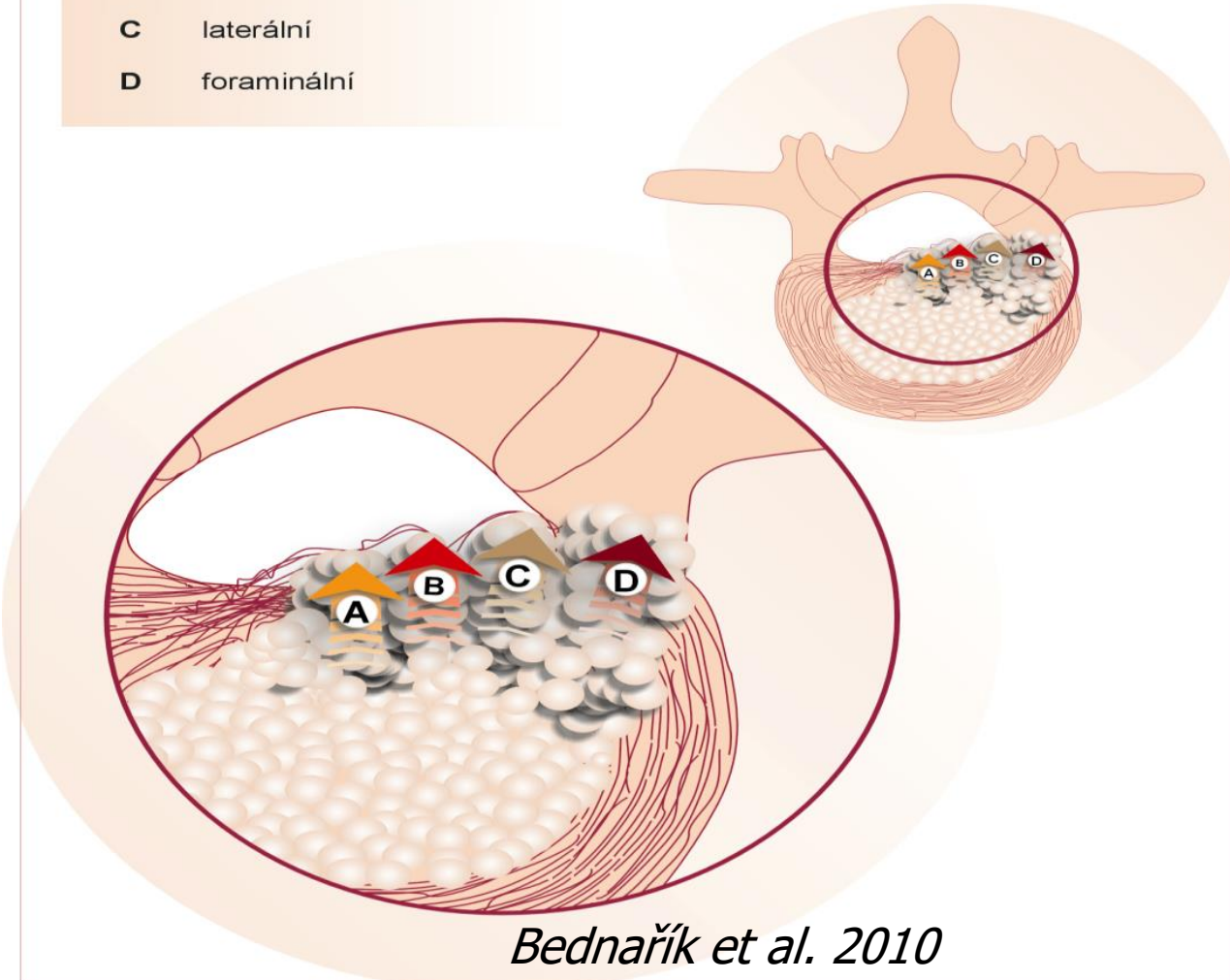
herpes zoster, borreliosis, diabetes mellitus

Lumbar radiculopathy

- A. Medial herniation
- B. Mediolateral herniation
- C. Lateral herniation
- D. Foraminal herniation
- E. Extraforaminal herniation

Schéma lokalizace výhřezů disku

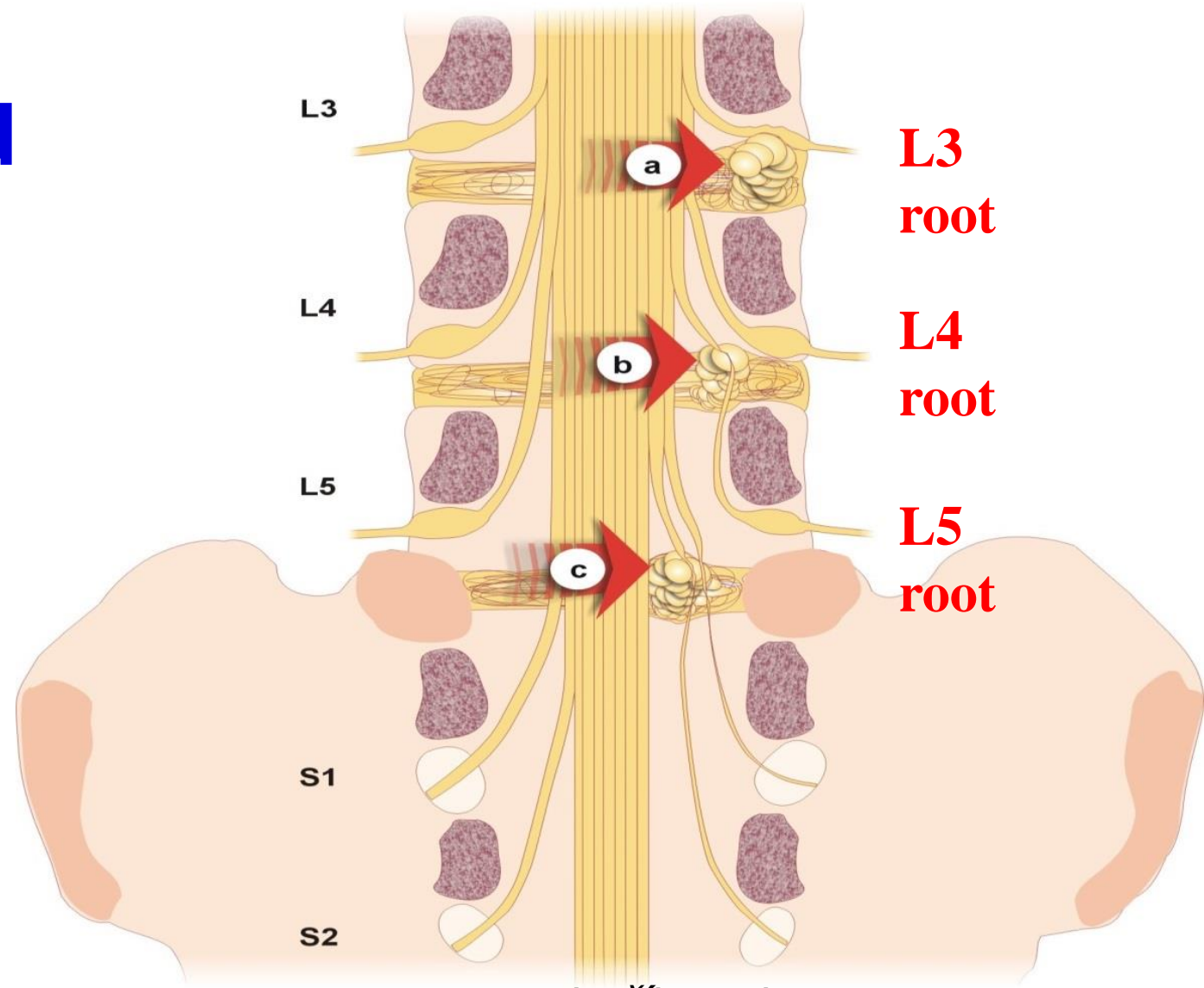
- A mediální (dorzální)
- B paramediální (dorzolaterální)
- C laterální
- D foramínální



Bednařík et al. 2010

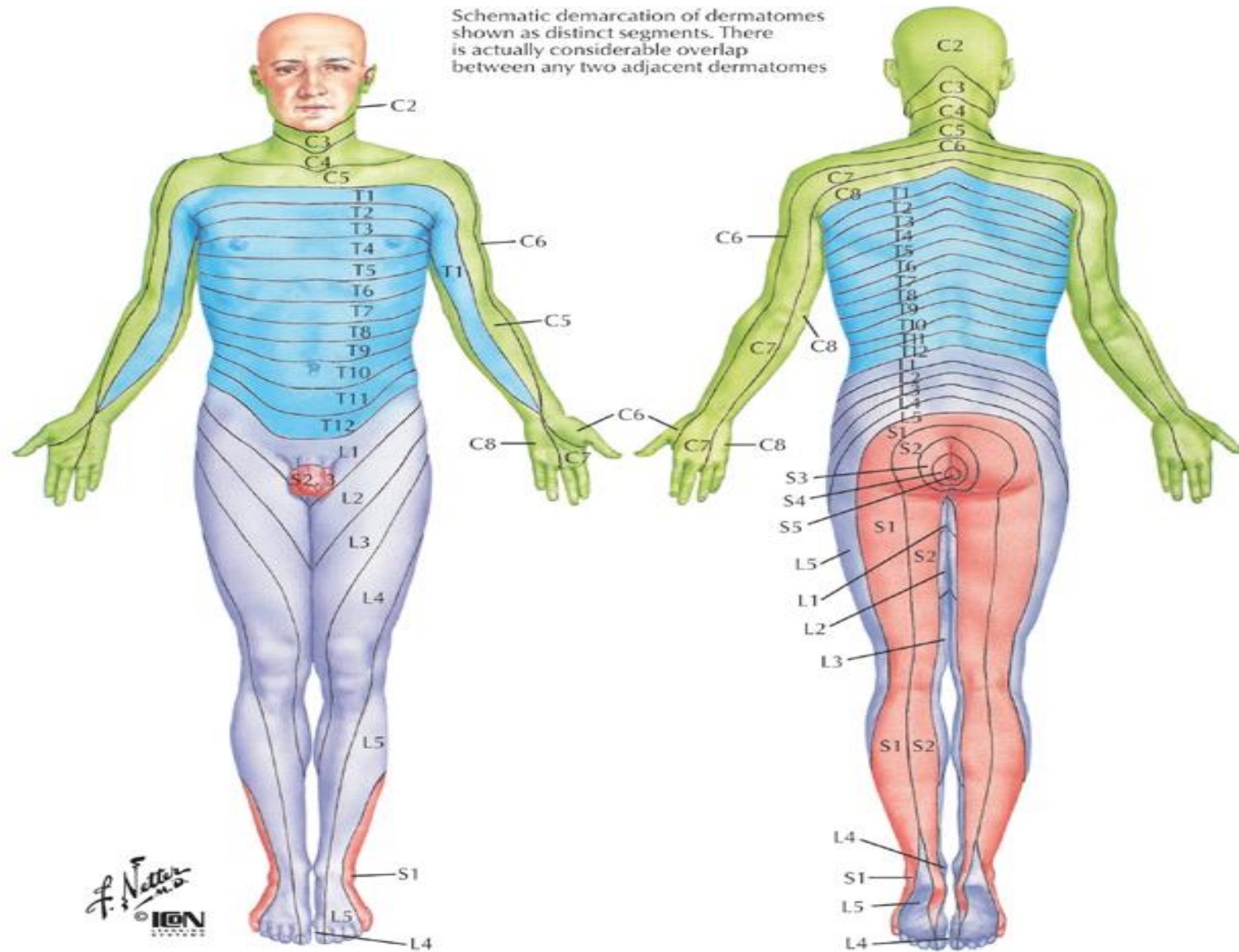
Topography of disc herniations and injured roots

- a foraminální výhřez disku L3 / 4 s kompresí kořene L3
- b laterální výhřez disku L4 / 5 s kompresí kořene L5
- c paramediální výhřez disku L5 / S1 s kompresí kořenů S1 a S2



Bednařík et al. 2010

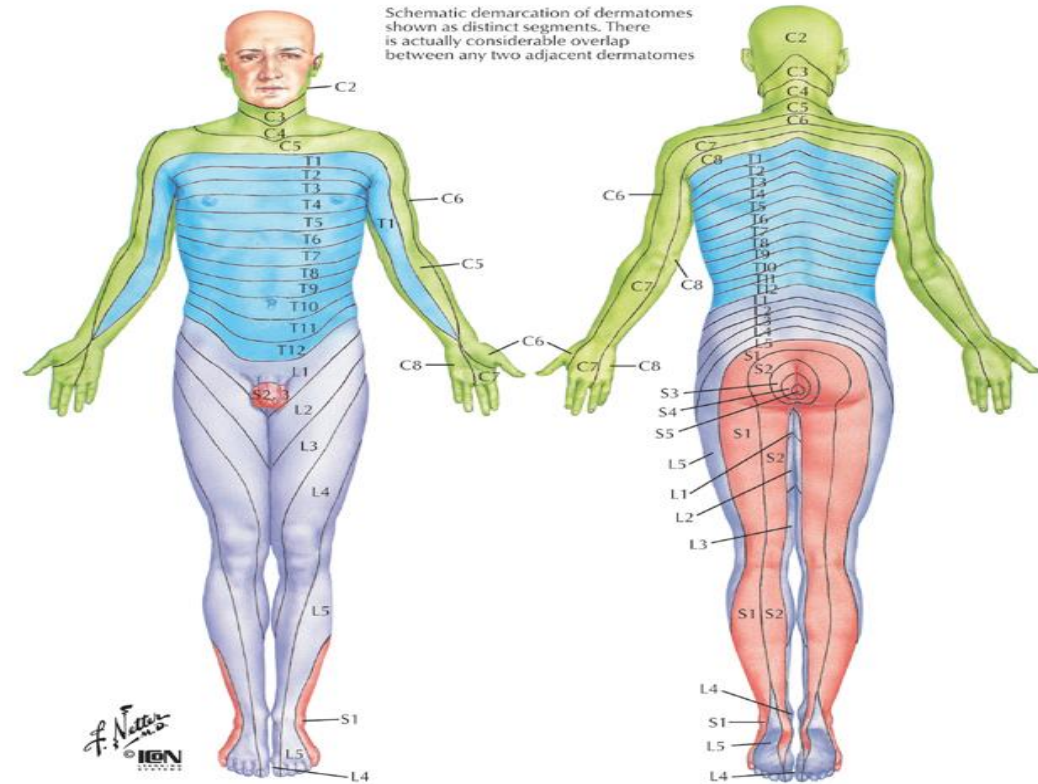
Dermatomes (areae radicales)



F. Netter M.D.
© IGM

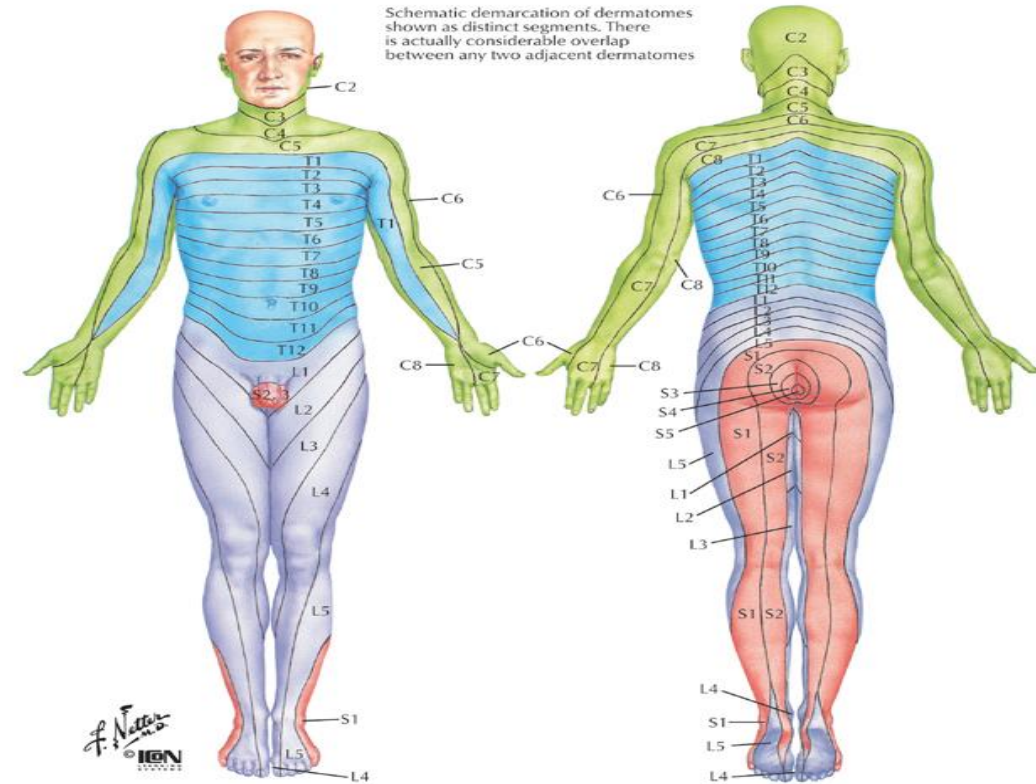
Radiculopathy C5

- PAIN, SENSATION - neck, shoulder
- STRENGTH – weakened arm abduction
- REFLEXES: unelicited bicipital reflex



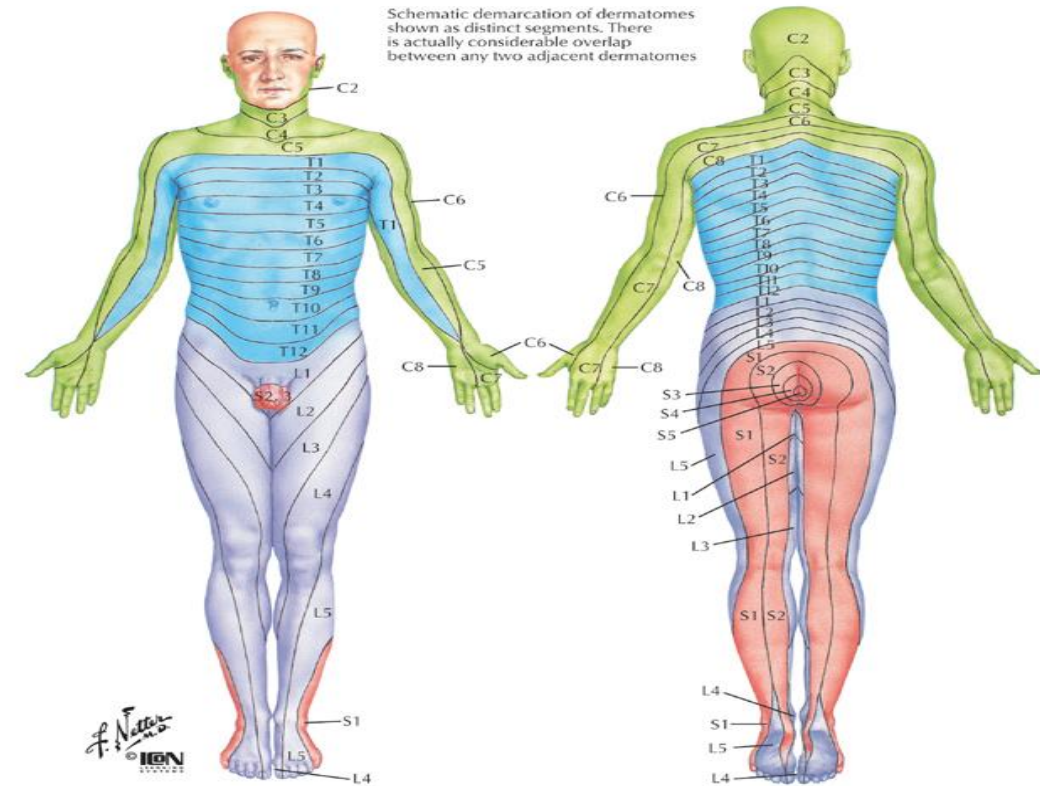
Radiculopathy C6

- PAIN, SENSATION: shoulder, lateral arm, forearm, thumb
- STRENGTH – weakened forearm flexion
- REFLEXES: unelicited bicipital reflex



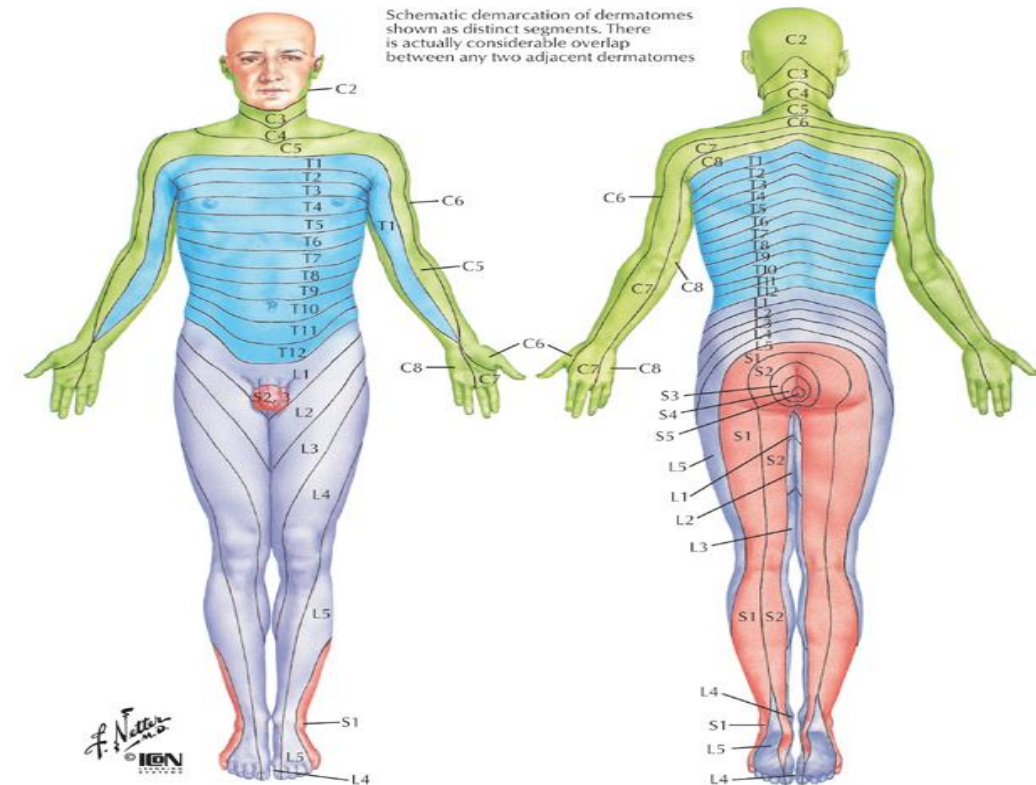
Radiculopathy C7

- PAIN, SENSATION - dorsal aspect of arm, forearm, hand dorsum, digit II-IV.
- STRENGTH – weakened forearm extension
- REFLEXES: unelicited triceps reflex



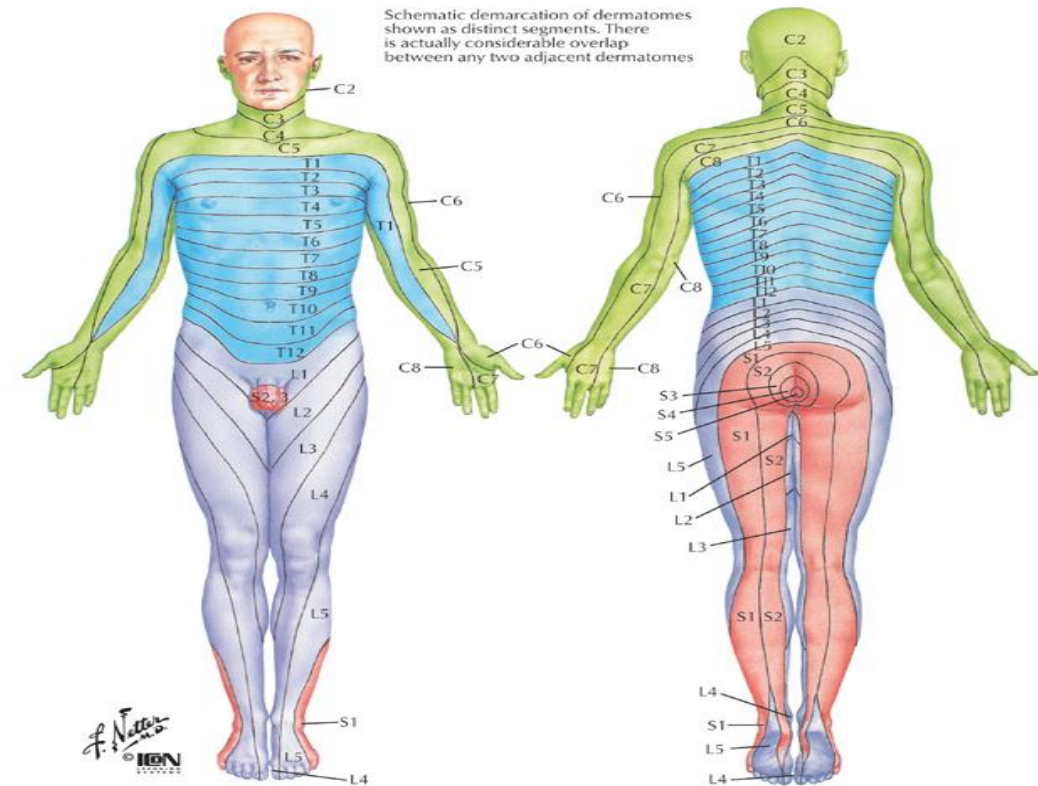
Radiculopathy C8

- PAIN, SENSATION – medial aspect of arm, forearm, digit IV-V.
- STRENGTH – weakened hand muscles
- REFLEXES: unelicited flexor digitorum reflex



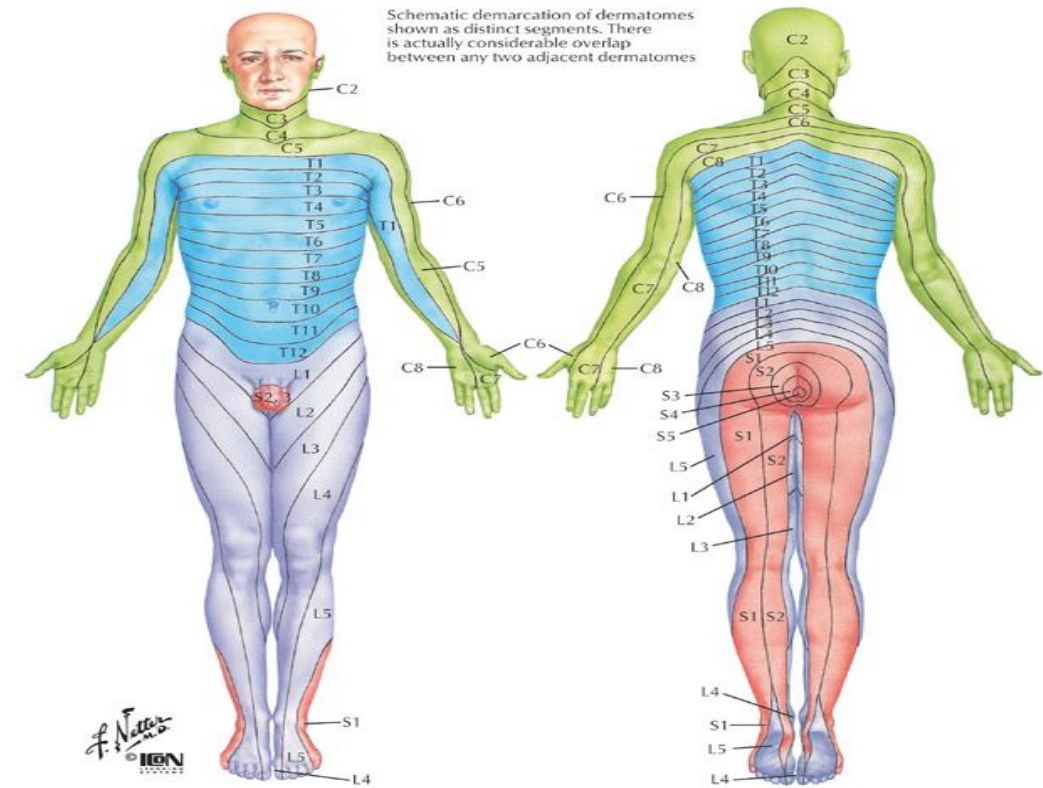
Radiculopathy L4

- PAIN, SENSATION – anterior aspect of thigh, medial aspect of leg
- STRENGTH – weakened knee extension
- REFLEXES: unelicited patellar (knee) reflex



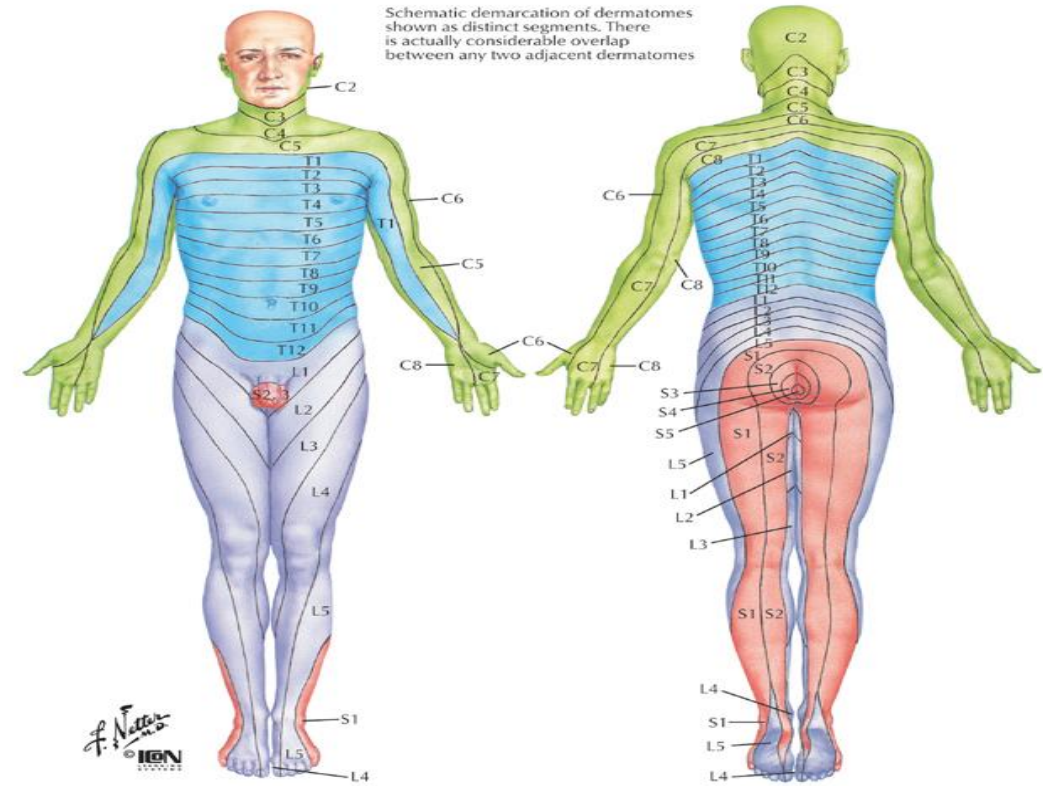
Radiculopathy L5

- PAIN, SENSATION – lateral aspect of thigh, anterolateral aspect of leg, dorsum of hand, big toe
- STRENGTH – weakened foot dorsiflexion
- REFLEXES: 0



Radiculopathy S1

- PAIN, SENSATION – gluteal region, dorsal aspect of thigh, leg, lateral aspect of foot, digit. II-V
- STRENGTH – weakened flexion of foot
- REFLEXES: unelicited Achilles tendon reflex



Diagnostic workup

- Clinical examination: pain characteristics and topography, strength, sensation, reflexes, compressive maneuvers
- Radiograms (AP, lateral, oblique projections, dynamic scans)
- MRI
- CT
- Myelography, myelo/CT
- Electrophysiological exams (EMG, SEP, MEP)

Compressive root tests



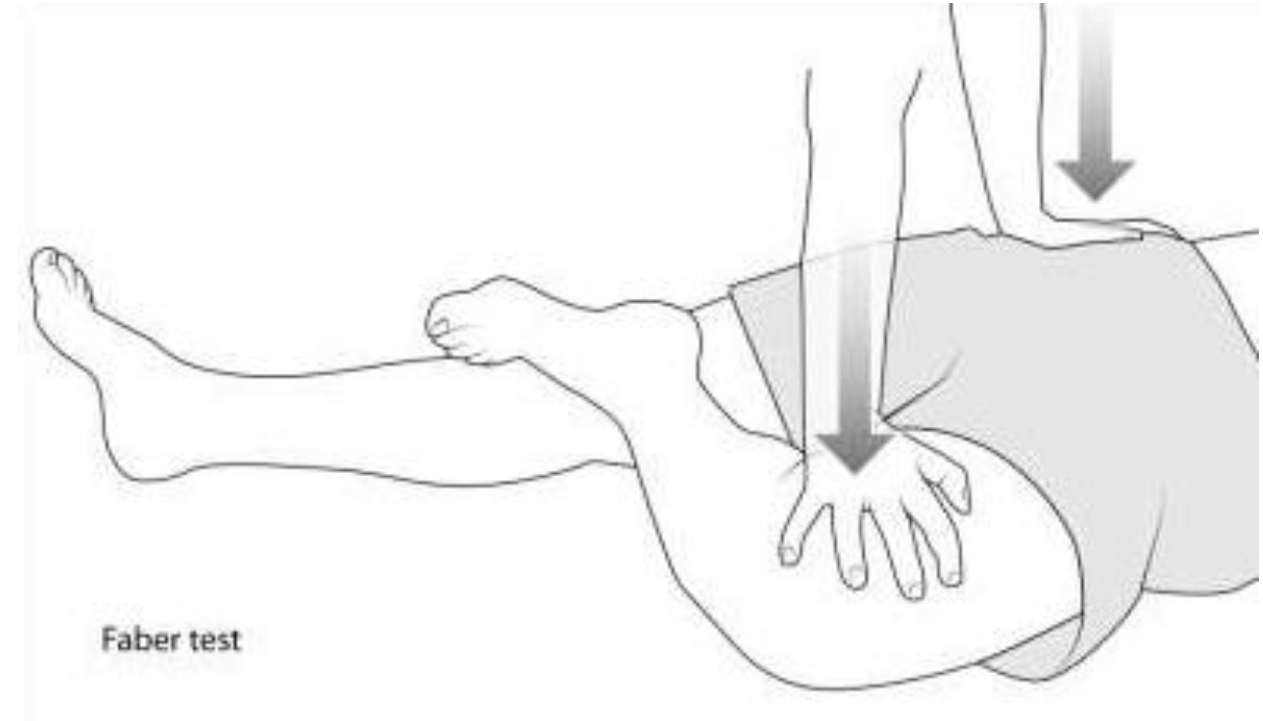
Femoral nerve stretch test
– L4



Straight leg raising test
(Lassegue): L5 a S1

Patrick-Faber test

It is positive if pain arise from the hip or sacroiliacal joint



Adiga et al. McMaster Musculoskeletal Clinical Skills Manual

RTG

**Torg-Pavlov Index = a/b
(C5)**

**TA index < 0,82 =
congenital stenosis**



LSS - myelography (radiculosacography)

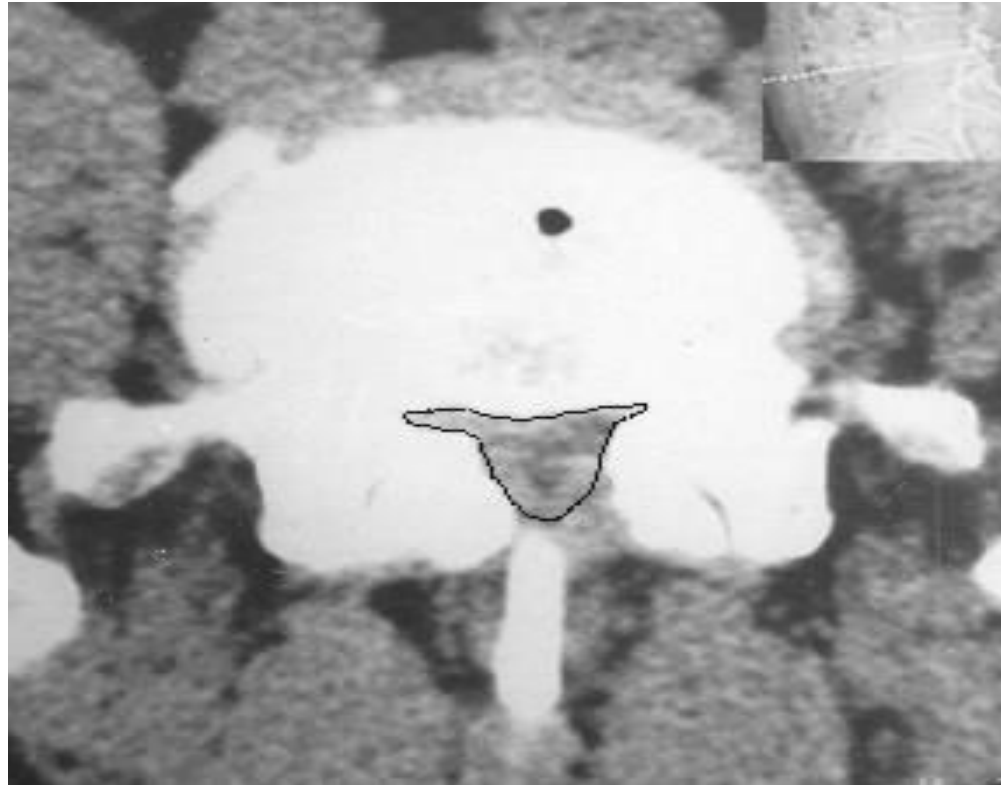
- „Gold standard“
- Quantification of dural sac compression (Porter 1992)



Provided by Dept. Radiology, University Hospital Brno

CT exam

Degenerative lumbar stenosis



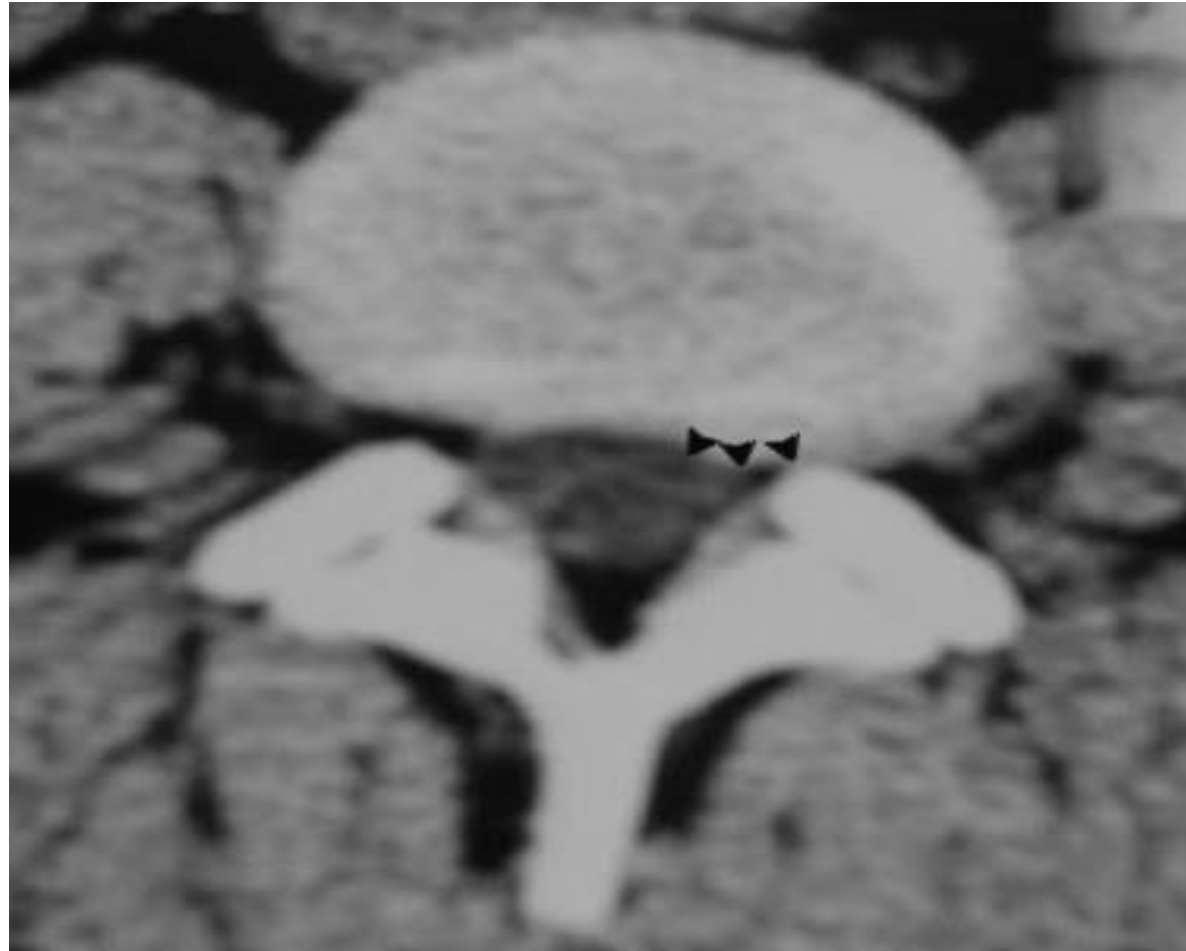
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CT exam: medial disc herniation L5/S1 (axial scan)



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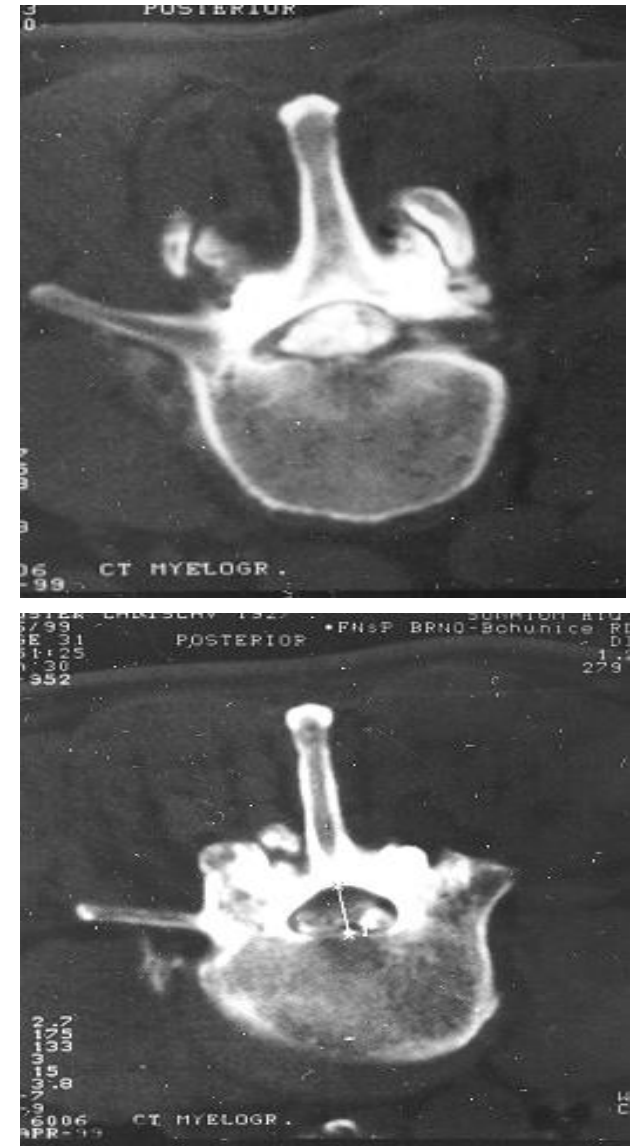
CT exam: lateral disc herniation L5/S1 (axial scan)



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Myelo CT

Axial CT scan above and below block



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Magnetic resonance imaging: medial herniation C6/7 with cervical cord compression (MR sagittal scan, T2W image)



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Magnetic resonance imaging: cervical cord compression by dorsal osteophytes at C5/6 and C6/7 level (MR sagittal scan, T2W image)



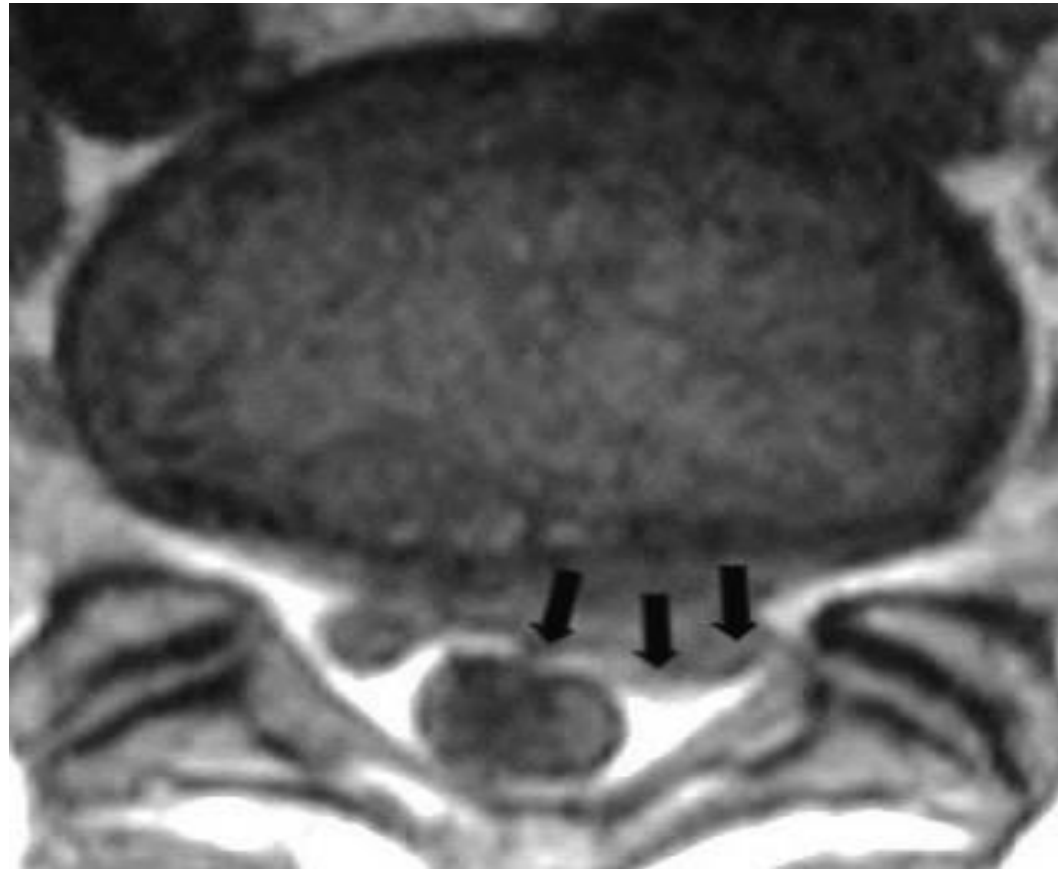
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MRI: paramedial sequestered L5/S1 disc herniation (MR sagittal scan, T1W image)



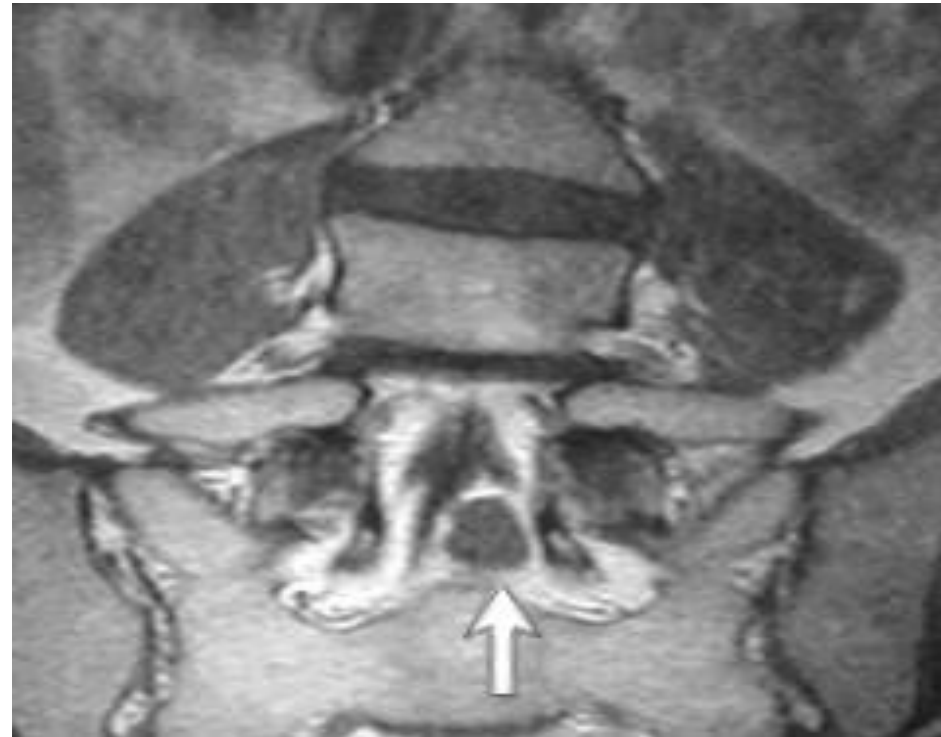
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MRI: paramedial L5/S1 disc herniation on the left side (T1W image, axial scan)



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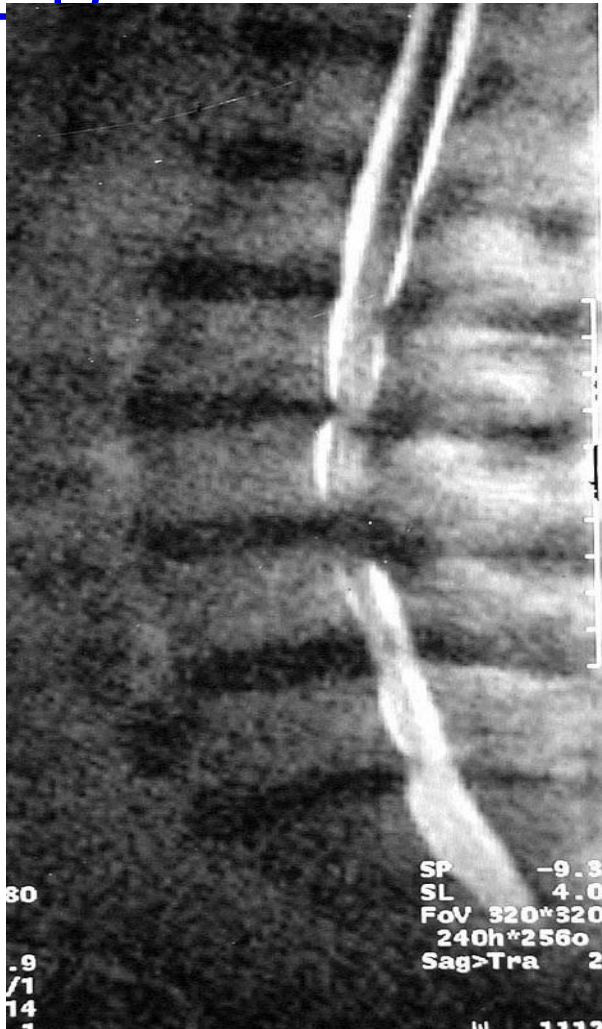
MRI: left-sided paramedia L5/S1 disc herniation (TW1 image, frontal scan)



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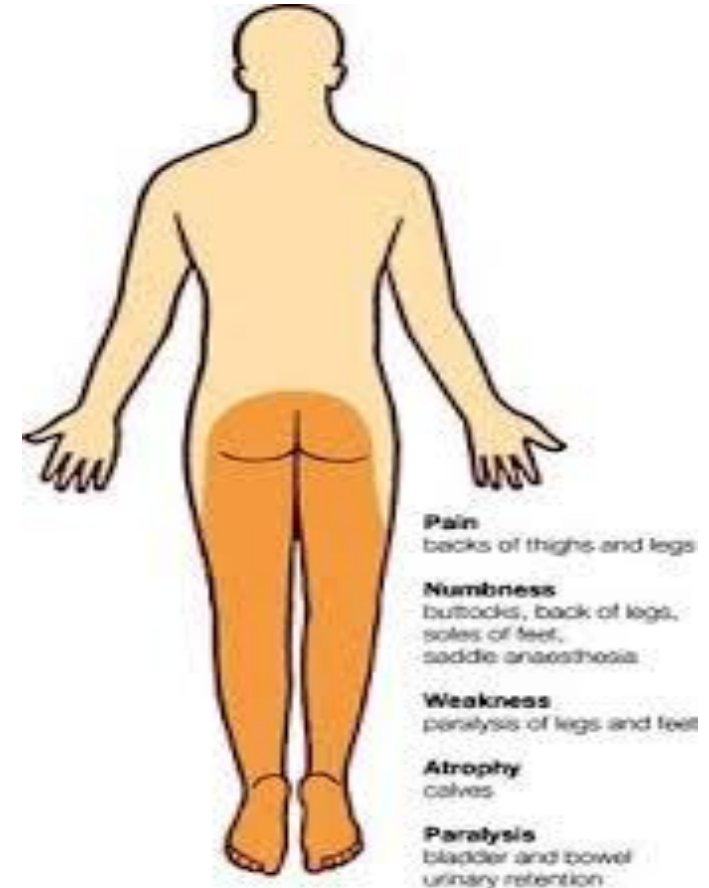
MRI myelography: multisegmental degenerative lumbar stenosis



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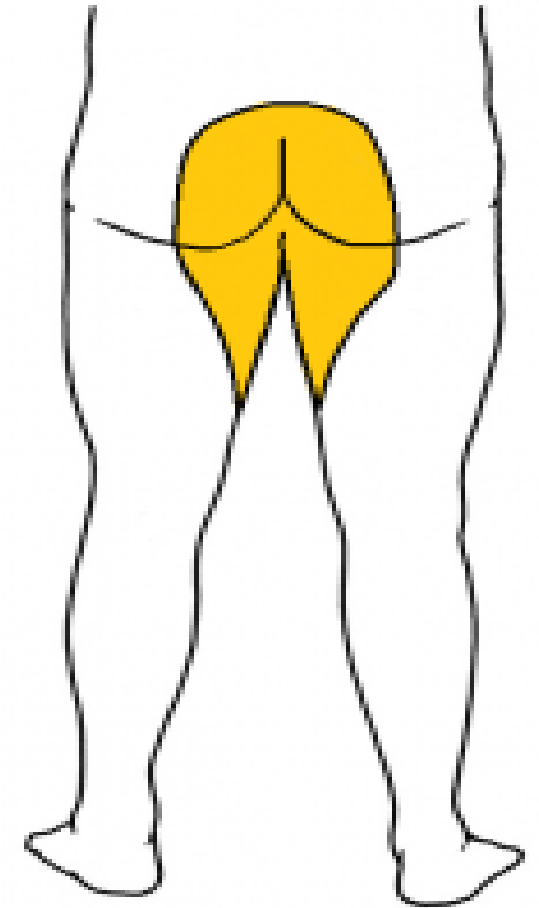
Cauda equina syndrome

- Sphincter dysfunction
- Sensation, pain: Saddle hypo/anaesthesia + more proximal dermatomes
- Possible asymmetry
- Flaccid paraparesis
- Positive compressive tests (Lassegue)



Conus medullaris syndrome

- Sensation: saddle hypo/anaesthesia, no pain
- Sphincter disturbances



Degenerative (spondylotic) cervical myelopathy (DCM)

Epidemiology: the most frequent cause of lower paraparesis above the age of 55 years

Pathophysiology:

- Cervical cord mechanical compression (static, dynamic)
- Vascular factor
- Repetitive microtraumas

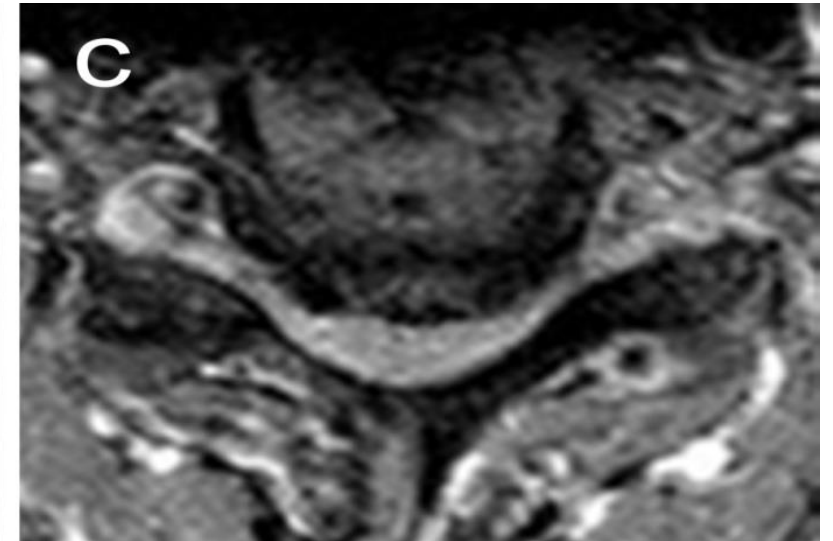
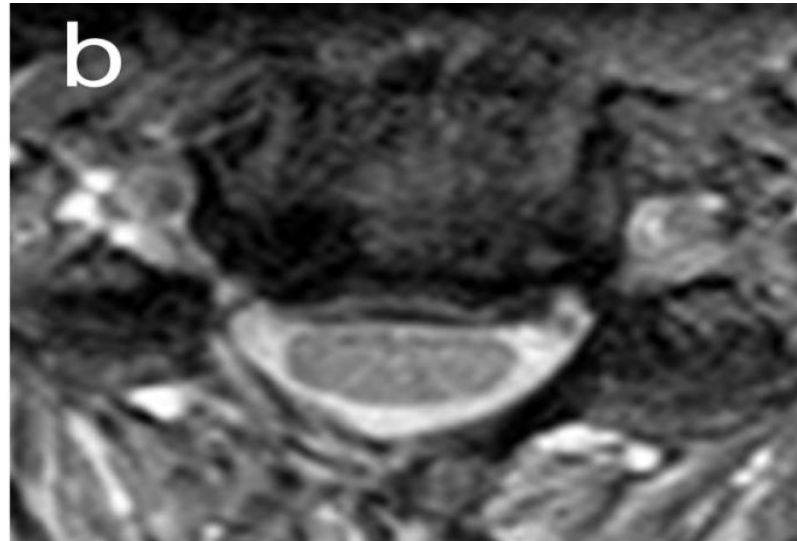


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Most frequent clinical symptoms and signs of DCM

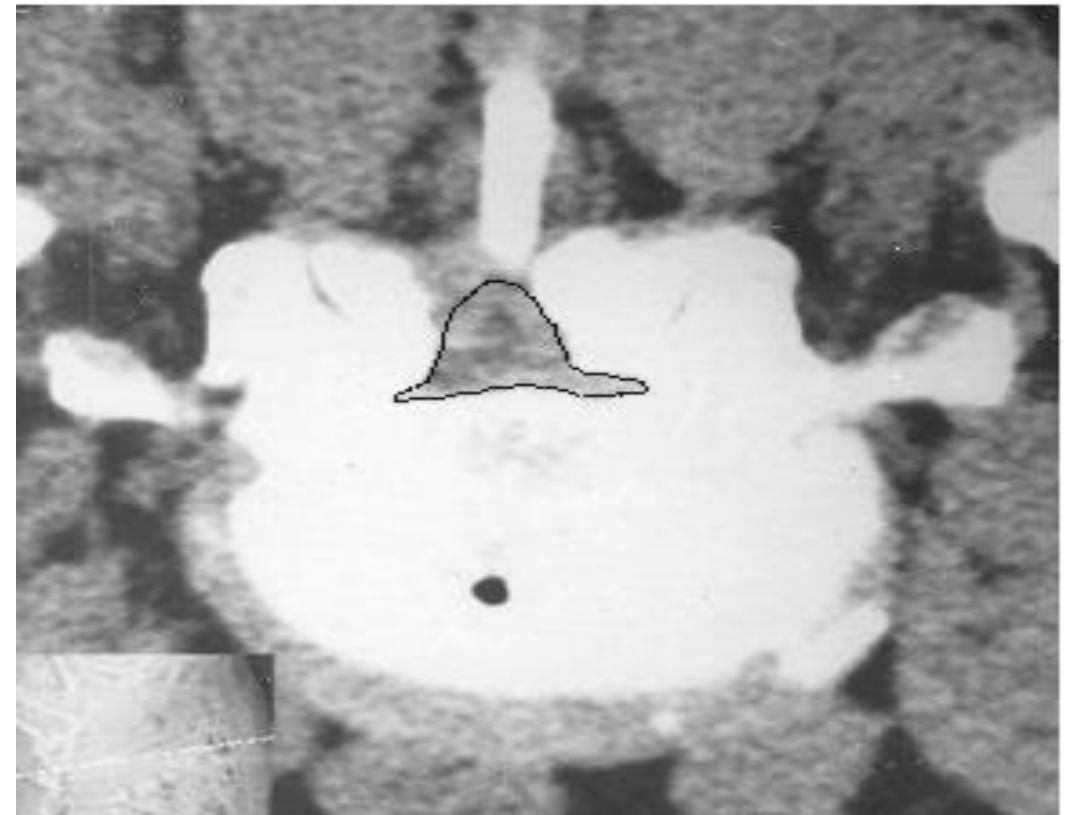
- Clumsy hands
- Disturbance of gait
- Cervical pain, radicular cervical pain
- Paretic signs
- Sensory signs
- Sphincter disturbance

MRI: degenerative cervical cord compression (T1W image, axial scans)



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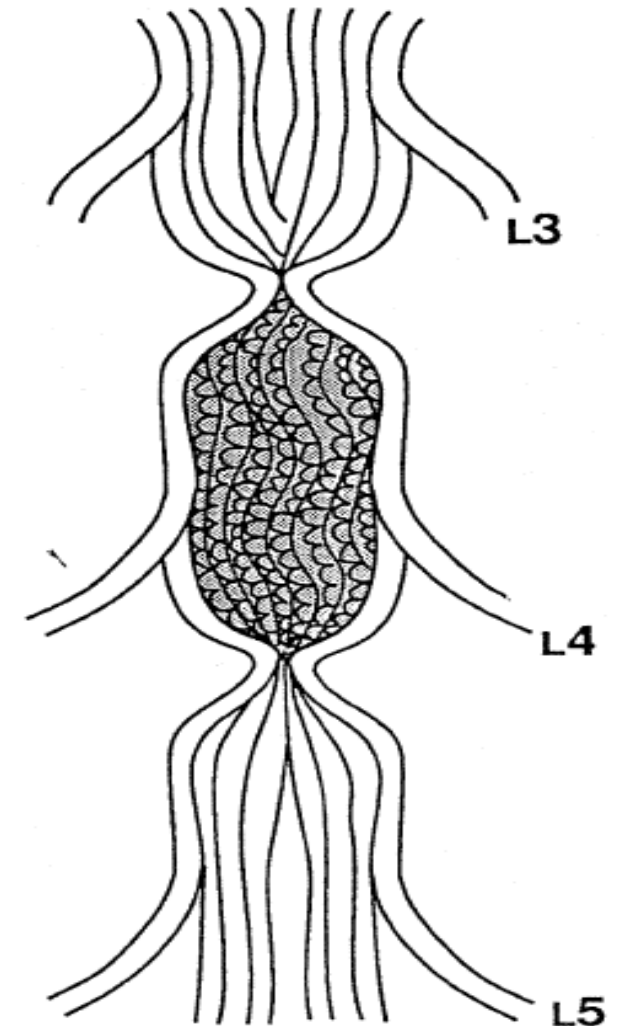
Lumbar stenosis



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Symptomatic lumbar spinal stenosis

- Neurogenic claudication
- Chronic cauda equina syndrome



Diagnosis of claudication

Clinical spectrum of claudications

Ischemic claudications	Neurogenic claudications	Venous claudications
Bolest is in the muscles of calf, thigh or buttock	Pain is in the whole leg and can be accompanied by tingling, hypesthesia or weakness	Pain in the whole leg, that is „going to burst“
Unilateral in femoropopliteal, bilateral in aorto-iliac disease	Mostly bilateral	Mostly unilateral
Gradual onset after walding „claudication distance“	Onset after standing or walking, riding bicycle is possible	Gradual onset after beginning to walk
Pain is relieved by rest	Relieved by bending over or sitting	Relief on elevating the leg
Absent/reduced pulsations	Walking and stance in semiflection	Oedema, varicose veins, cyanosis

A Systematic Review for an American College of Physicians (Chou et al. 2017): LBP

Table 2. Pharmacologic Therapies Versus Placebo for Acute Low Back Pain

Drug	Pain			Function		
	Magnitude of Effect	Evidence	SOE	Magnitude of Effect	Evidence	SOE
Acetaminophen	<u>No effect</u>	1 RCT	Low	No effect	1 RCT	Low
NSAIDs	<u>Small (pain intensity); no effect (pain relief)</u>	1 SR (4 RCTs), 1 RCT	Moderate	Small	2 RCTs	Low
Opioids	No evidence	-	-	No evidence	-	-
Skeletal muscle relaxants	<u>Pain relief: relative risk, 1.72 (95% CI, 1.32-2.22) at 5-7 d</u>	1 SR (4 RCTs), 1 RCT	Moderate	No evidence	-	-
Benzodiazepines	Unable to estimate	2 RCTs	Insufficient	Unable to estimate	2 RCTs	Insufficient
Antiseizure medications	No evidence	-	-	No evidence	-	-
Systemic corticosteroids	No effect	2 RCTs	Low	No effect	2 RCTs	Low

NSAID = nonsteroidal anti-inflammatory drug; RCT = randomized, controlled trial; SOE = strength of evidence; SR = systematic review.

A Systematic Review for an American College of Physicians (Chou et al. 2017): LBP

Table 3. Pharmacologic Therapies Versus Placebo for Chronic Low Back Pain

Drug	Pain			Function		
	Magnitude of Effect	Evidence	SOE	Magnitude of Effect	Evidence	SOE
Acetaminophen	No evidence	-	-	No evidence	-	-
NSAIDs	<u>Small to moderate</u>	1 SR (4 RCTs), 2 RCTs	Moderate	None to small	4 RCTs	Low
Opioids (strong opioids)	<u>Small</u>	1 SR (6 RCTs), 4 RCTs	Moderate	Small	1 SR (4 RCTs), 4 RCTs	Moderate
Opioids (buprenorphine patch or sublingual)	<u>Small</u>	3 RCTs	Low	Unable to estimate	3 RCTs	Insufficient
Tramadol	<u>Moderate</u>	1 SR (5 RCTs), 2 RCTs	Moderate	Small	1 SR (5 RCTs), 2 RCTs	Moderate
Skeletal muscle relaxants	Unable to estimate	3 RCTs	Insufficient	-	-	-
Benzodiazepines: tetrazepam	Failure to improve at 10-14 d: relative risk, 0.71 (95% CI, 0.54-0.93)	1 SR (2 RCTs)	Low	-	-	-
Tricyclic antidepressants	No effect	1 SR (4 RCTs)	Moderate	No effect	1 SR (2 RCTs)	Low
Antidepressants: selective serotonin reuptake inhibitors	No effect	1 SR (3 RCTs)	Moderate	-	-	-
Antidepressants: duloxetine	<u>Small</u>	3 RCTs	Moderate	Small	3 RCTs	Moderate
Gabapentin/pregabalin	Unable to estimate	2 RCTs	Insufficient	Unable to estimate	2 RCTs	Insufficient

NSAID = nonsteroidal anti-inflammatory drug; RCT = randomized, controlled trial; SOE = strength of evidence; SR = systematic review.

A Systematic Review for an American College of Physicians (Chou et al. 2017): LBP: what is new?

- New evidence of no-effectiveness of paracetamol in acute LBP!!!
- New evidence of effectiveness of duloxetine in chronic LBP!!!
- NSAIDs have lower effect in acute and chronic LBP compared to previously believed effect
- Myorelaxants has short-lasting effect in acute LBP, but cause sedation
- Opioids – moderate effect in chronic LBP
- Effect of systemic administration of corticosteroids doesnot seem to be proved
- Generally, all proved effects are short-lasting and of mild or moderate degree

Recommendation NICE 2016

(<https://www.nice.org.uk/guidance/ng59>)

- Consider NSAIDs with respect to side-effect profile and risk for an individual patient
- After NSAIDs administration monitor a patient, side effects and use gastroprotection, use lowest-possible dose and shortest-possible duration of treatment!
- Consider weak opioids (as monotherapy or in combination with paracetamol) in case of ineffectiveness, intolerance or contraindication of NSAIDs!
- Don't use paracetamol in monotherapy!!
- Don't use opioids routinely for acute LBP
- Don't use opioids for chronic LBP
- Don't use SSRI, SNRI??? and TCA in LBP
- Don't use anticonvulsants (pregabalin, gabapenti) in LBP (except radicular pain)

Conclusions

Always consider the use of pharmacotherapy in LBP:

- “most episodes of acute LBP are self-limited and not every patient needs pharmacotherapy!
- It is recommended to explain to patients benign character of acute LBP episodes, expected benefit of pharmacotherapy and possible side-effects
- Risks of side effects of pharmacotherapy could overweight its benefit!
- Use non-pharmacological treatment?

Conclusions

In acute LBP after decision to start pharmacotherapy:

- Short-lasting therapy, for necessary episode only, follow side effects, instruct a patient!
- Consider NSAIDs, non-benzodiazepin myorelaxants
- In severe pain (even in chronic LBP) consider weak opioids and their combination with paracetamol, strong opioids (oxycodon), tapentadol

Conclusions

In chronic LBP:

- Consider pharmacotherapy (complex problem, change of regimen, exercise, yellow flags!!!)
- In case of acute exacerbations of pain consider NSA, opioids (weak, strong, tapentadol)
- In case of a neuropathic component of pain consider gabapentinoids, duloxetine, opioids, tapentadol, eventually in combination with analgesics relieving nociceptive pain (NSAIDs, paracetamol)
- Short-lasting therapy!

Conclusions

As non-indicated procedures in LBP are currently considered:

- Paracetamol in monotherapy
- Myorelaxants in chronic LBP
- Antidepressants (TCA, SSRI)
- Long-term pharmacotherapy (especially opioids, NSAIDs)
- Systemic administration of corticosteroids

Indications for surgical therapy

- Secondary lesions of diseases of the spine or spinal cord (if possible)
- Acute cauda equina syndrome due to medial disc herniation (within 24 hours)
- Compressive monoradiculopathies due to disc herniation or radicular canal stenosis (refractory to non-surgical therapy, prominent neurological deficit)
- Degenerative cervical myelopathy (moderate-to-severe deficit, progression of the deficit)

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