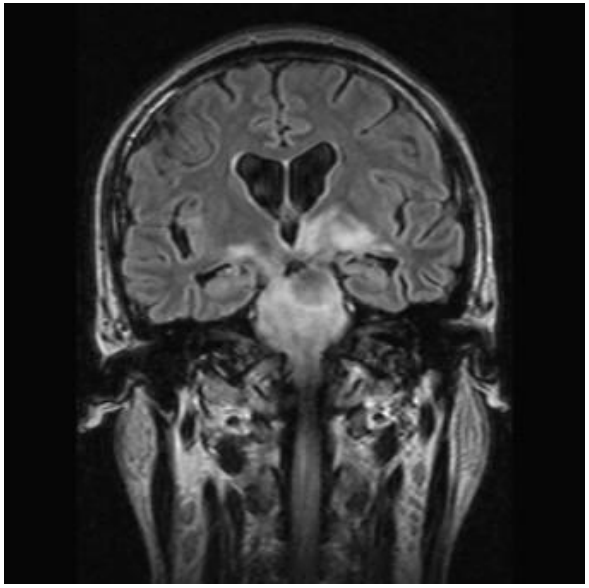
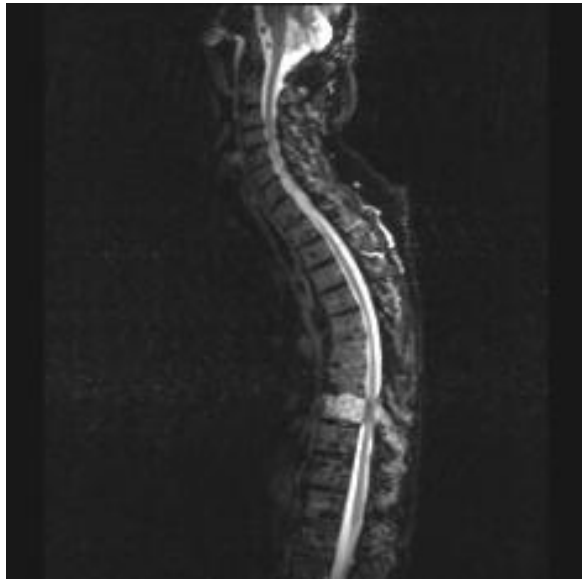
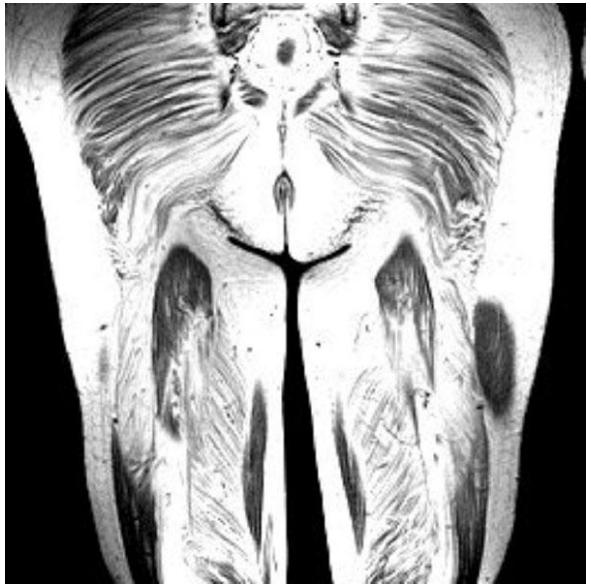
Management of ascending paresis

PB 2013

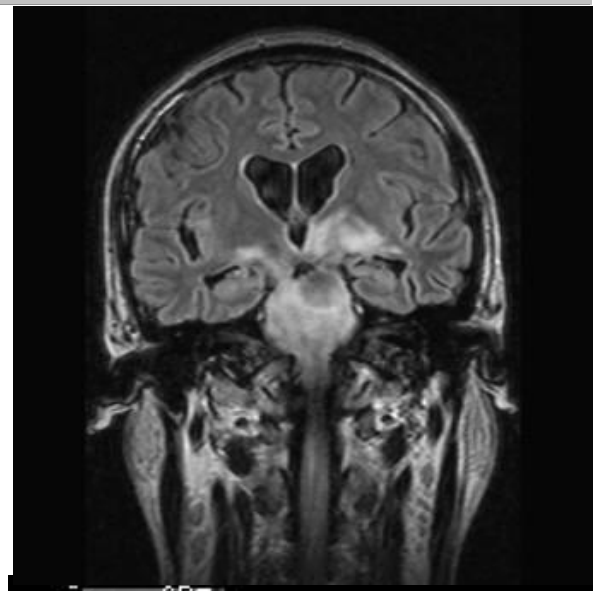
Peter Berlit

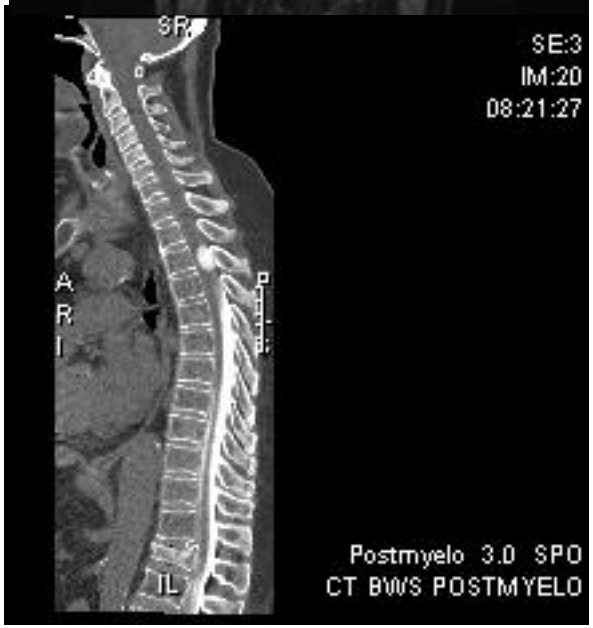
Alfried Krupp Hospital Essen, Germany

Ascending paresis– central causes

PB 2013

- Brain:
  - brainstem ischemia, hemorrhage
  - CPM
  - MS
- Spinal cord:
  - tumor
  - myelitis
  - spinal ischemia
  - spinal hemorrhage
  - rabies

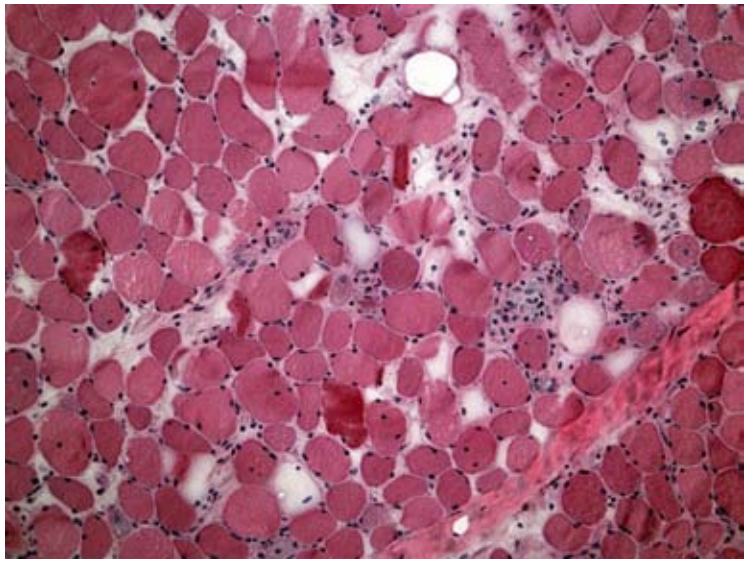


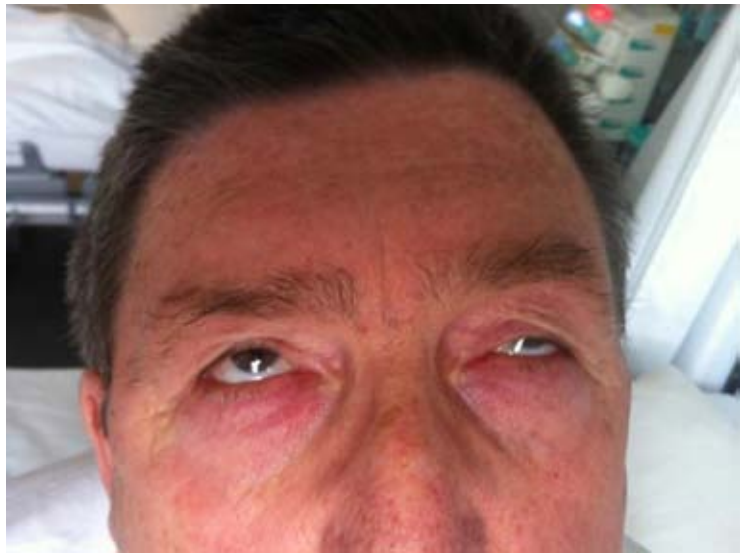


Ascending paresis– peripheral causes

PB 2013

- Peripheral nerves:
  - acute polyneuritis (GBS)
  - diphtheria
- Neuromuscular synapsis:
  - myasthenia gravis
  - LEMS
  - botulism
- Muscle:
  - acute myopathies
  - rhabdomyolysis
  - periodic paralysis





Spinal cord	**Acute transverse myelitis**
	Spinal cord infarct
	Spinal epidural or subdural hemorrhage
	Central intervertebral disc herniation
	Tumors (metastatic or primary)
	Multiple sclerosis
Peripheral	
Spinal nerve root	*Intervertebral disc herniation*
	Epidural abscess
	Tumors
	Leptomeningeal metastases
Polyneuropathies	**Guillain-Barré syndrome**
	Diabetic
	Ciguatoxin (ciguatera poisoning)
	Saxitoxin (paralytic shellfish poisoning)
	Tetrodotoxin poisoning (pufferfish poisoning)
	Organophosphate poisoning
	Tick paralysis
Myopathy	**Electrolyte induced**
	Inflammatory (polymyositis)
	Alcohol or drug-induced
	Muscular dystrophy
	Endocrine related
Nonphysiologic/noncategorical	
Conversion disorder	
Chronic fatigue syndrome	
Anxiety disorders	
Fibromyalgia	
Malingering	

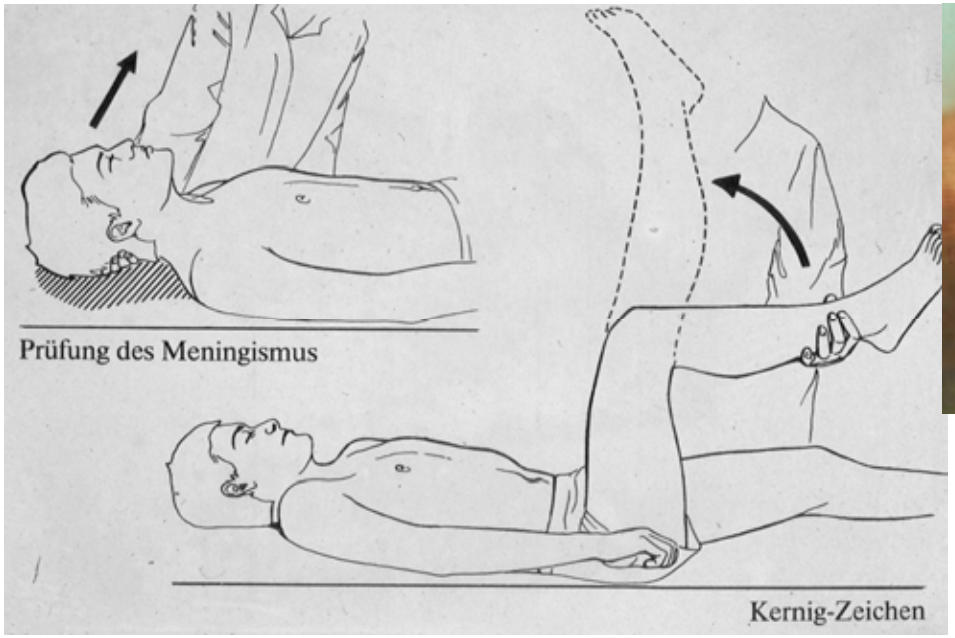
Italicized: common conditions.
Bold: serious conditions.

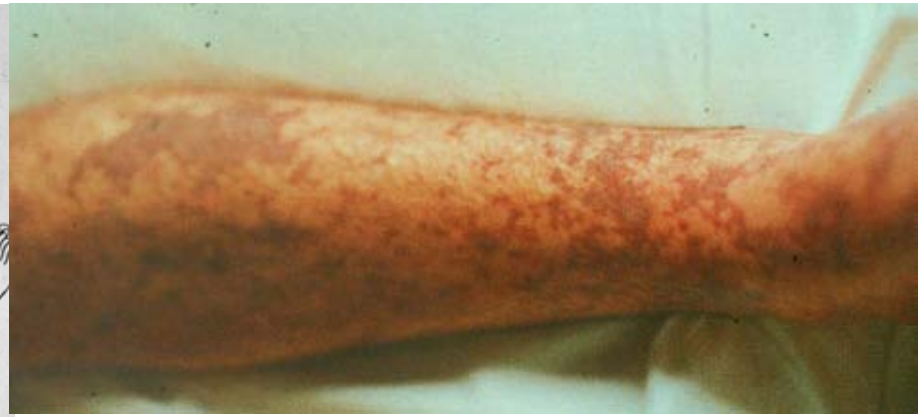
ascending paresis– important cerebral causes

- Brainstem ischemia in basilar artery thrombosis
- Brainstem hemorrhage
- Brainstem compression with tumor or hemorrhage
- Central pontine myelinolysis
- Brainstem encephalitis (MS, infection)

Meningitis

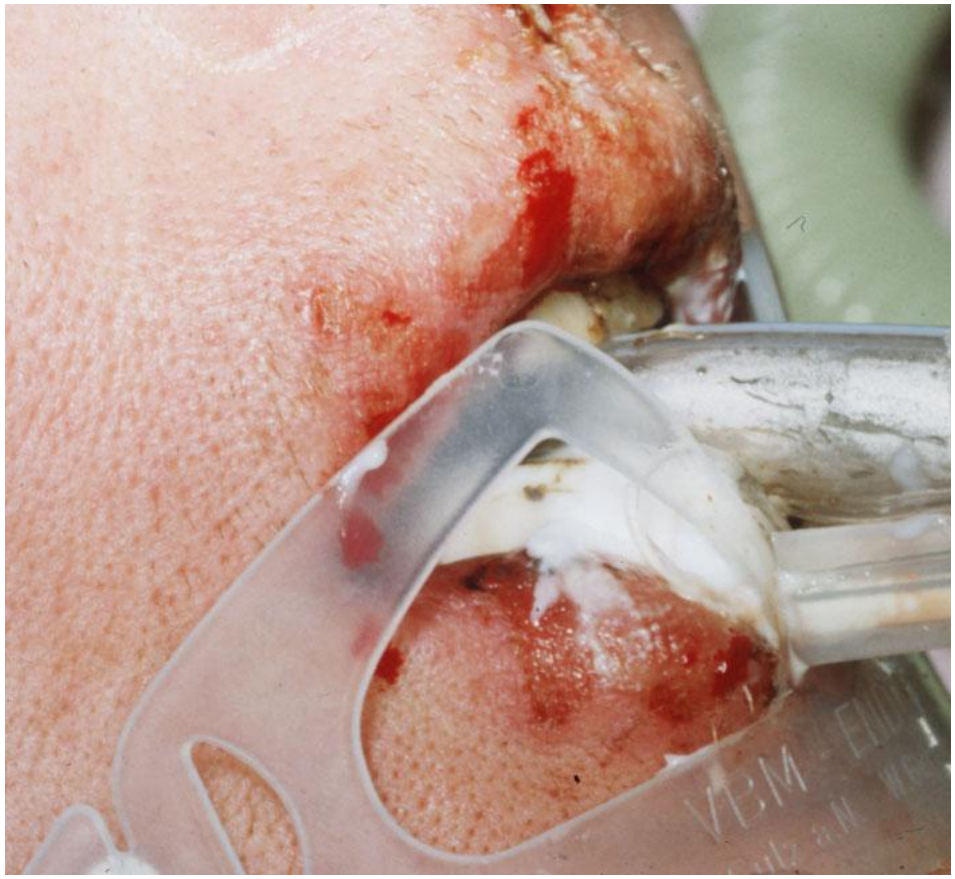
PB 2013

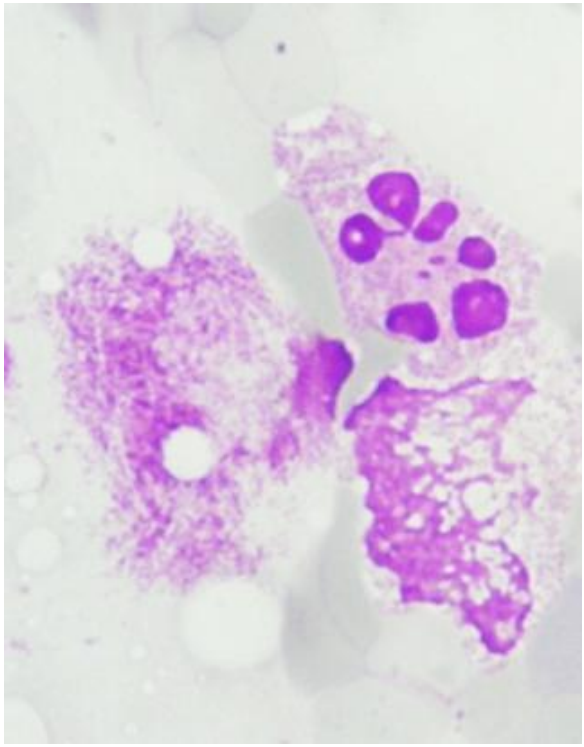




Meningitis

PB 2013





The Cochrane Collaboration
Cochrane Reviews

PB 2013

[Intervention Review]

Corticosteroids for acute bacterial meningitis

Diederik van de Beek[1], Jan de Gans[1], Peter McIntyre[2], Kameshwar Prasad[3]

Main results

Eighteen studies involving 2750 people were included. Overall, adjuvant corticosteroids were associated with lower case fatality (relative risk (RR) 0.83, 95% CI 0.71 to 0.99), lower rates of severe hearing loss (RR 0.65, 95% CI 0.47 to 0.91) and long-term neurological sequelae (RR 0.67, 95% CI 0.45 to 1.00). In children, corticosteroids reduced severe hearing loss (RR 0.61, 95% CI 0.44 to 0.86). In adults, corticosteroids gave significant protection against death (RR 0.57, 95% CI 0.40 to 0.81) and short-term neurological sequelae (RR 0.42, 95% CI 0.22 to 0.87). Subgroup analysis for causative organisms showed that corticosteroids reduced mortality in patients with meningitis due to *Streptococcus pneumoniae* (RR 0.59, 95% CI 0.45 to 0.77) and reduced severe hearing loss in children with meningitis due to *Haemophilus influenzae* (RR 0.37, 95% CI 0.20 to 0.68); subgroup analysis for patients with meningococcal showed a nonsignificant favourable trend in mortality (RR 0.71, 95% CI 0.31 to 1.62). Sub analyses for high-income and low-income countries of the effect of corticosteroids on mortality showed RRs of 0.83 (95% CI 0.52 to 1.05) and 0.87 (95% CI 0.72 to 1.05), respectively. Corticosteroids were protective against short-term neurological sequelae in patients with bacterial meningitis in high-income countries (RR 0.56, 95% CI 0.3 to 0.84); in low-income countries this RR was 1.09 (95% CI 0.83 to 1.45). For children with bacterial meningitis admitted in high-income countries, corticosteroids showed a protective effect against severe hearing loss (RR 0.61, 95% CI 0.41 to 0.90) and favourable point estimates for severe hearing loss associated with non-*Haemophilus influenzae* meningitis (RR 0.51, 95% CI 0.23 to 1.13) and short-term neurological sequelae (RR 0.72, 95% CI 0.39 to 1.33). For children in low-income countries, the use of corticosteroids was neither associated with benefit nor with harmful effects. Overall, adverse events were not increased significantly with the use of corticosteroids.

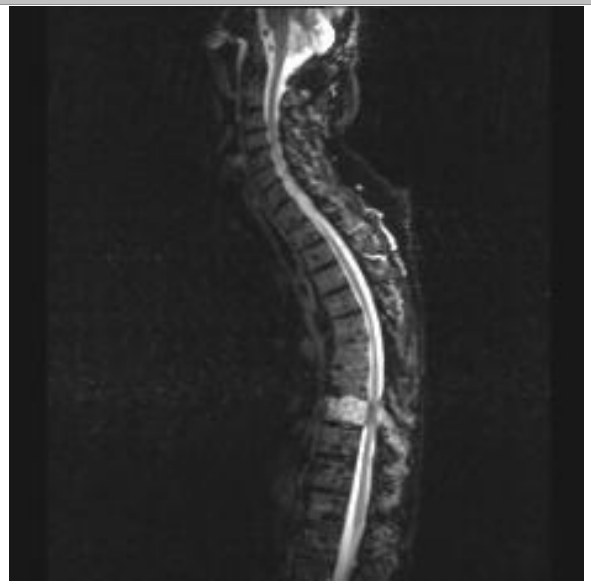
Authors' conclusions

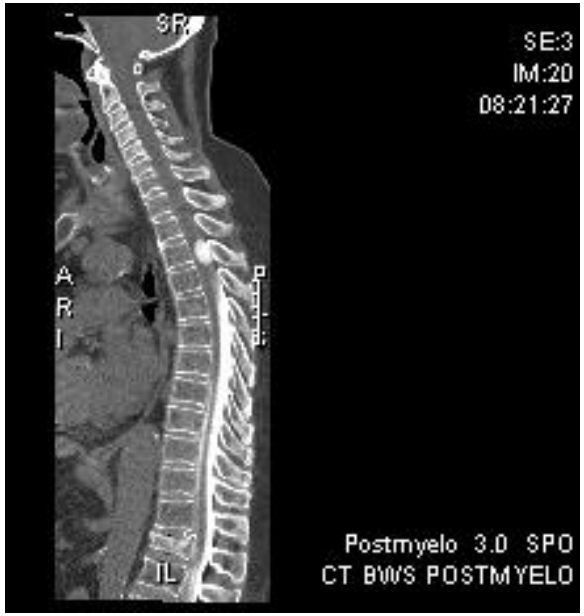
Overall, corticosteroids significantly reduced rates of mortality, severe hearing loss and neurological sequelae. In adults with community-acquired bacterial meningitis, corticosteroid therapy should be administered in conjunction with the first antibiotic dose. In children, data support the use of adjunctive corticosteroids in children in high-income countries. We found no beneficial effect of corticosteroids for children in low-income countries.

Ascending paresis– important spinal causes

PB 2013

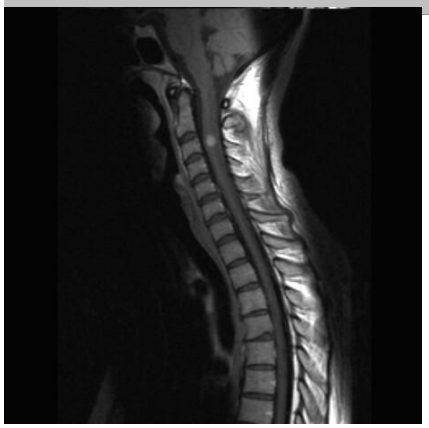
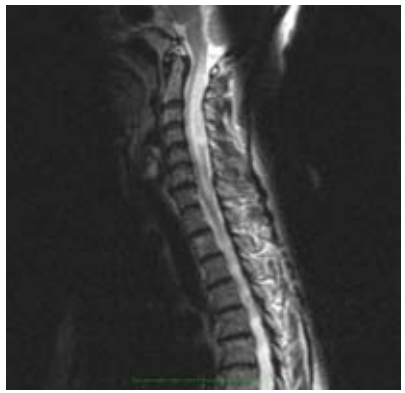
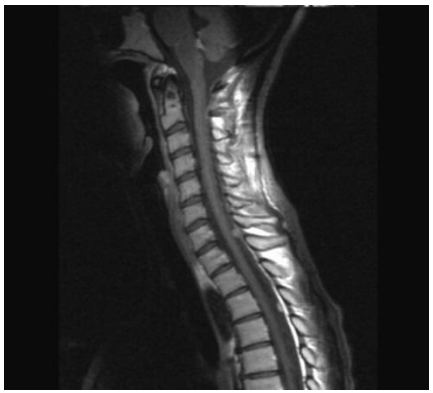
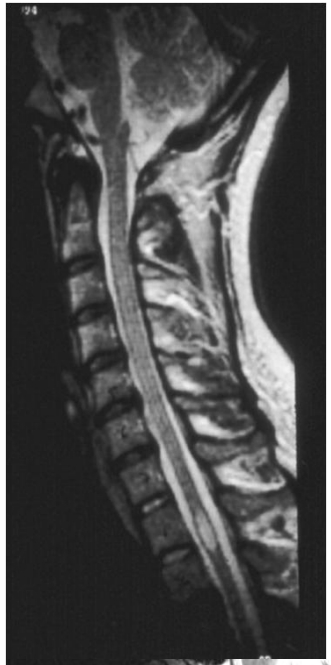
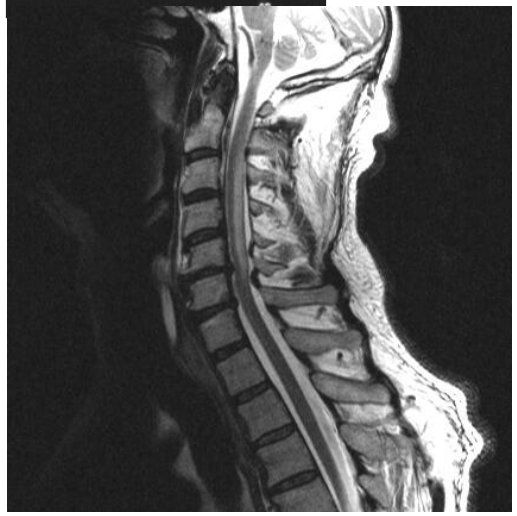
- Space occupying lesions
  - Disc prolapse or bone
  - Metastases
  - Meningeoma
  - Neurinoma
- Inflammatory lesions:
  - Myelitis (infection – parainfektiös)
  - MS, NMO
  - Epidural abscess
- Ischemia:
  - Anterior spinal artery syndrome
  - Radiculomedullary artery syndrome
  - Leriche syndrome
- Spinal hemorrhage





Myelitis and differential diagnoses

PB 2013

- Subacute ascending paresis with sensory level and autonomic dysfunction
- Lhermitte sign

Ascending paresis – peripheral nerves

PB 2013

- AIDP (GBS)
- Borreliosis
- Multiplex mononeuritis (Vaskulitis)
- Diphtheria
- Acute Porphyria

Ascending paresis – neuromuscular synapsis

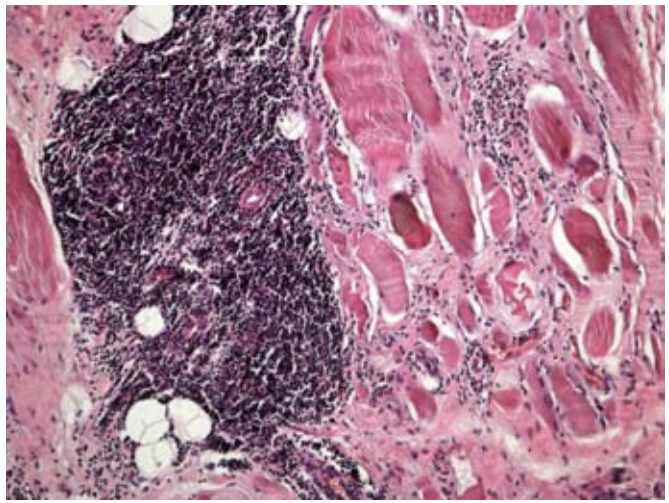
PB 2013

- Myasthenic crisis (with infection, wrong medication)
- Botulism
- Hypermagnesiemia
- Organophosphate intoxication
- LEMS

Ascending paresis – muscle

PB 2013

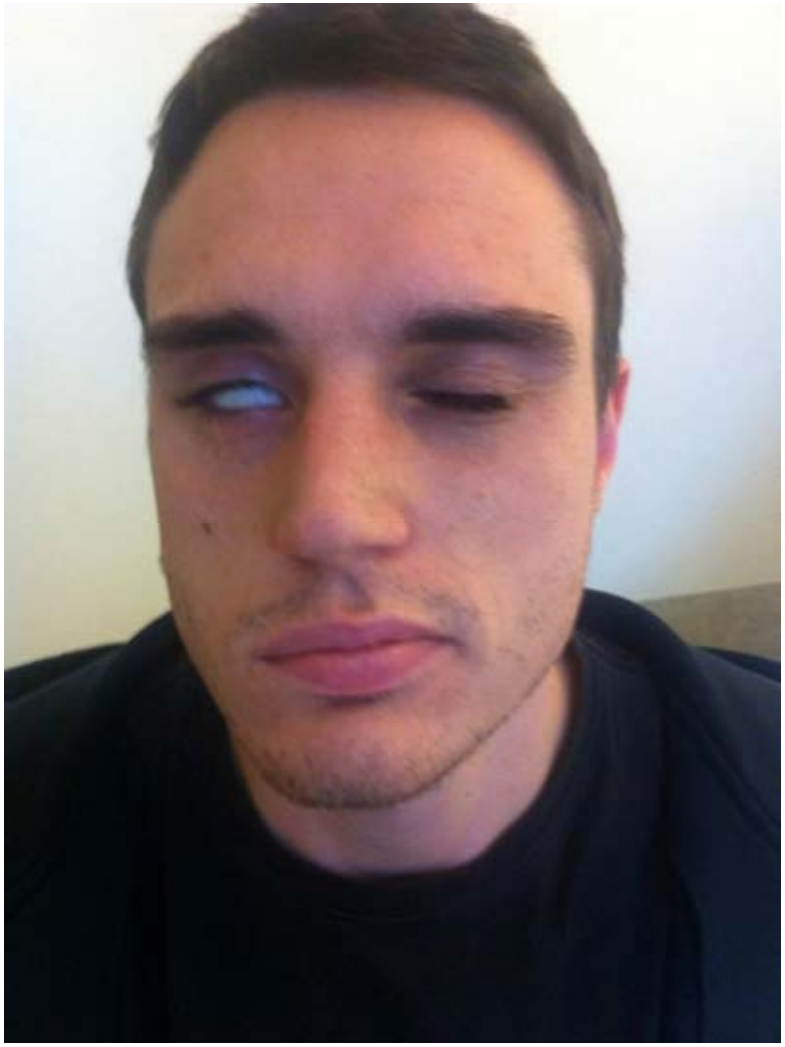
- Acute necrotizing myopathy (with alcohol intoxication)
- Acute polymyositis
- Rhabdomyolysis (concussion, exertion, drug induced)
- Acute metabolic myopathies
- Periodic paralysis



Ascending paresis – other causes:

PB 2013

- Metabolism:
  - Hypoglycemia
  - Hypermagnesiemia
  - Hypophosphatemia
- Intoxication:
  - Medication
  - Drugs
- Toxins
  - Botulism
  - Venom (snakes, insects)
- Conversion disorder



Many possibilities in an urgent situation

- How do I handle the situation?
- What to do first?
- How do I get the correct diagnosis?
- How can I avoid making mistakes?
- What apparative measures are necessary?

Ascending paresis – important questions

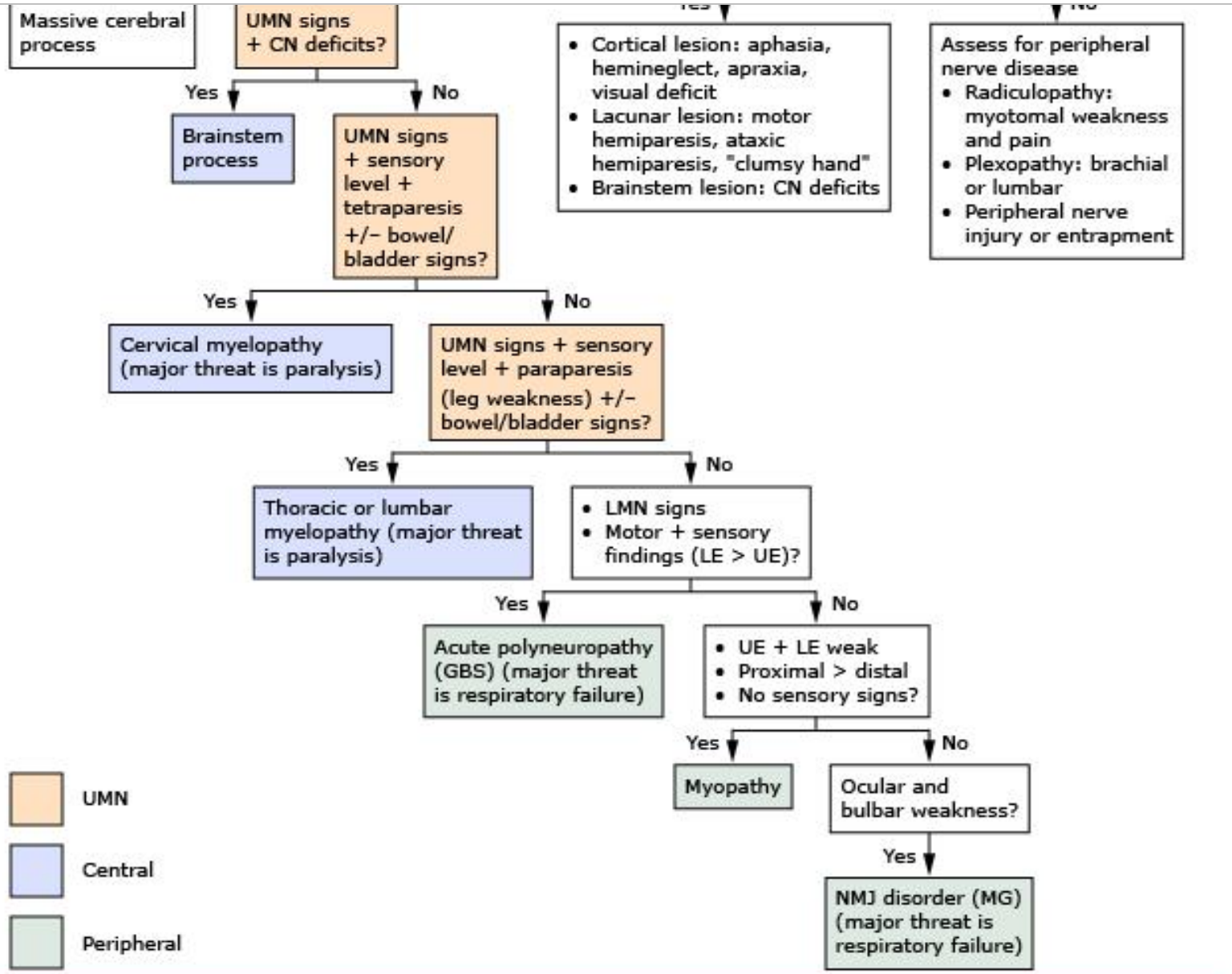
PB 2013

- Since when? How did it occur?
- Where did it start?
- Occurrence during sports or other exertions?
- Recent trauma?
- Pain? Where?
- Preceding infection?
- Similar events in the past? In connection with?
- Medication, drugs?
- Insect bites?

Ascending paresis – important neurologic findings

PB 2013

- Ventilation problems?
- Heart? Circulation?
- Sensory level?
- Pyramidal tract signs?
- Distribution of pareses?
- Reflexes?
- Cranial nerve symptoms?
- Bladder, bowel?
- Other autonomic disturbances?



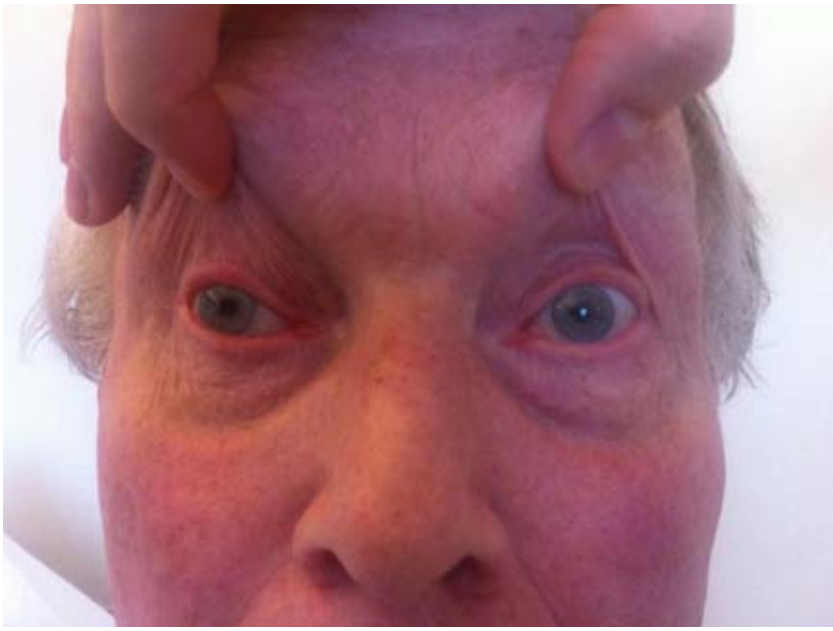
ACS: acute coronary syndrome; CN: cranial nerve; GBS: Guillan-Barré syndrome; ICH: intracerebral hemorrhage; LE: lower extremities; LMN: lower motor neuron; MG: myasthenia gravis; NMJ: neuromuscular junction; UE: upper extremities; UMN: upper motor neuron.

Data from: Asimos AW. Weakness: A systematic approach to acute, non-traumatic, neurologic and neuromuscular causes. Emerg Med Pract 2002; 4:1.

Neurologic signs of brain stem disorder

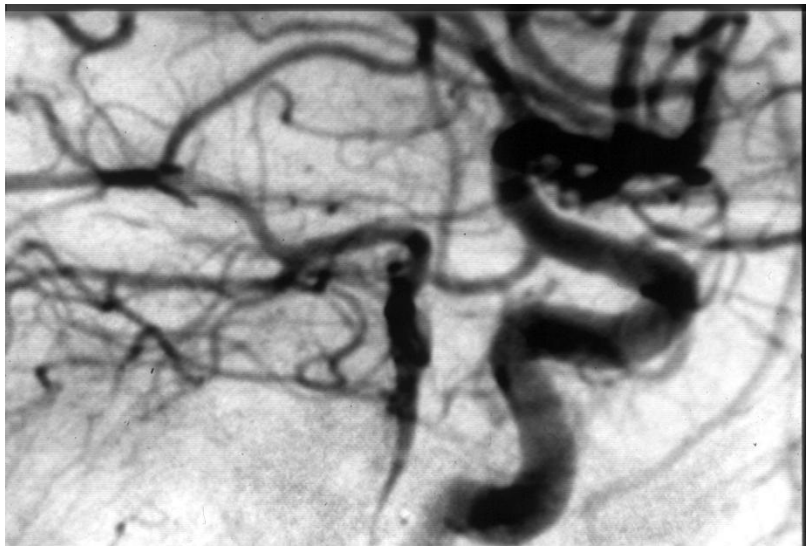
PB 2013

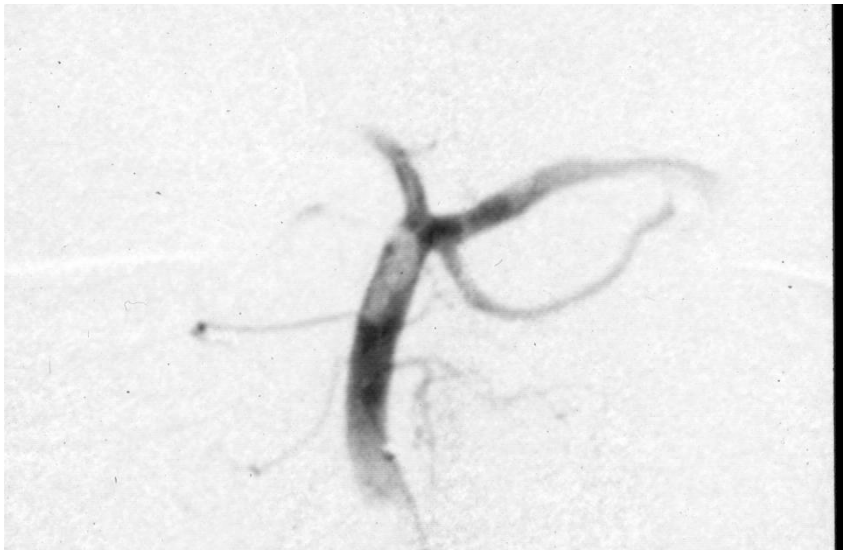
- Cranial nerve symptoms, i.e. oculomotor signs, vertigo
- Pyramidal tract signs
- Para- or tetraparesis
- Disturbance of consciousness

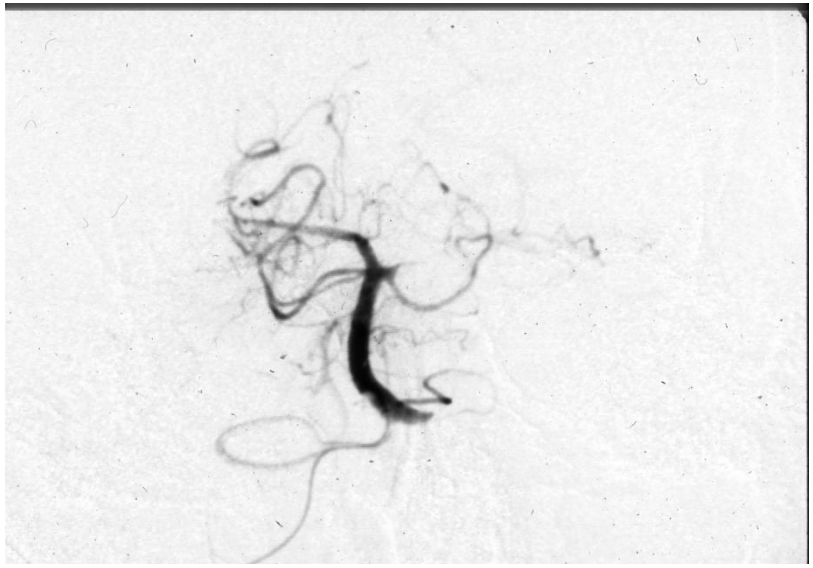


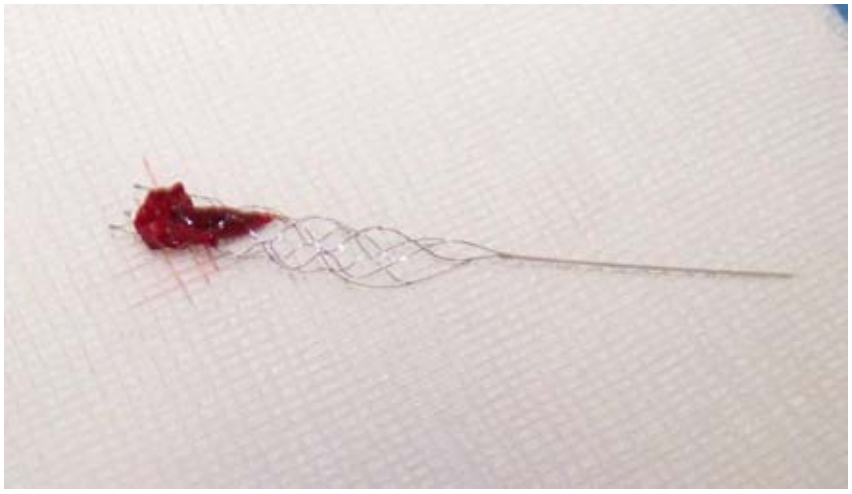
BA occlusion

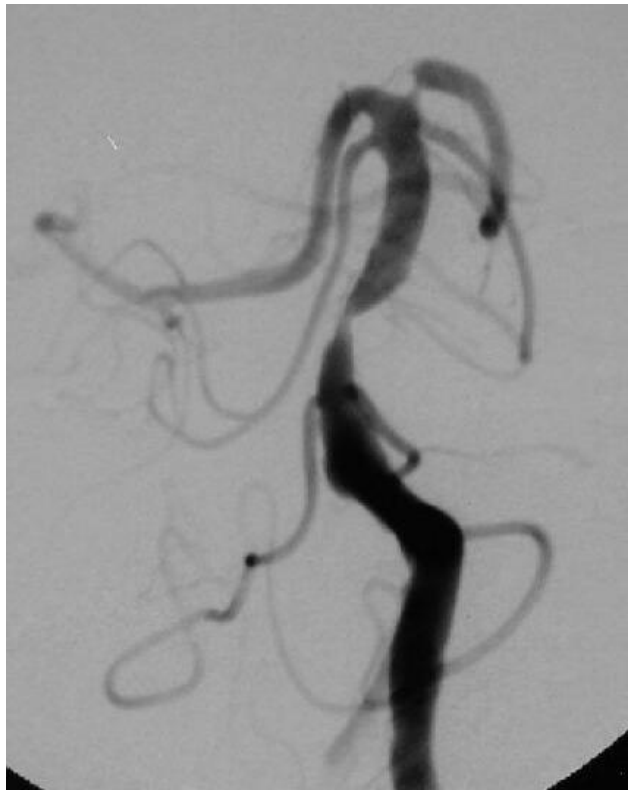
PB 2013

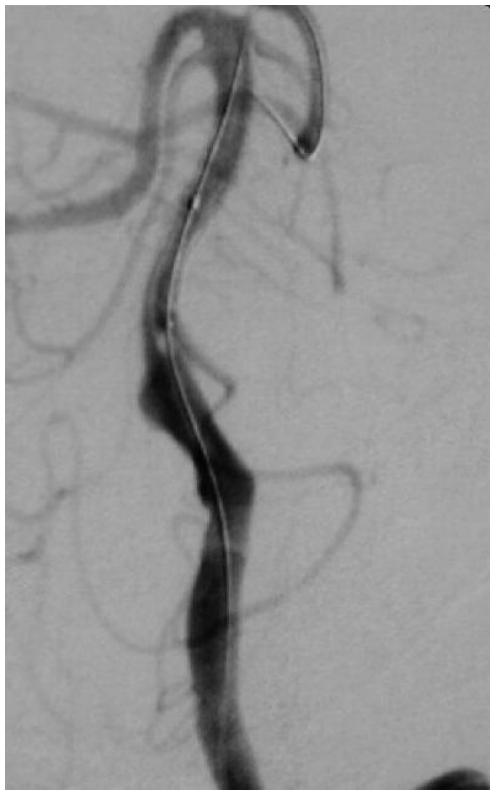


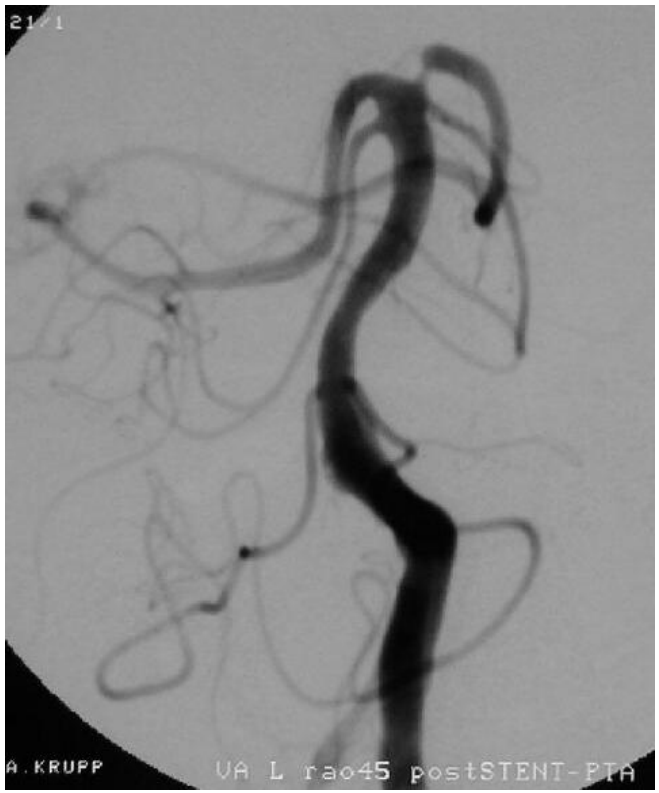








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A.KRUPP
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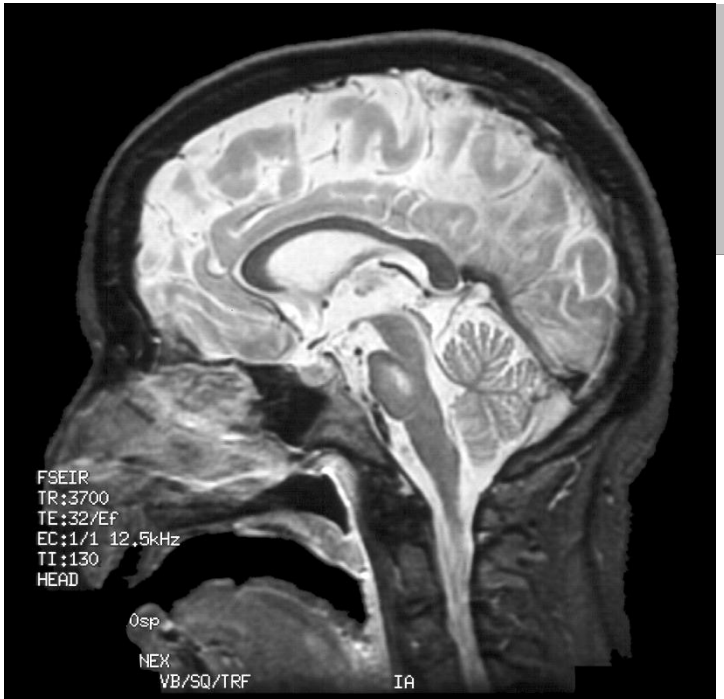
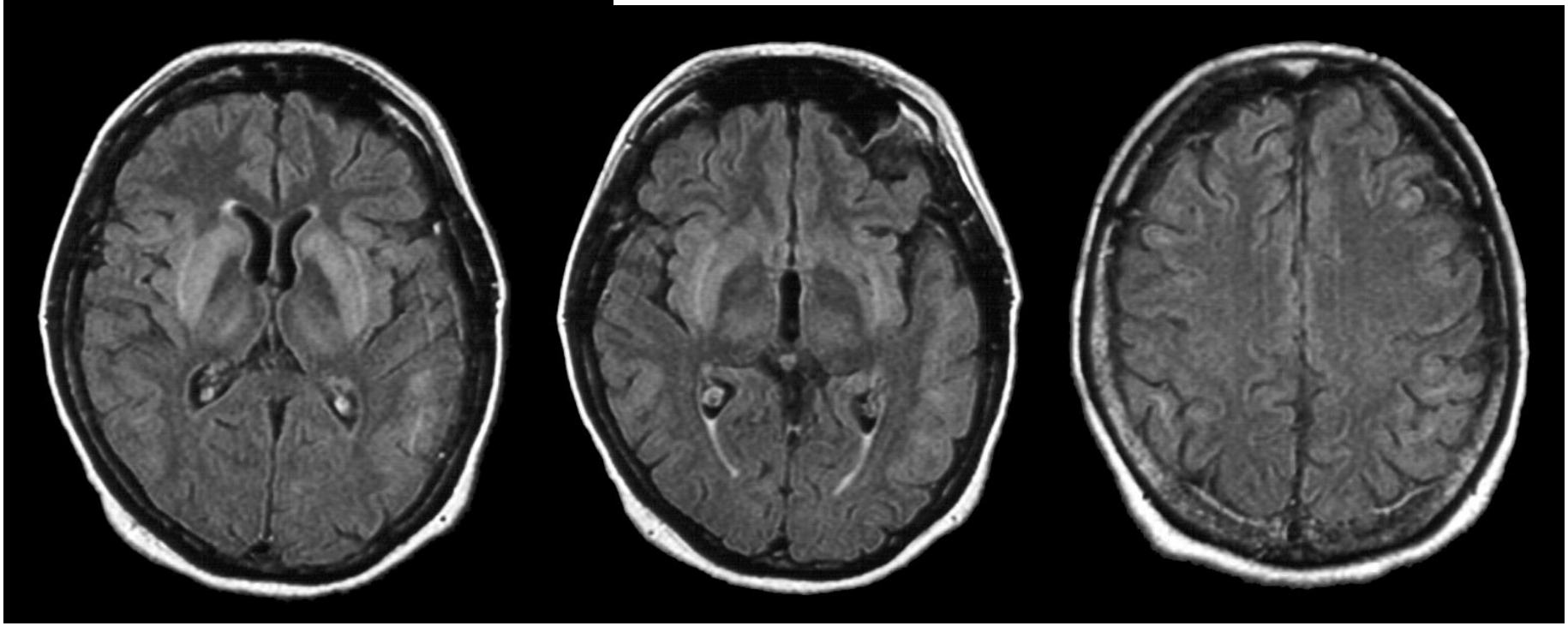
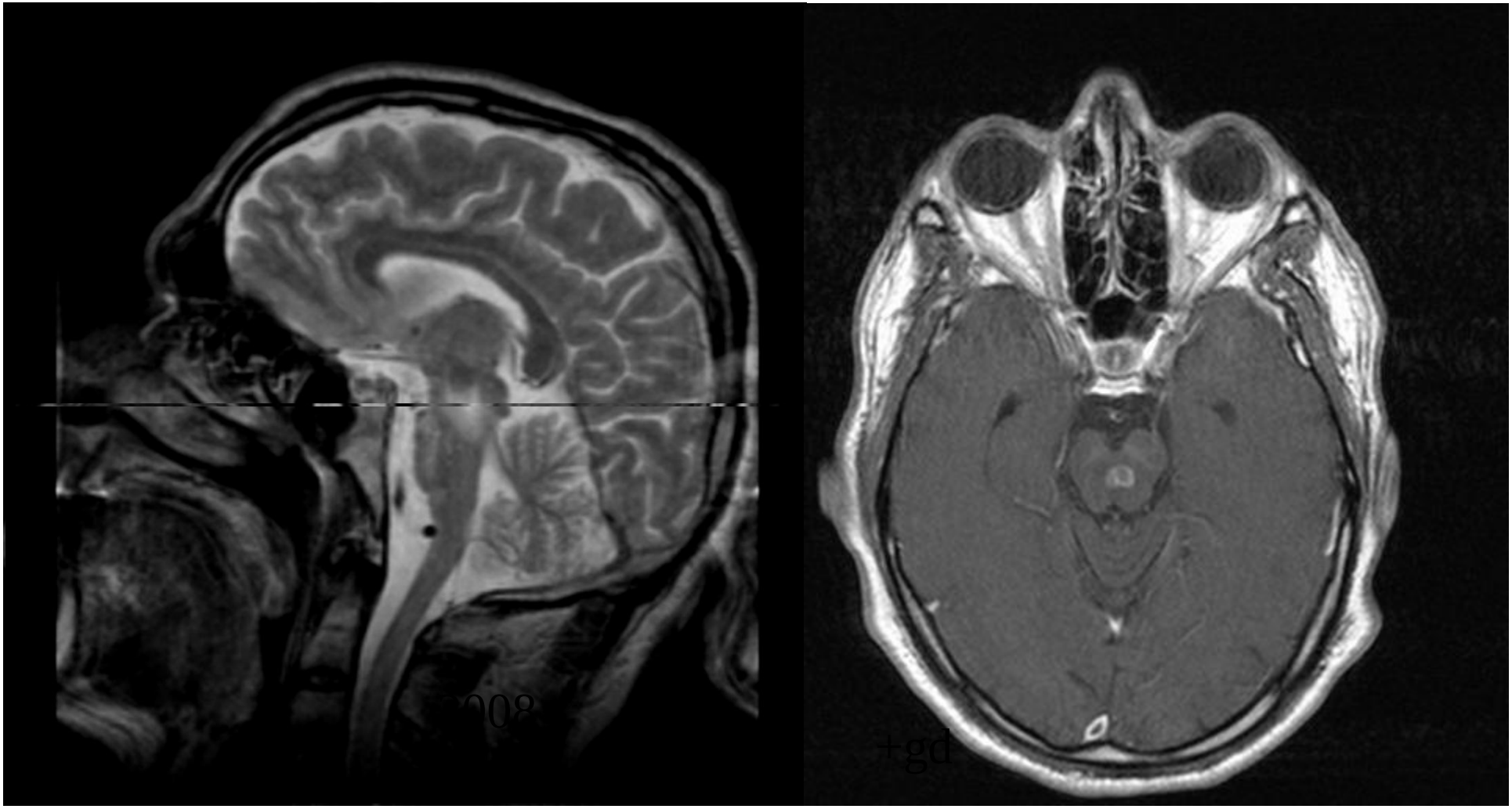
Central pontine myelinolysis





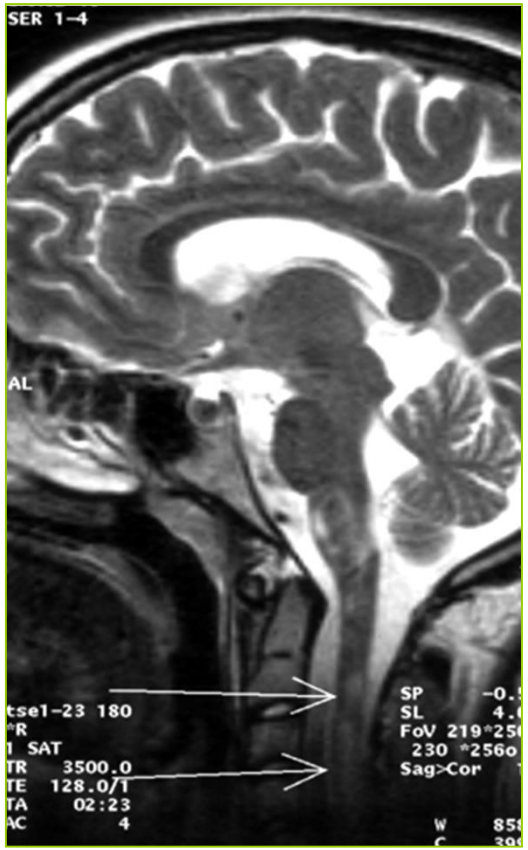
Brain stem affection in Behcet`s disease

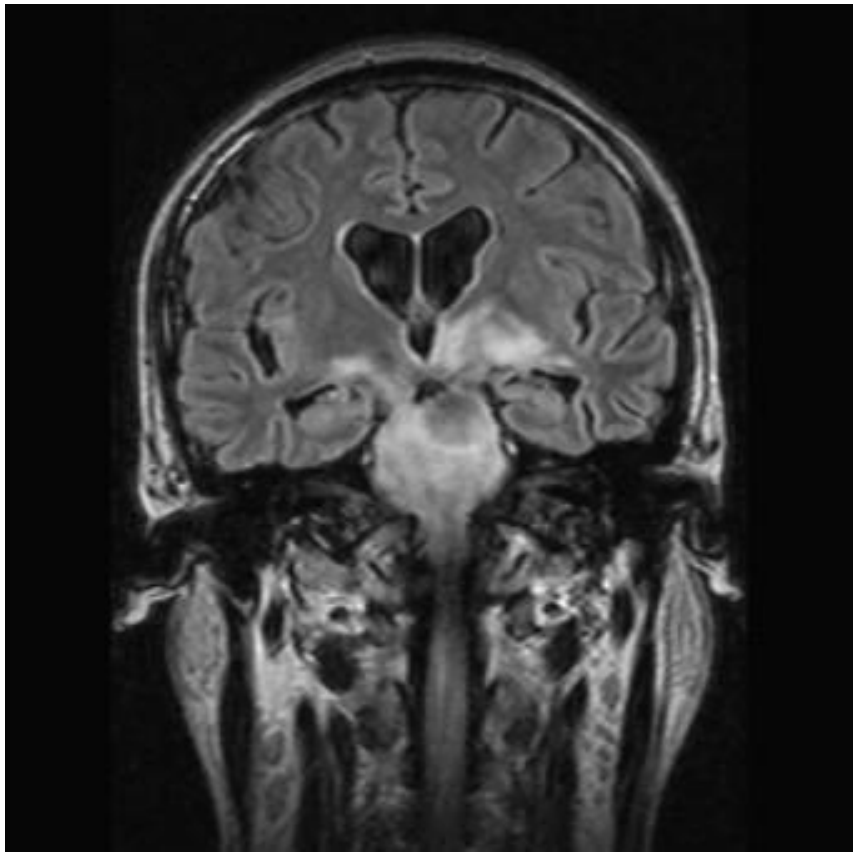
PB 2013



Infectious brain stem affection: tuberculosis

PB 2013



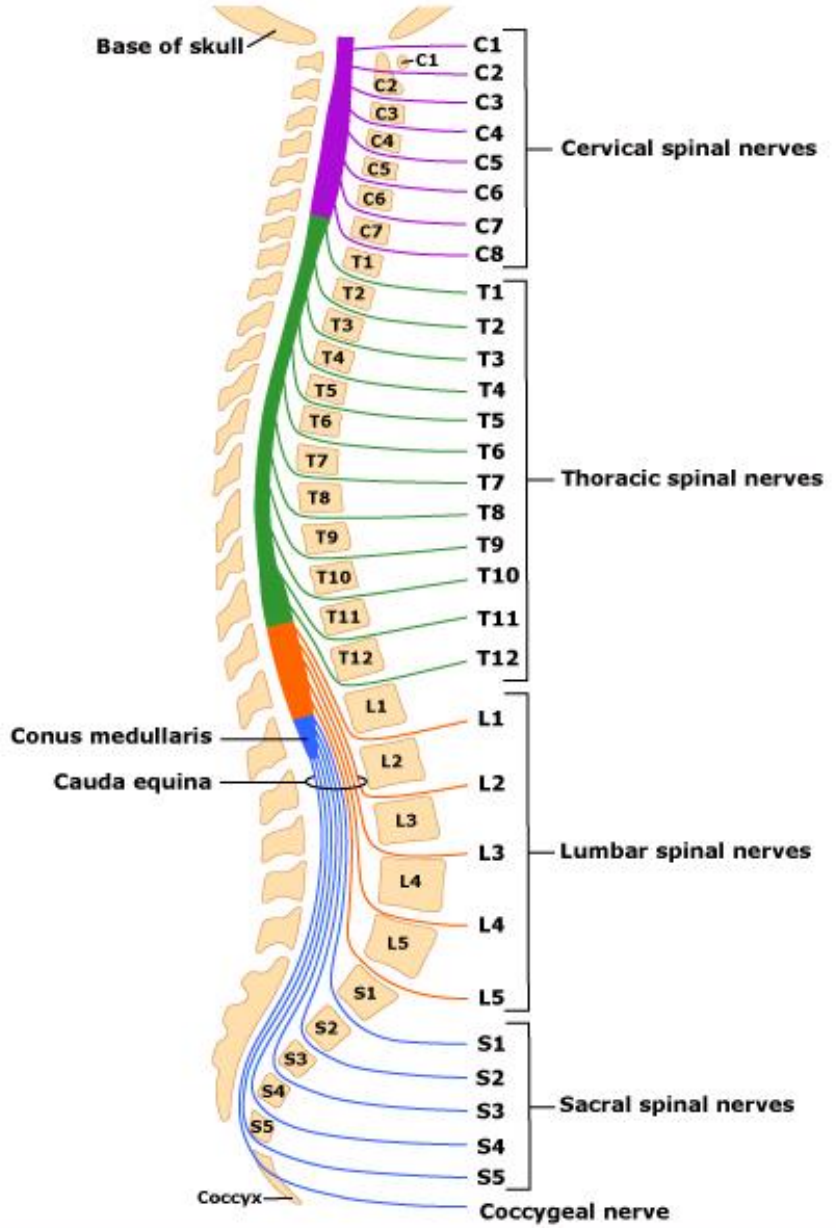


Neurologic signs of spinal disorder

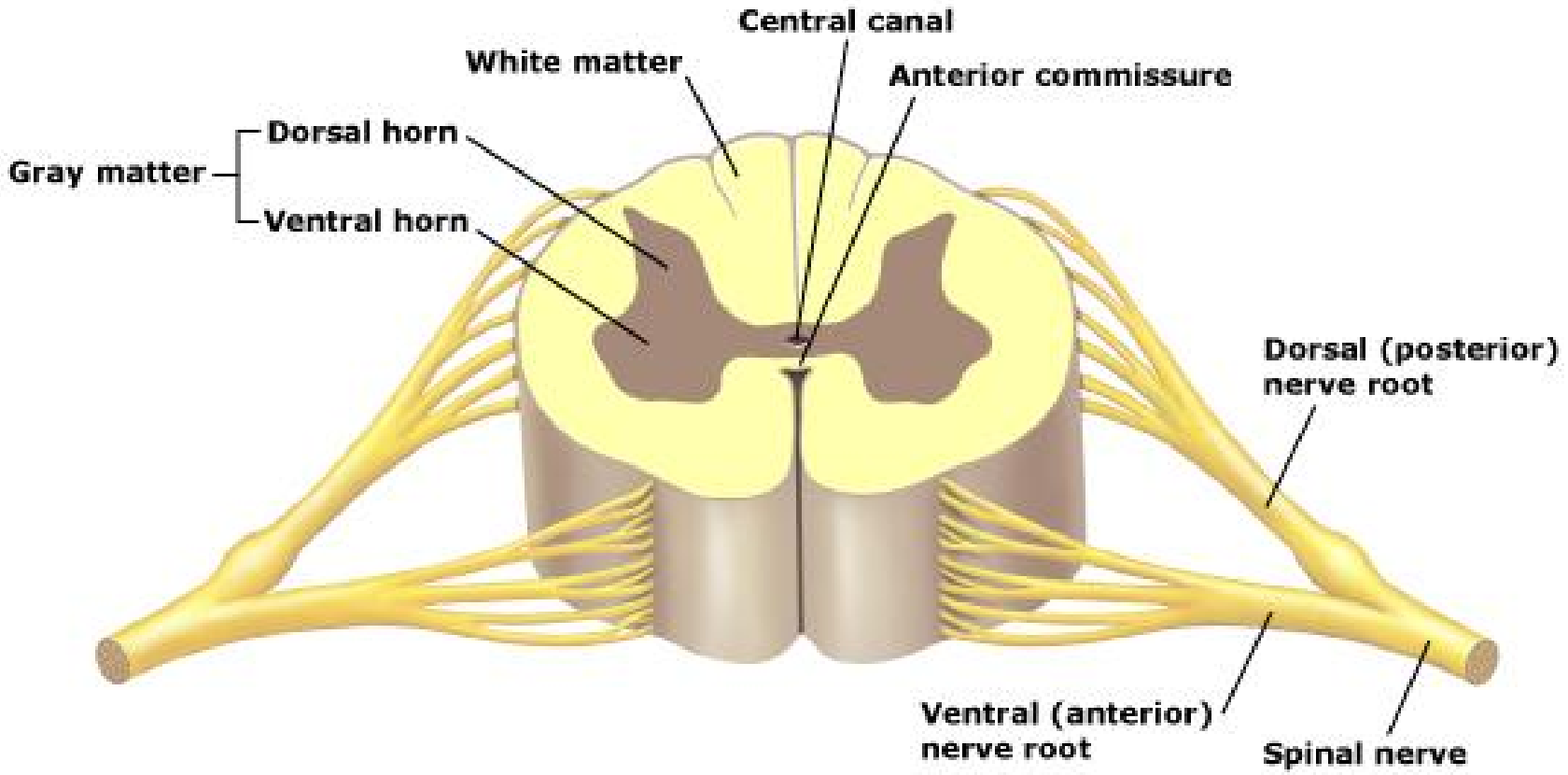
PB 2013

- Sensory loss all qualities or temperature and pain
- Pyramidal tract signs
- Para- or tetraparesis
- Bladder and bowel problems
- Back pain

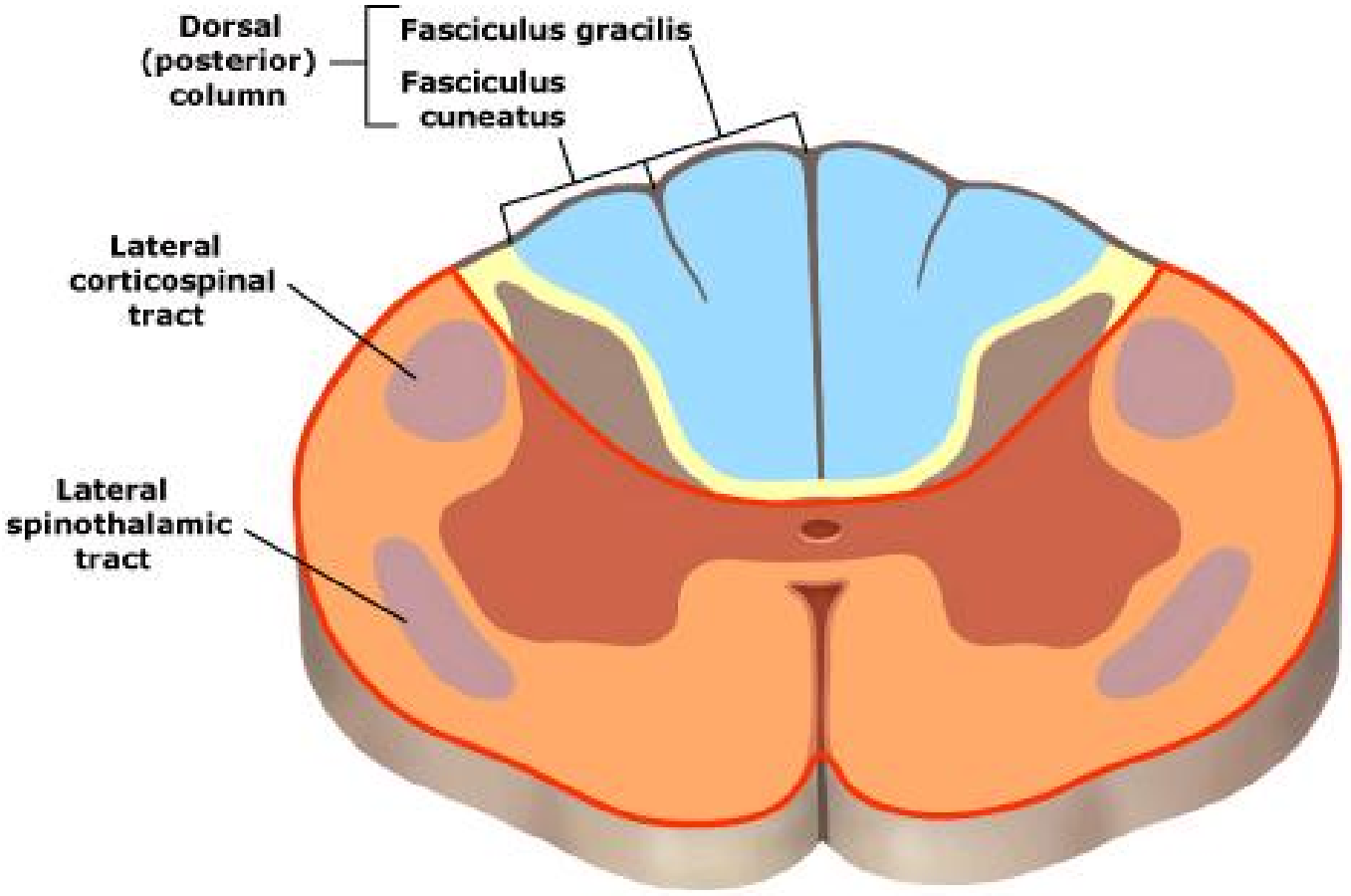
Longitudinal organization of spinal cord, spinal nerves, and vertebrae



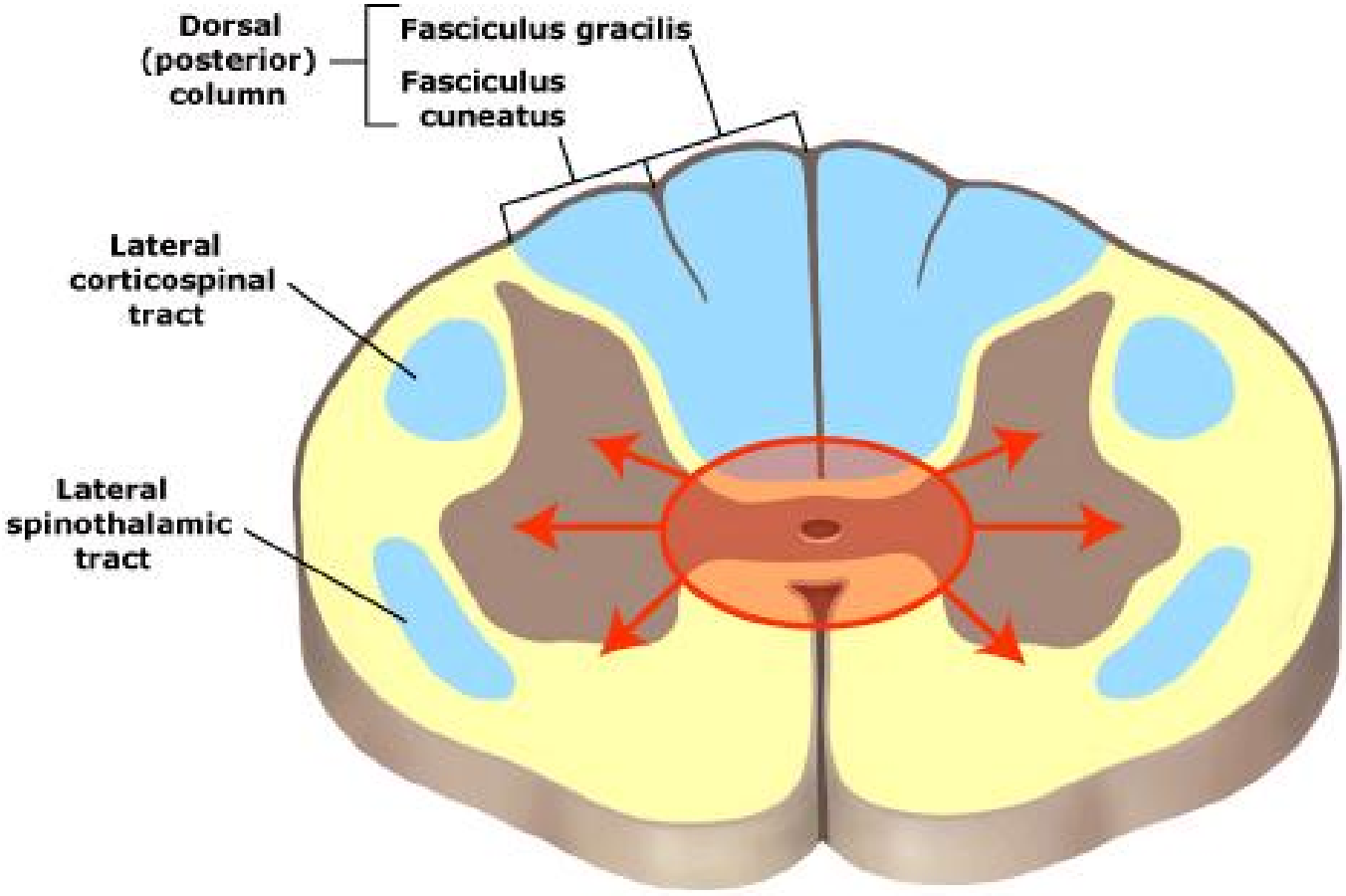
Cross-sectional anatomy of the spinal cord



Location of lesion in ventral cord syndrome

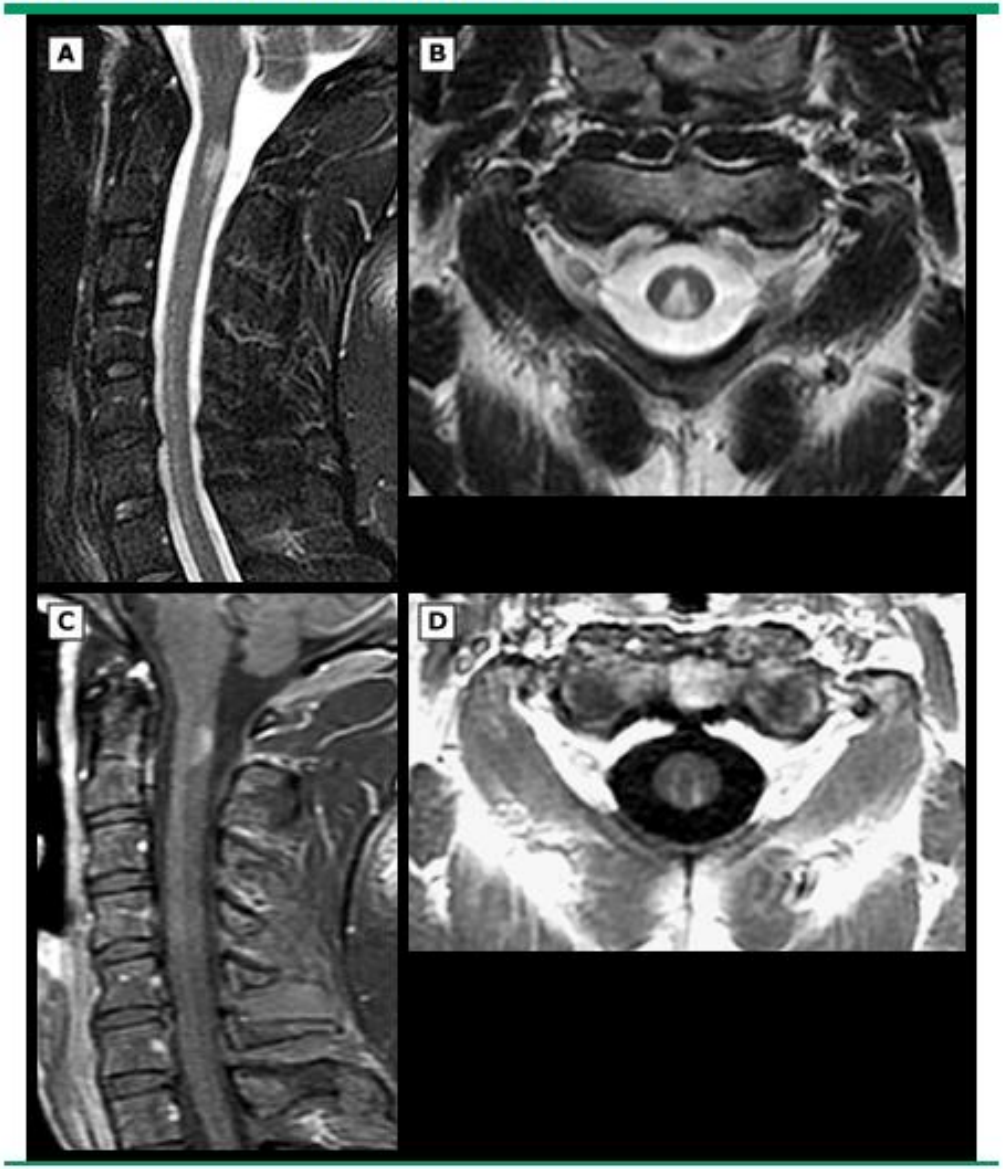


Location of lesion in central cord syndrome



syndrome	below affected level; bladder dysfunction	myelitis, epidural metastasis
Dorsal cord syndrome	Loss of proprioception, vibratory sensation; variable weakness and bladder dysfunction	Tabes dorsalis, Friedreich ataxia, subacute combined degeneration, AIDS myelopathy, epidural metastases, cervical spondylotic myelopathy, multiple sclerosis
Ventral cord syndrome (anterior spinal artery syndrome)	Loss of pain and temperature sensation, weakness, bladder dysfunction	Spinal cord infarction, disc herniation, radiation myelopathy, HTLV-1
Brown Sequard syndrome	Ipsilateral weakness and loss of proprioception; contralateral loss of pain and temperature sensation	Knife or bullet injury, multiple sclerosis
Central cord syndrome	Segmental loss of pain and temperature, weakness often greater in the arms than legs	Syringomyelia, intramedullary tumor, acute injury in cervical spondylotic myelopathy
Pure motor syndrome	Weakness without sensory disturbance	Poliomyelitis, amyotrophic lateral sclerosis, HTLV-1, hereditary spastic paraplegia, lathyrism
Conus medullaris syndrome	Bladder and rectal dysfunction, saddle anesthesia	Disc herniation, trauma, tumors
Cauda equina syndrome	Asymmetric multiradicular pain, leg weakness, and sensory loss; bladder dysfunction	Disc herniation, arachnoiditis, tumor, lumbar spine stenosis

a 37 year old man with multiple sclerosis

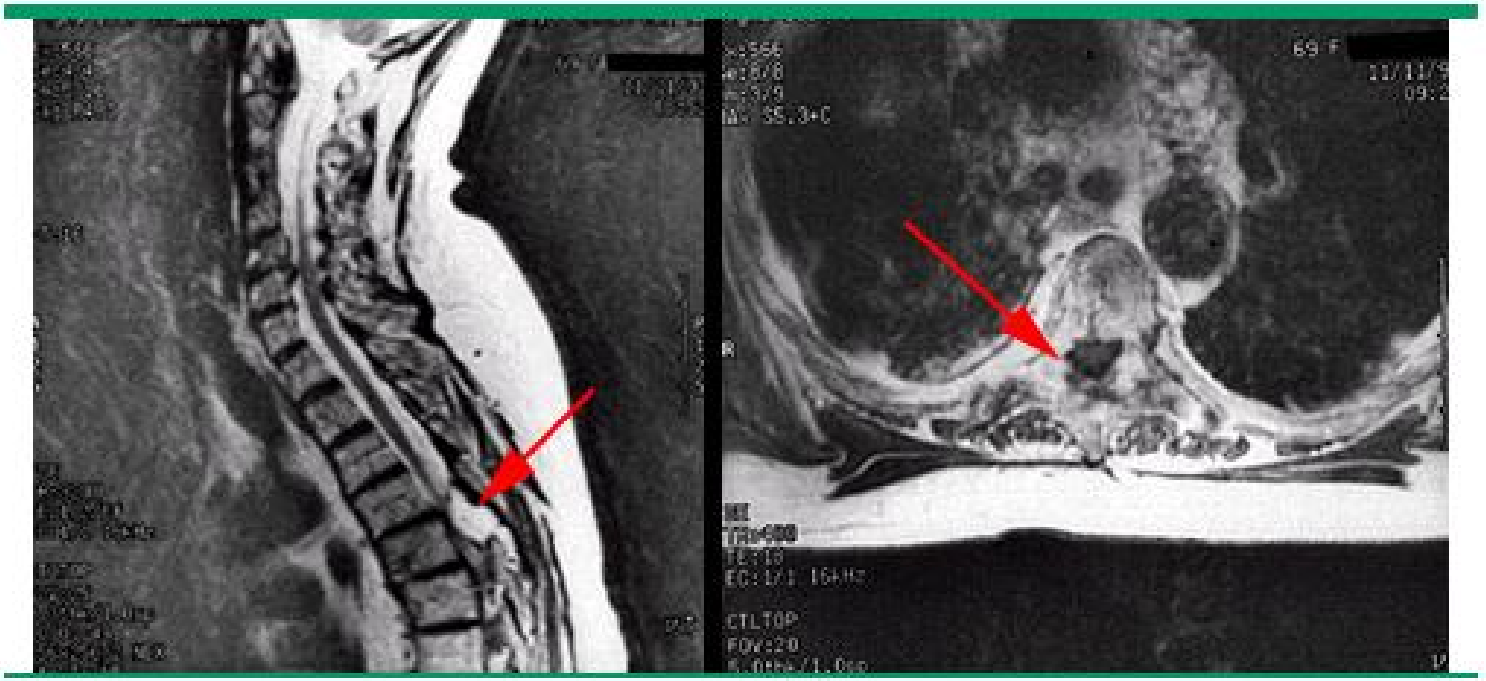


T2-weighted sagittal (A) and axial (B) images show a focus of hyperintensity in the posterior columns of the cervical spinal cord at the C2 level. Post-gadolinium T1-weighted sagittal (C) and axial (D) images demonstrate enhancement consistent with an active plaque.

Concurrent systemic infection	CSF acid-fast bacilli smear and tuberculous culture
Immunocompromised state	CSF HSV, VZV, and HTLV-1 antibodies
Recurrent genital infection	CSF anti-*Borrelia burgdorferi* antibodies
Symptoms of zoster radiculopathy	CSF VDRL
Adenopathy	CSF India ink and fungal culture
Residence in area endemic for parasitic infections	Chest radiograph
Lymphadenopathy	Serology for antibodies to HIV, HSV, VZV, HTLV-1, *B. burgdorferi*
	Serology for hepatitis A, B, C, and *Mycoplasma*
	Consider serology for parasites
	Blood cultures
Systemic inflammatory disease (vasculitis, collagen vascular diseases, mixed connective tissue disease)	
Rash	Serum ACE
Oral or genital ulcers	Auto-antibodies: ANA, ds-DNA, Ro/SSA, La/SSB, Sm, RNP
Adenopathy	Complement levels
Livedo reticularis	Urinalysis with microscopic analysis for hematuria
Serositis	Lip/salivary gland biopsy
Photosensitivity	Chest CT with intravenous contrast
History of arterial and venous thrombosis	
Multiple sclerosis	
Previous demyelination event	Brain MRI
Incomplete deficit clinically with MRI abnormality ≤2 spinal segments and <50 percent of cord diameter	Evoked potentials
	CSF oligoclonal bands and IgG index
Neuromyelitis optica (Devic's disease)	
Optic neuritis	Evoked potentials
Clinical deficit with MRI abnormality ≥3 spinal segments	Brain MRI (usually negative)
	NMO-IgG testing
Idiopathic transverse myelitis	
No clinical or paraclinical features suggestive of another diagnostic category	Evoked potentials
	Electromyography/nerve conduction velocity

ACE: angiotensin-converting enzyme; ANA: anti-nuclear antibodies; CMV: cytomegalovirus; CSF: cerebrospinal fluid; EBV: Epstein-Barr virus; HHV: human herpes virus; HIV: humam immunodeficiency virus; HSV: herpes simplex virus; HTLV-1: human T-cell lymphotropic virus 1; IgG: immunoglobulin G; NMO-IgG: neuromyelitis optica IgG autoantibody; VDRL: Veneral Disease Research Laboratory; VZV: varicella zoster virus.

MRI image of a patient with an epidural spinal cord compression



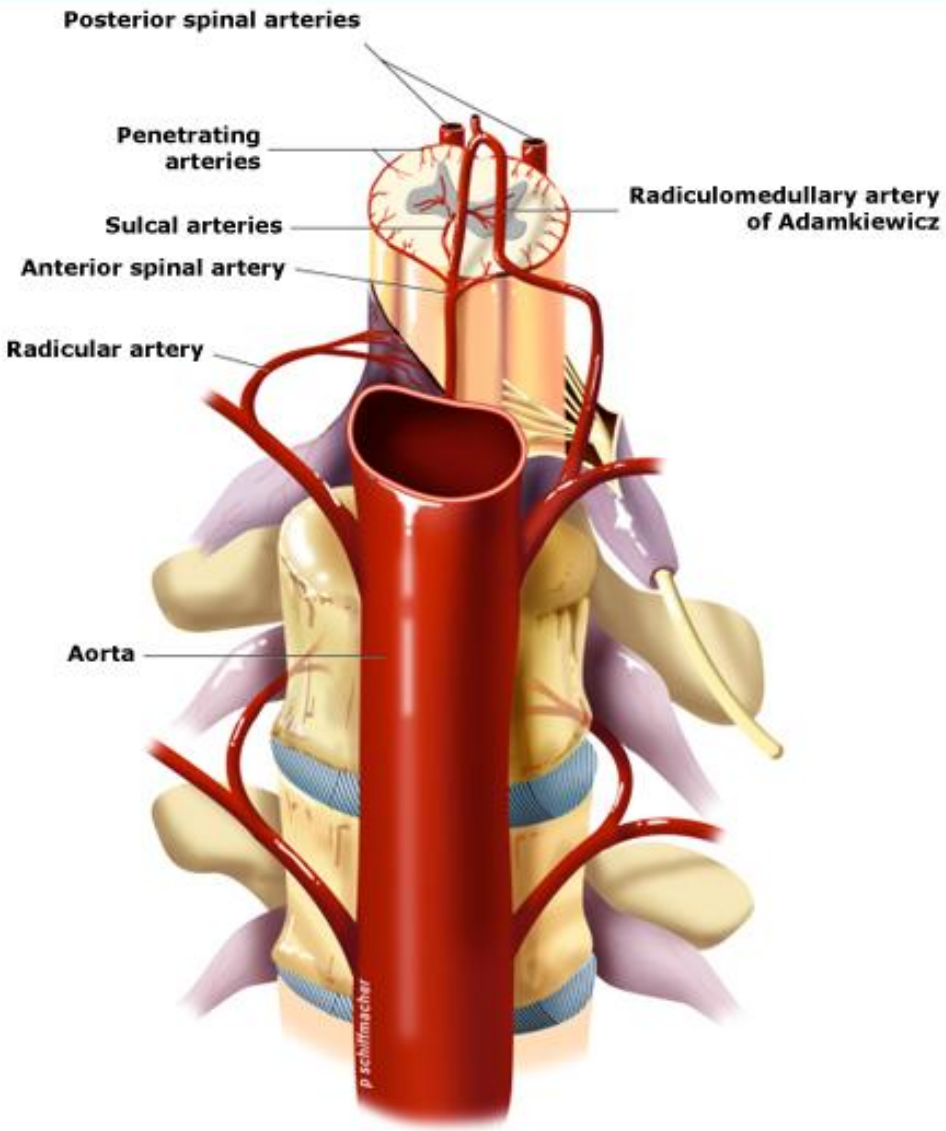
Sagittal (left panel) and axial (right panel) gadolinium-enhanced spinal MR scan of a 69-year-old women with a remote history of breast cancer, interscapular back pain for one month, and a normal neurologic examination. The scans demonstrate a large epidural lesion compressing the spinal cord (arrows).

Courtesy of David Schiff, MD.

myelitis	young adults		syndrome	
Viral myelitis	Any age	Acute-subacute	Pure motor syndrome or Segmental cord syndrome	MRI and CSF
Epidural abscess	Any age	Subacute; may worsen abruptly	Segmental cord syndrome	MRI
Infarction	Usually >60 years	Abrupt onset	Anterior cord syndrome	MRI with diffusion weighted sequences
Vascular malformation	>40 years (dural fistula) 20's (intramedullary AVM)	Acute and/or stepwise	Radicuomyelopathy	MRI, spinal angiography
Subacute combined degeneration	Any age	Slowly progressive	Dorsal cord syndrome	Vitamin B12 levels
Radiation	Any age	Slowly progressive; beginning 6-12 months after radiation therapy	Segmental cord syndrome or Ventral cord syndrome	MRI, clinical history
Syringomyelia	Children, young adults	Slowly progressive	Central cord syndrome	MRI
Epidural metastasis	Usually >50 years	Subacute, may worsen abruptly	Segmental cord syndrome	MRI
Intramedullary tumor	Young adults	Slowly progressive	Central cord syndrome	MRI with gadolinium enhancement
ALS	Usually >60 years	Slowly progressive	Pure motor syndrome	Electromyography

MRI: magnetic resonance imaging; CSF: cerebrospinal fluid; AVM: arteriovenous malformation; ALS: amyotrophic lateral sclerosis.
* This is a partial list of causes. Please see topic "Disorders affecting the spinal cord" for a more complete differential diagnosis.

Arterial supply of the spinal cord



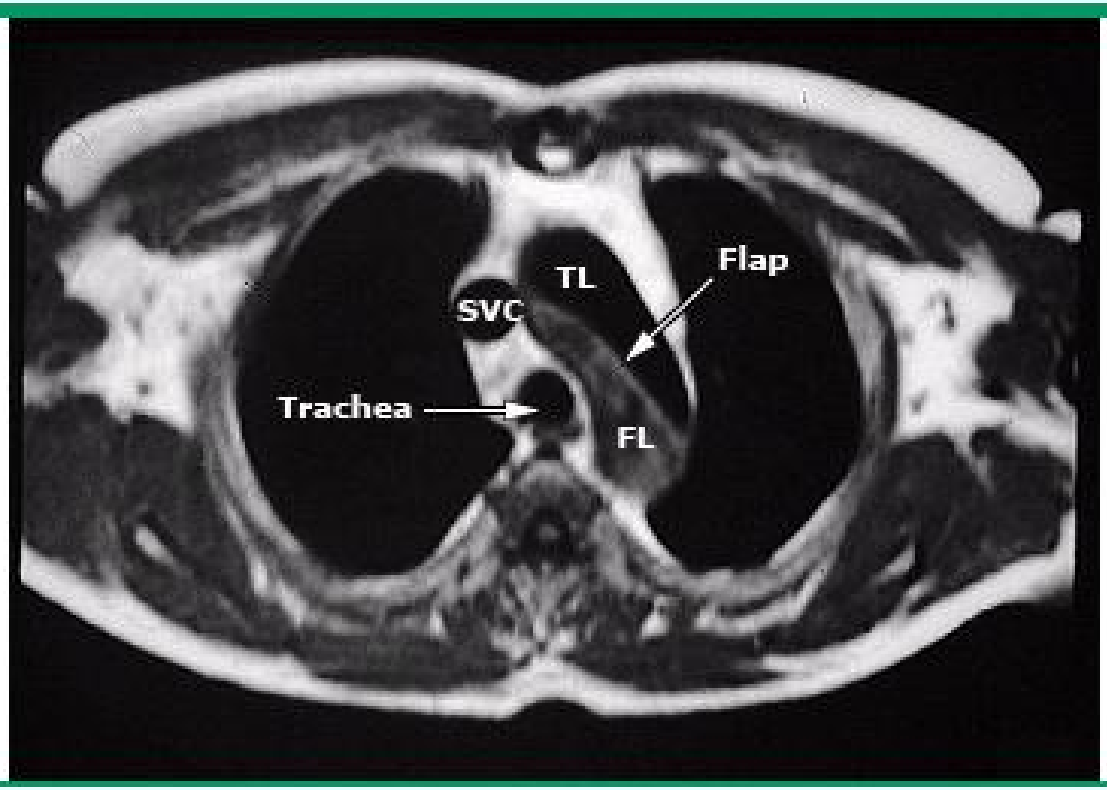
Reproduced with permission from: Prasad S, Price RS, Kranick SM, et al. Clinical Reasoning: A 59-year-old woman with acute paraplegia. Neurology 2007; 69:E41. Copyright © 2007 Lippincott Williams & Wilkins.

Causes of spinal cord infarction

Aorta disease, procedures
Aortic surgery
Thoracic endovascular aortia repair (TEVAR)
Aortic dissection
Traumatic rupture of the aorta
Aortic thrombosis
Aortic aneurysm
Coarctation of the aorta
Aortography
Systemic hypoperfusion
Cardiac arrest
Systemic bleeding
Cardiogenic embolism
Atrial myxoma
Mitral valve disease
Patent foramen ovale
Bacterial endocarditis
Cardiac catheterization
Vasculitis
Systemic lupus erythematosus
Polyarteritis nodosa
Behcet syndrome
Giant cell arteritis

Infection
Bacterial meningitis
Syphilis
Mucormycosis
Hematologic disease
Hypercoagualable conditions
Sickle cell anemia
Non-aortic surgeries
Spine disease
Spine surgery
Cervical spondylosis
Fibrocartilagenous embolism
Epidural steroid injections
Miscellaneous
Cocaine abuse
Vertebral artery dissection
Spinal vascular malformation
Decompression sickness

Aortic dissection on magnetic resonance imaging



Transverse (axial) spin-echo CMR in a patient with an aortic dissection at the level of the aortic arch. The true lumen (TL), false lumen (FL), and intimal flap can be easily identified. The trachea and superior vena cava (SVC) are also seen.

Courtesy of Warren Manning, MD.

1. Detailed medical history and complete physical examination (whenever possible)	I
2. Intravenous line, blood sample (CK, troponin I, myoglobin, WBC, D-dimer, hematocrit, LDH)	I
3. ECG: documentation of ischemia	I
4. Heart rate and blood pressure (BP) monitoring	I
5. Pain relief (morphine sulphate)	I
6. Reduction of systolic blood pressure using beta blockers (IV propranolol, metoprolol, esmolol, or labetalol)	I
7. Transfer to intensive care unit	I
8. In patients with severe hypertension additional vasodilator (IV sodium nitroprusside to titrate BP to 100-120 mmHg)	I
9. In patients with obstructive pulmonary disease, blood pressure lowering with calcium channel blockers	II
10. Imaging in patients with ECG signs of ischemia before thrombolysis if aortic pathology is suspected	II
11. Chest x-ray	III

Classification

Class I: Conditions for which there is evidence and/or general agreement that a given procedure or treatment is useful and effective.

Class II: Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.

Class IIa: Weight of evidence/opinion is in favor of usefulness/efficacy.

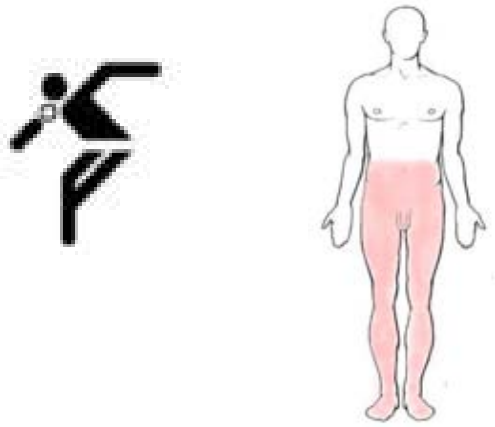
Class IIb: Usefulness/efficacy less well established by evidence/opinion.

Class III: Conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful and in some cases may be harmful.

Data reproduced with permission from Erbel, R, Alfonso, F, Boileau, C, et al, Eur Heart J 2001; 22:1642.

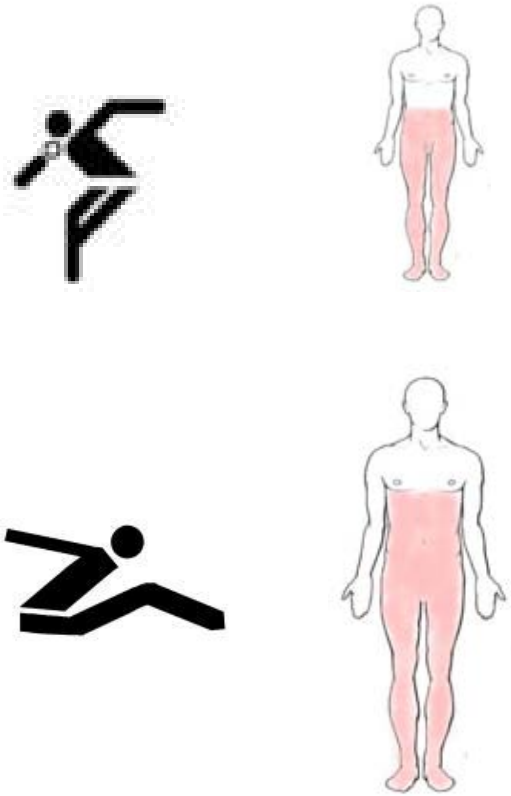
16 yr old high school absolvent

- 4 hours after sports competition (ball encounter) thoracic pain
- Within 20 min paresis left leg
- Ascending pareses of both legs
- Loss of pain and temperature sensation below Th 9
- Bladder disturbance
- MRI, CSF: normal



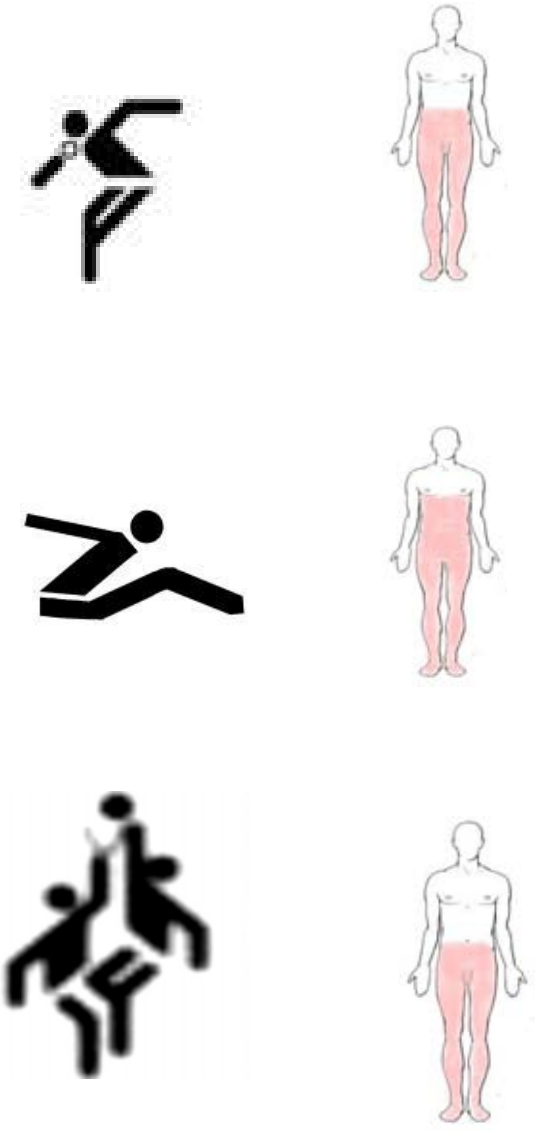
16 yr old female

- During long jumping breast pain
- Ascending paresis of both legs
- Loss of pain and temperature sensation below Th 6
- Bladder disturbance
- MRI, CSF: normal

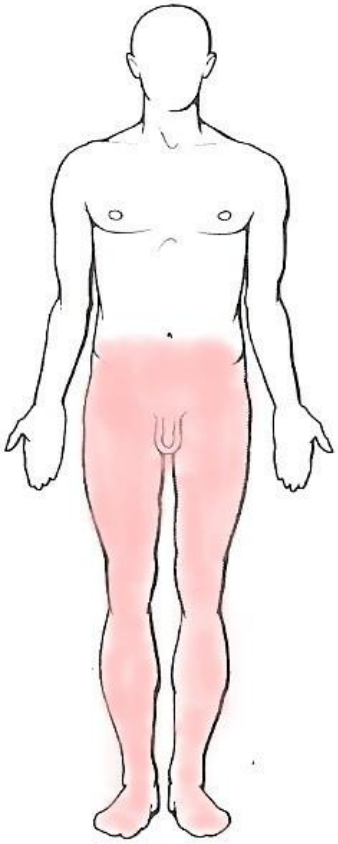


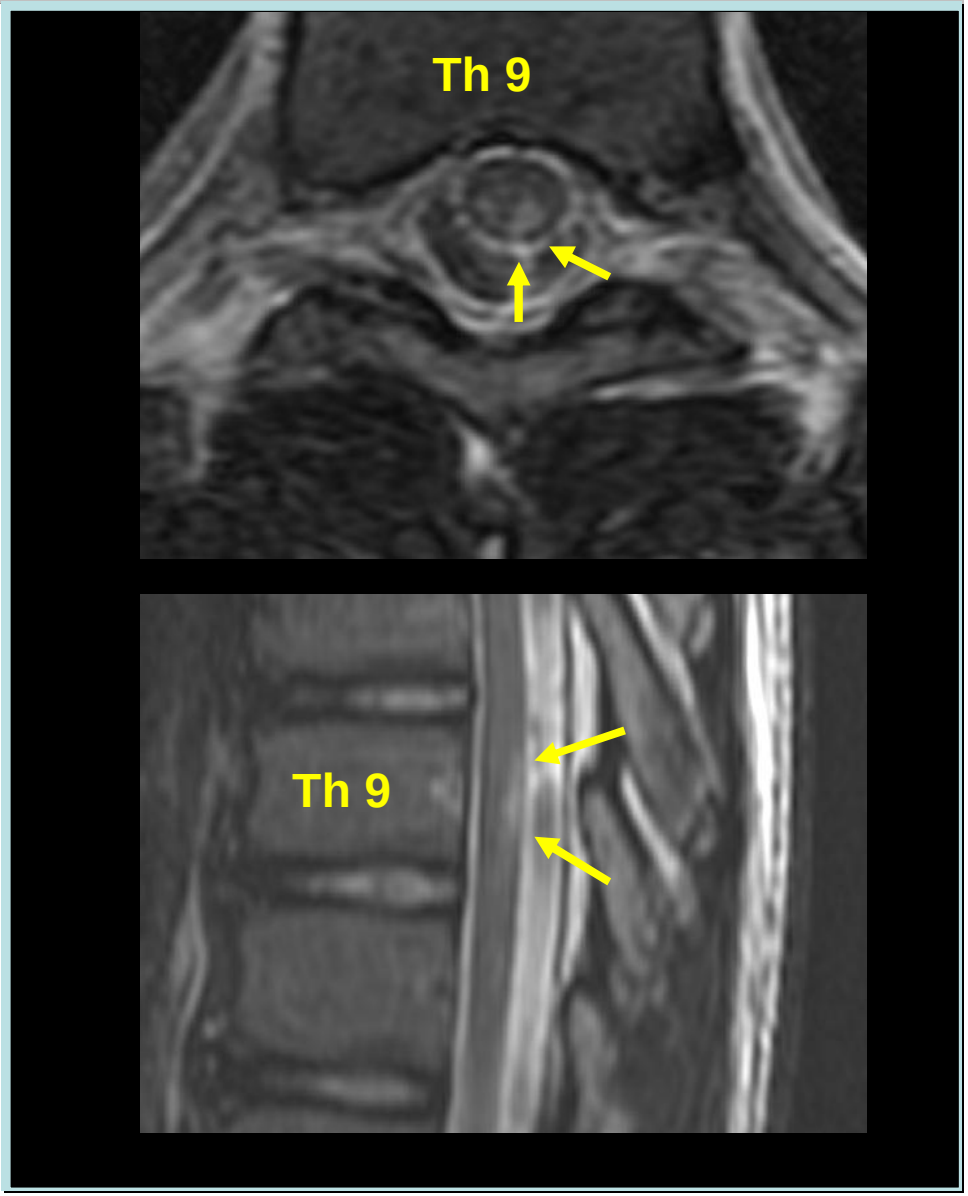
18 yr old student

- Abdominal pain during basket ball game
- Flaccid paraparesis of the legs (MRC 4/5)
- Loss of pain and temperature sensation below Th10
- CSF: normal
- MRI T2 hyperintense lesion at the D9 level



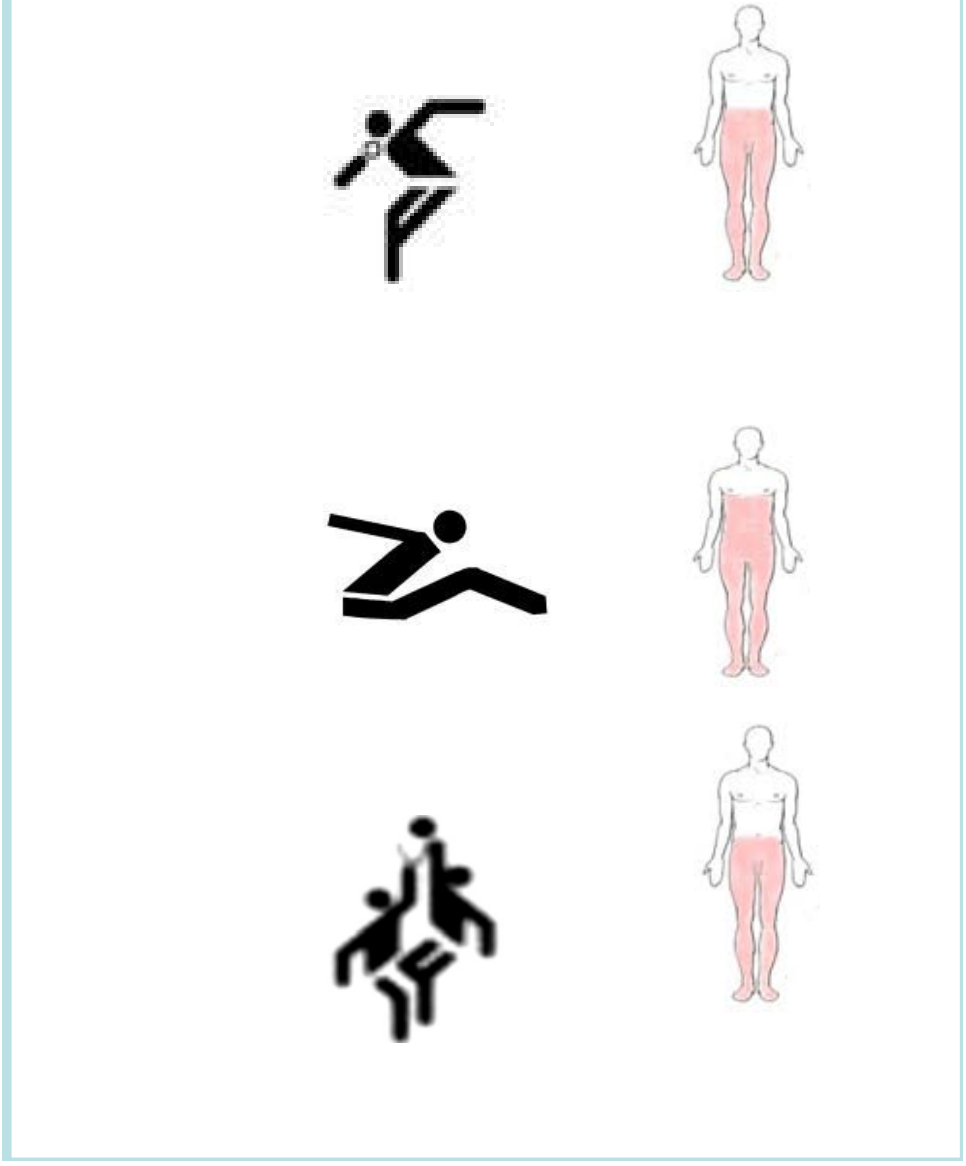
18 yr old student





Sports injury?

- Ascending paresis in young patients during sports
- Vascular clinical pattern
- CSF normal



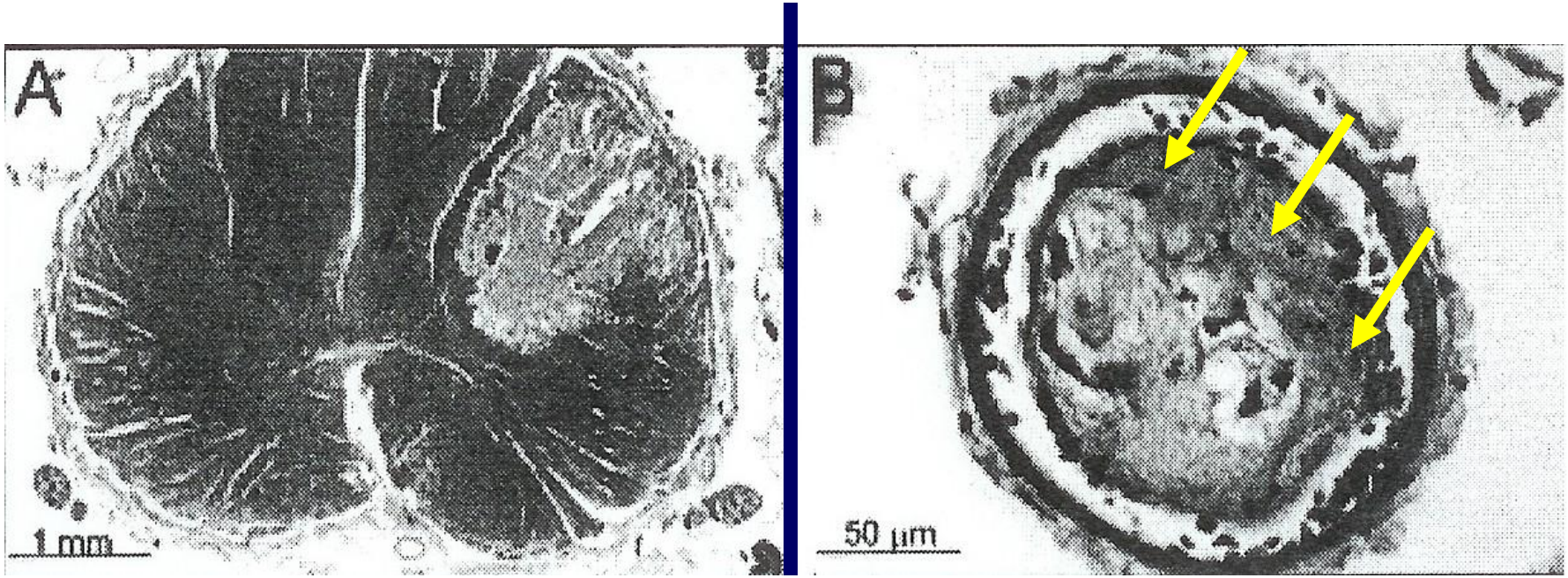
Fibrocartilaginous embolism (FCE)

PB 2013

Fibrocartilaginous embolism is a rare phenomenon that can cause spinal cord infarction. FCE originates from herniated intervertebral discs.
A temporal relationship to minor head or neck injury or heavy lifting is a clue to this etiology, but is not always present. A broad age range of patients (7 to 78 years) can be affected by this phenomenon
Most cases involve the cervical cord; some involve the thoracic cord. Local pain typically precedes neurologic symptoms by 15 minutes to 48 hours

MRI may show a collapsed intervertebral disc at the appropriate level.

Fibrocartilaginous embolism (FCE)



Freyaldenhoven et al. Neurology 2001

Fibrocartilaginous embolism (FCE)

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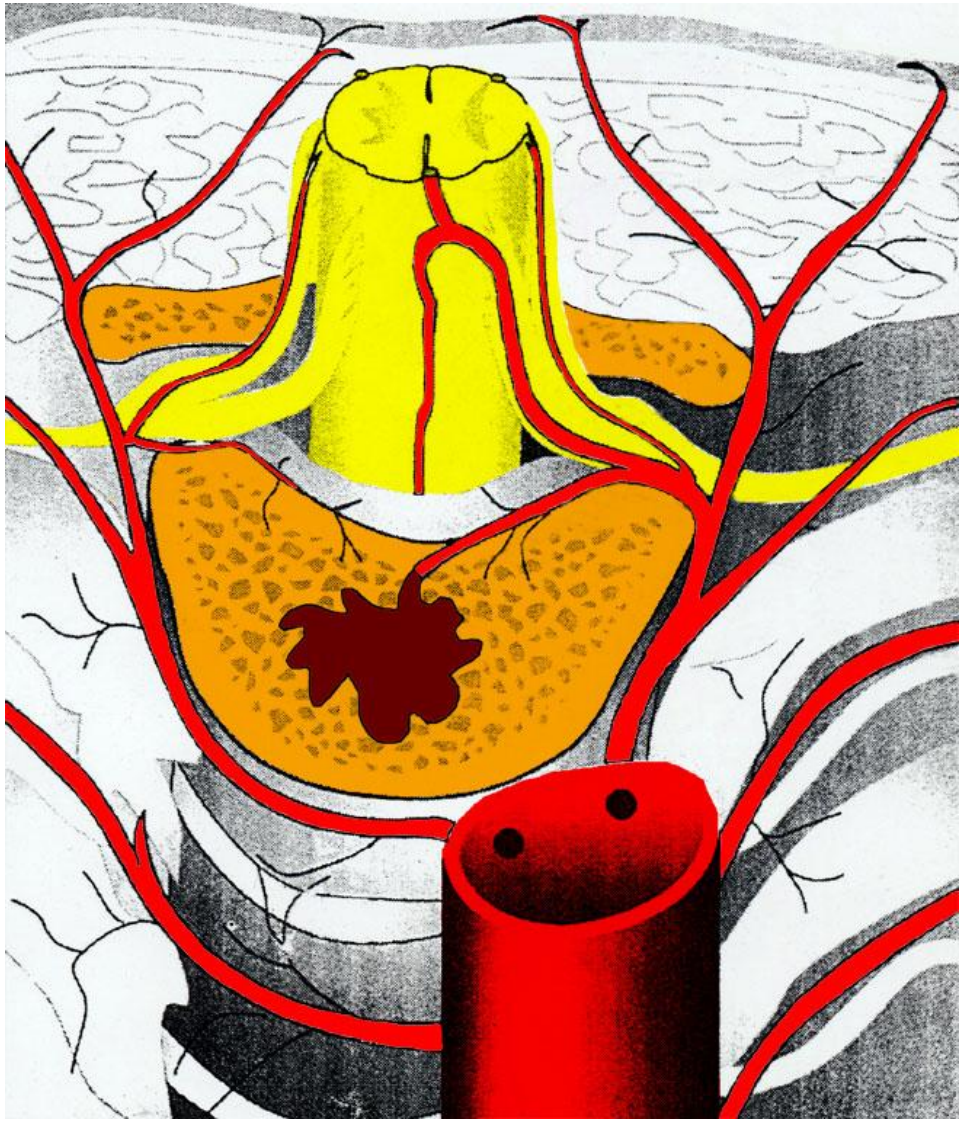

Cervical myelopathy

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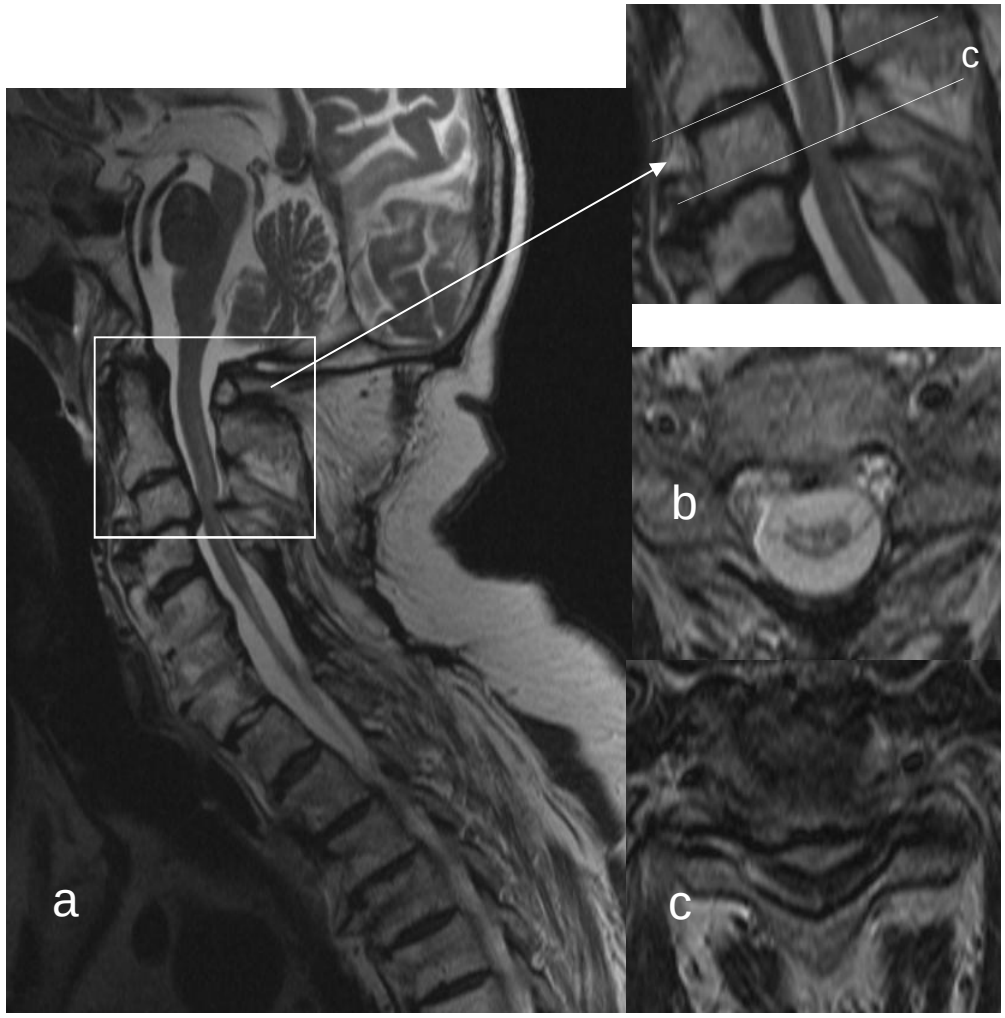
- Lower motor neuron signs (arms)
- Upper motor neuron signs (legs)
- Bladder disturbance
- pain
- MRI



Spinal dural fistula

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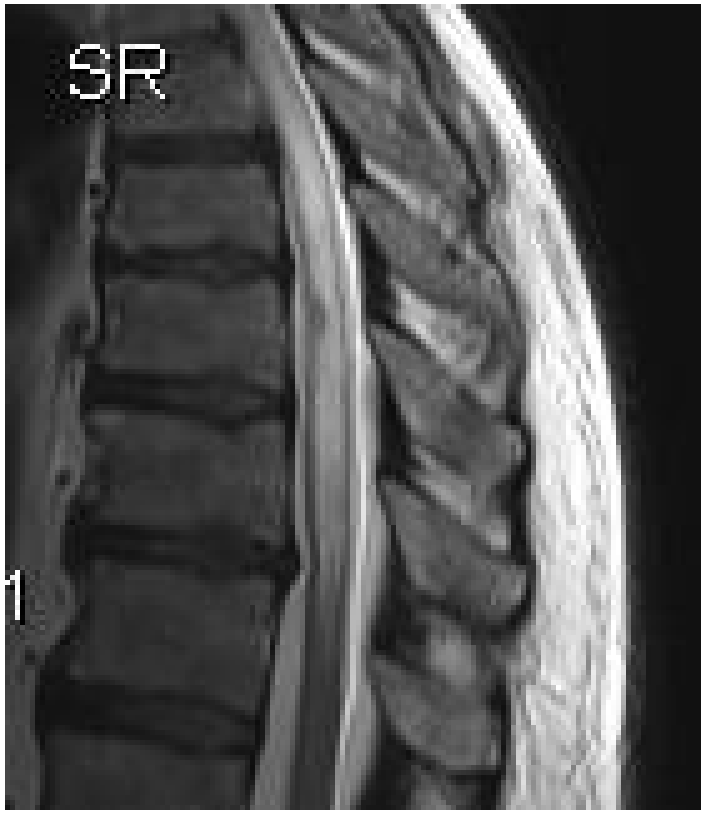

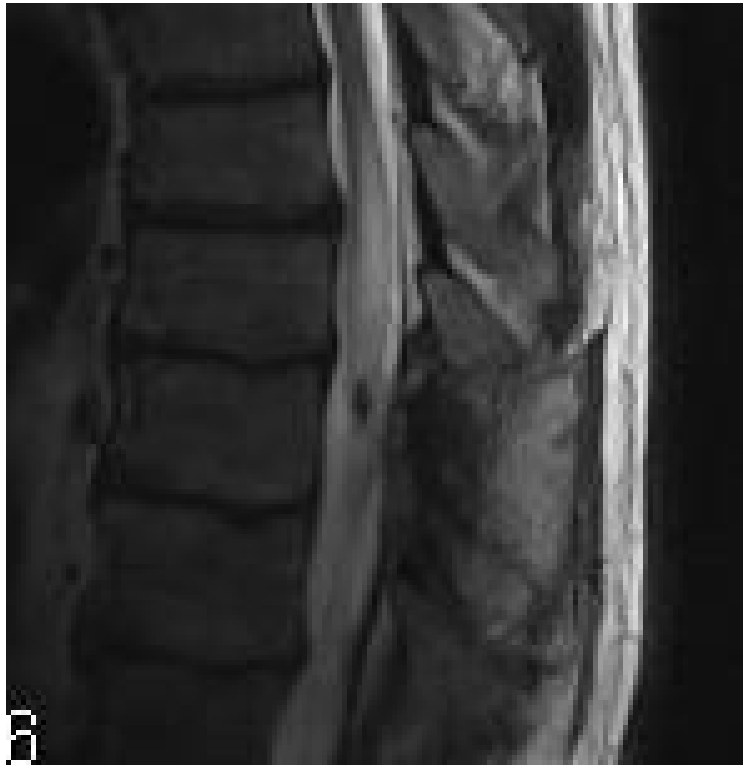

Acute flaccid paraparesis

- Spinal shock syndrome (vascular? trauma? myelitis? tumor? disc prolapse?)
- AIDP (CSF, F-waves, dmL, MEP)
- Low potassium (ECG changings!): pure motor syndrome
- Spinal angioma/fistula with congestive edema, hemorrhage

Comparison of findings in neuropathy, myopathy, and NMJ disorders

	Neuropathy	Myopathy	Neuromuscular junction
Typical distribution	Distal > proximal	Proximal > distal	Diffuse (oculomotor and bulbar early)
Reflexes	Decreased	Normal to decreased	Normal
Sensory involvement	Present	Absent	Absent

Reprinted with permission from: Asimos, AW. Weakness: A Systematic Approach to Acute, Non-traumatic, Neurologic and Neuromuscular Causes. Emergency Medicine Practice 2002; 4:1. Copyright © 2002 EB Practice, LLC. All rights reserved. http://www.ebmedicine.net.

Hints for the diagnosis of polyneuritis

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- Sock/glove pattern of sensory disturbances
- Areflexia
- Para- or tetraparesis
- No upgoing toes
- Risk of respiratory disorder
- Risk of autonomic failure

Guillain-Barré syndrome (GBS) - treatment

PB 2013

No steroids!

ICU

Plasmapheresis

or

immunglobulins (0,4 g/kg/day for 5 days)

Frequency of plasma exchange

PE (2x) more effective than none

PE (4x) more effective than PE (2x)

PE (6x) not better than PE (4x) n=556

French Cooperative Study Group, Ann Neurol, 1997

–6 more effective than 3

Raphael et al, J Neurol Neurosurg Psychiatry 2001

–4 more cost effective than 2

Esperou, Intensive Care Med, 2000

–Start before day 6 – as early as possible

Mortality in Guillain-Barré syndrome

van den Berg B et al, *Neurology 2013 80:1650-1654*

PB 2013

- Prospectively collected data were reviewed from a cohort of 527 patients with GBS previously included in 1 observational and 3 therapeutic studies. Risk factors were identified by comparing deceased and surviving patients with GBS.
- Death after GBS predominantly occurs in the elderly and severely affected patients, especially during the recovery phase.
- Future research is required to determine whether mortality of GBS can be reduced by intensified monitoring in patients with an increased risk profile.

Hospital admission and transfers in patients with fatal Guillain-Barré syndrome

The bars indicate the number of days at home between onset of weakness and hospital admission and of the various care facilities for each of the deceased patients.

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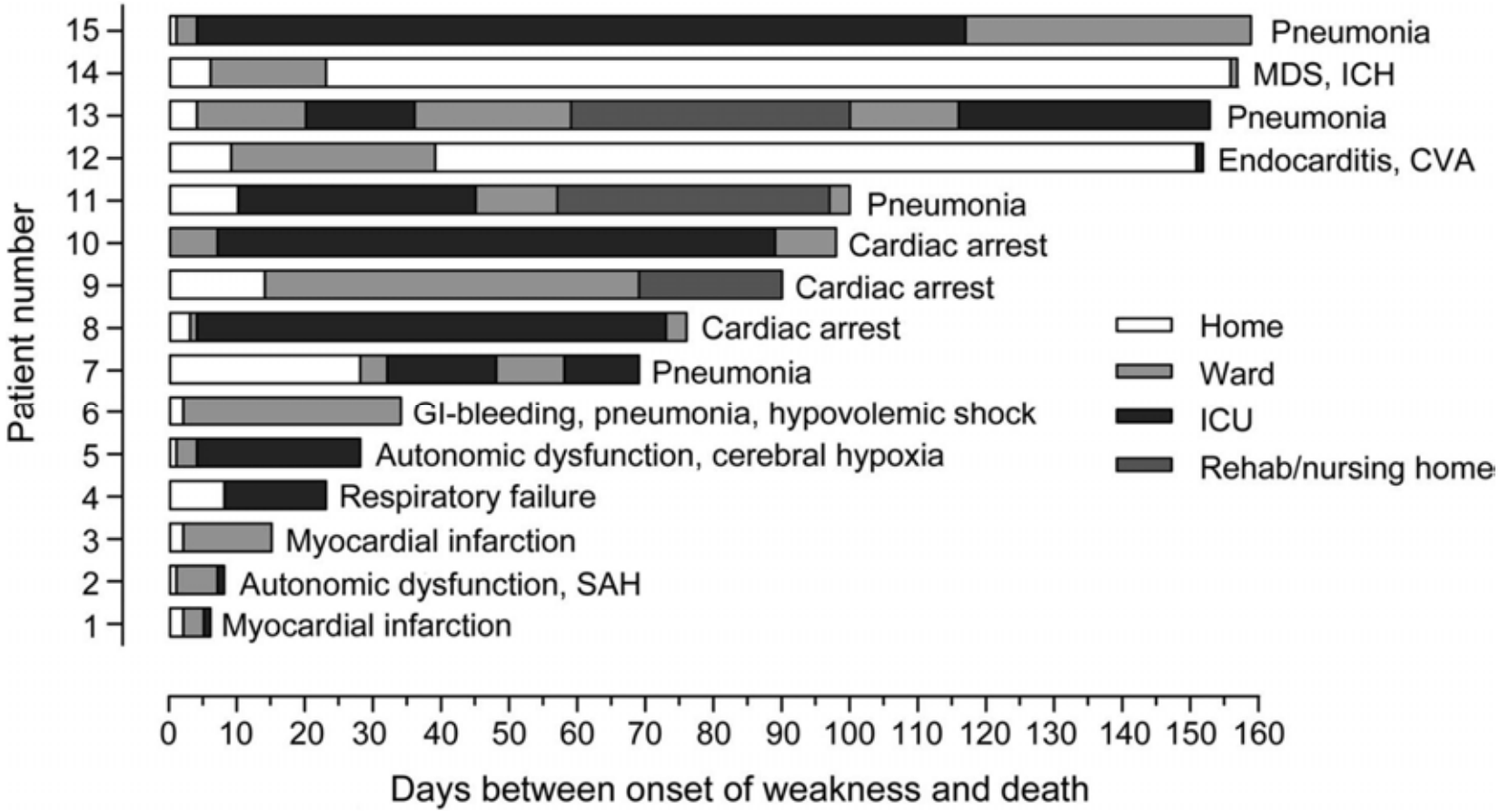

van den Berg B et al. Neurology 2013;80:1650-1654

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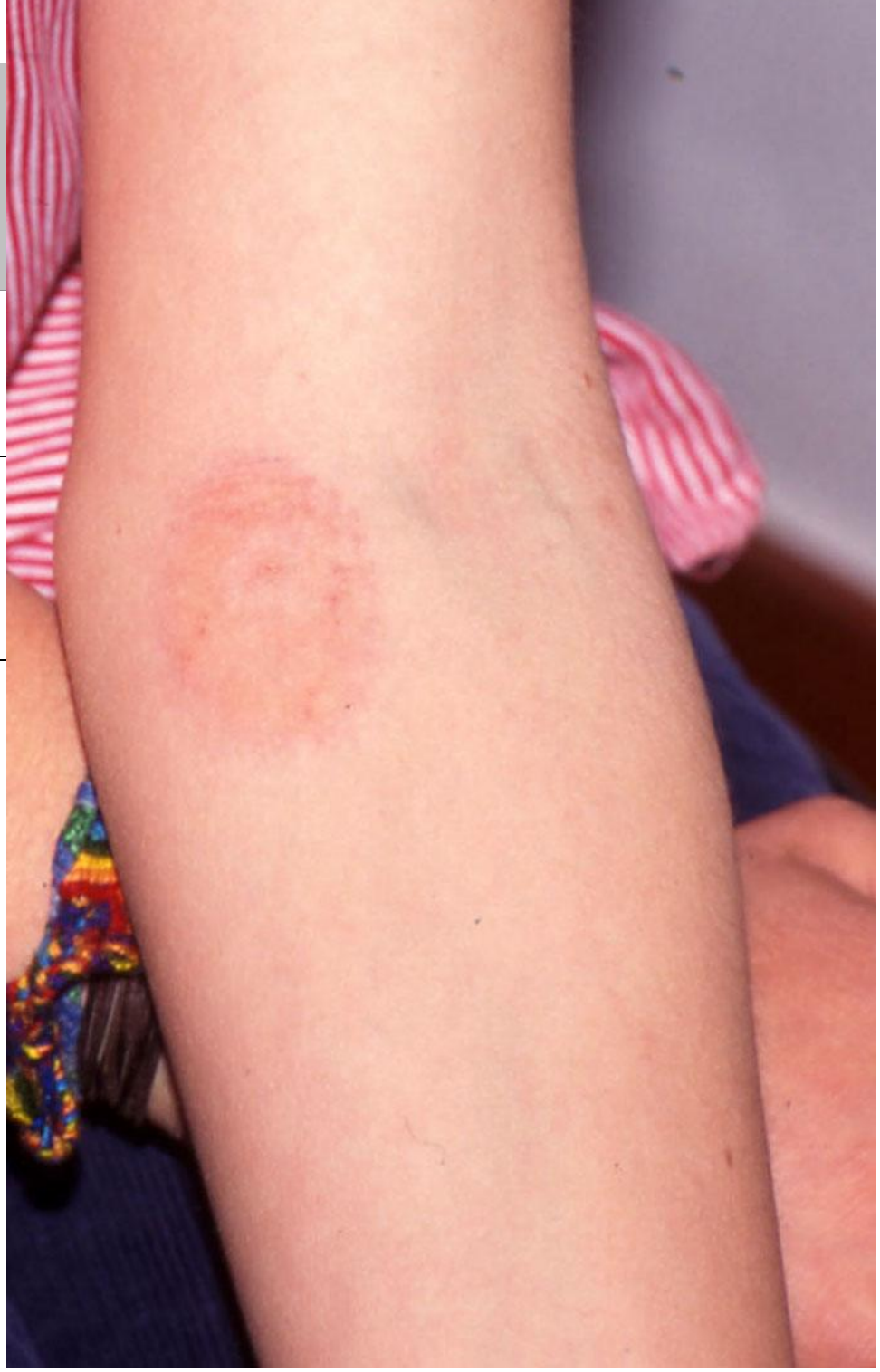

Erythema chronicum migrans

Spinal
Compressive myelopathy
Transverse myelitis
Anterior spinal artery syndrome
Poliomyelitis
Other infectious causes of acute myelitis (eg, West Nile virus, coxsackieviruses, echoviruses)
Peripheral nervous system
Toxic neuropathy
Drugs
Toxins
Critical care neuropathy
Diphtheria
Tick paralysis
Porphyria
Lyme disease
Vasculitis
Neuromuscular junction
Botulism
Myasthenia gravis
Neuromuscular blocking agents
Muscle disease
Acute viral myositis
Acute inflammatory myopathies
Metabolic myopathies (eg, hypokalemic, hyperkalemic)
Periodic paralysis

Data from:

1. *Evans OB. Guillain-Barré syndrome in children. Pediatr Rev 1986; 8:69.*
2. *Jones HR. Childhood Guillain-Barré syndrome: clinical presentation, diagnosis, and therapy. J Child Neurol 1996; 11:4.*
3. *Yuki N, Hartung HP. Guillain-Barré syndrome. N Engl J Med 2012; 366:2294.*

Hints for an underlying neuromuscular disease

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- No sensory signs
- No pyramidal tract signs
- Fluctuating pareses
- Risk of respiratory disorder
- Risk of autonomic failure

Treatment of myasthenic crisis

FVC: forced vital capacity; IVIG: intravenous immune globulin; NIF: negative inspiratory force.

Rapid therapies for myasthenia gravis

	Plasmapheresis	Intravenous immune globulin
Usual adult dose	Five exchange treatments of 3 to 5 liters over 10 to 14 days	400 mg/kg daily for five days
Onset of effect	1 to 7 days	1 to 2 weeks
Maximal effect	1 to 3 weeks	1 to 3 weeks
Adverse effects	Line infection, hypotension, thromboembolism	Headache, fluid overload, renal failure (rare)

Foodborne botulism

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Symptoms begin within 12 to 36 hours after ingestion of the toxin
(range from several hours to one week)

Prodromal symptoms: nausea, vomiting, abdominal pain, diarrhea, dry mouth with sore throat

First symptoms involve the cranial nerves: blurred vision and diplopia (secondary to fixed pupillary dilation and palsies of cranial nerves III, IV, and VI), nystagmus, ptosis, dysphagia, dysarthria, facial weakness
Then descending muscle weakness progressing to the trunk and upper extremities, followed by the lower extremities
Urinary retention and constipation
Respiratory difficulties (eg, dyspnea) requiring intubation and mechanical ventilation
Occasionally paresthesias

CSF normal

Hints for muscle diseases :

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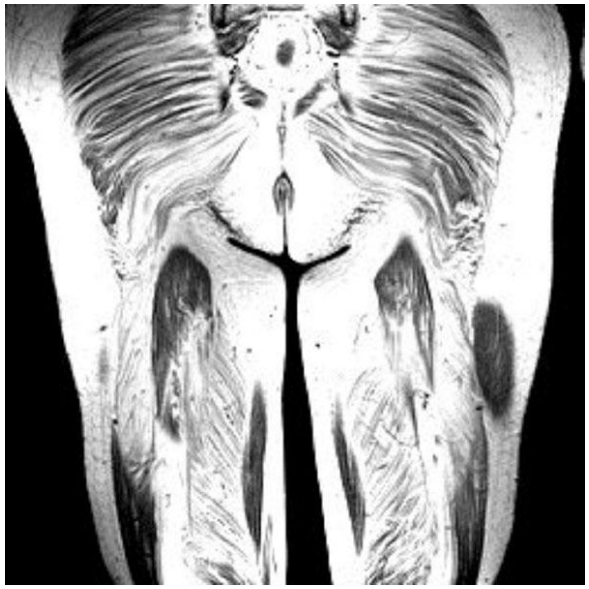
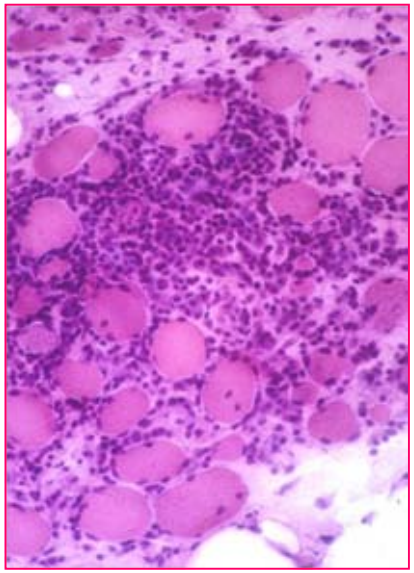
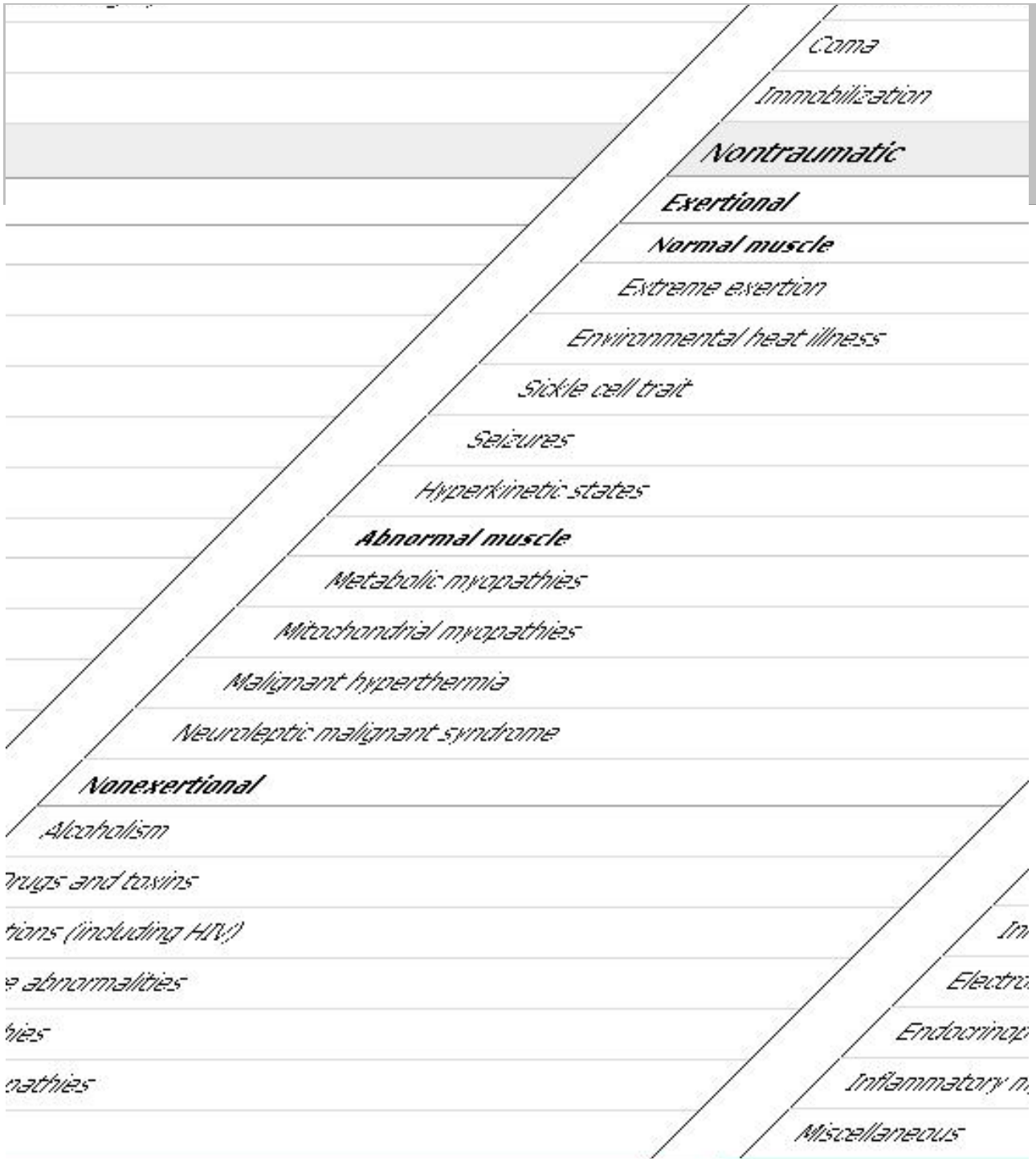


- No sensory signs
- No pyramidal tract signs
- Muscle pain, swelling
- Dark urine



Hints for metabolic causes

- repeated episodes
- provocation by feasting, heavy meals, coke (children!), physical stress, cold temperatures
- abdominal pain
- dark urine
- family anamnesis


Coma
Immobilization
Nontraumatic
Exertional
Normal muscle
Extreme exertion
Environmental heat illness
Sickle cell trait
Seizures
Hyperkinetic states
Abnormal muscle
Metabolic myopathies
Mitochondrial myopathies
Malignant hyperthermia
Neuroleptic malignant syndrome
Nonexertional
Alcoholism
rugs and toxins
tions (including HIV)
e abnormalities
hies
nathies
Electro
Endocrino
Inflammatory
Miscellaneous


Metabolic myopathies associated with
abdomyolysis
rders of glycogenolysis
horylase deficiency (McArdle's Disease)
kinase deficiency
colysis
eficiency
eficiency
ency
Disorders of lipid metabolism
Carnitine palmitoyltransferase deficiency
Carnitine deficiency
Short-chain acyl-CoA dehydrogenase deficiency
Long-chain acyl-CoA dehydrogenase deficiency
Disorders of purine metabolism
Myoadenylate deaminase deficiency
Others
Calcium adenosine triphosphatase deficiency

Major symptoms and signs of hypothyroidism

Mechanism	Symptoms	Signs
Slowing of metabolic processes	Fatigue and weakness Cold intolerance Dyspnea on exertion Weight gain Cognitive dysfunction Mental retardation (infantile onset) Constipation Growth failure	Slow movement and slow speech Delayed relaxation of tendon reflexes Bradycardia Carotenemia
Accumulation of matrix substances	Dry skin Hoarseness Edema	Coarse skin Puffy facies and loss of eyebrows Periorbital edema Enlargement of the tongue
Other	Decreased hearing Myalgia and paresthesia Depression Menorrhagia Arthralgia Pubertal delay	Diastolic hypertension Pleural and pericardial effusions Ascites Galactorrhea

Hyperkalemia

Ascending muscle weakness that begins with the legs and progresses to the trunk and arms, progressing to flaccid paralysis, mimicking Guillain-Barré syndrome. Sphincter tone and cranial nerve function are intact, no respiratory muscle weakness

In addition to acquired hyperkalemia, there is a genetic disorder hyperkalemic periodic paralysis that is caused by autosomal dominant mutations in the skeletal muscle cell sodium channel.
Patients with this disorder develop myopathic weakness during hyperkalemia induced by increased potassium intake or rest after heavy exercise.

Hypokalemia

Significant muscle weakness can occur at serum potassium concentrations below 2.5 meq/L or at higher values with hypokalemia of acute onset, as occurs in hypokalemic or thyrotoxic periodic paralysis

The pattern of weakness in hypokalemia is similar to that associated with hyperkalemia. Weakness usually begins in the lower extremities, progresses to the trunk and upper extremities, and can worsen to the point of paralysis.

Hypokalemia

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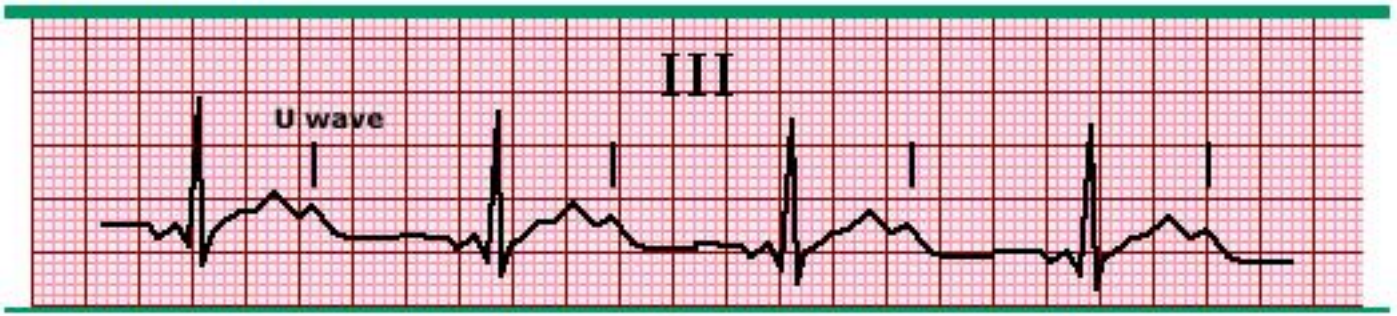
In addition to causing muscle weakness, severe potassium depletion (serum potassium less than 2.5 meq/L) can lead to muscle cramps, rhabdomyolysis, and myoglobinuria

Hypokalemia



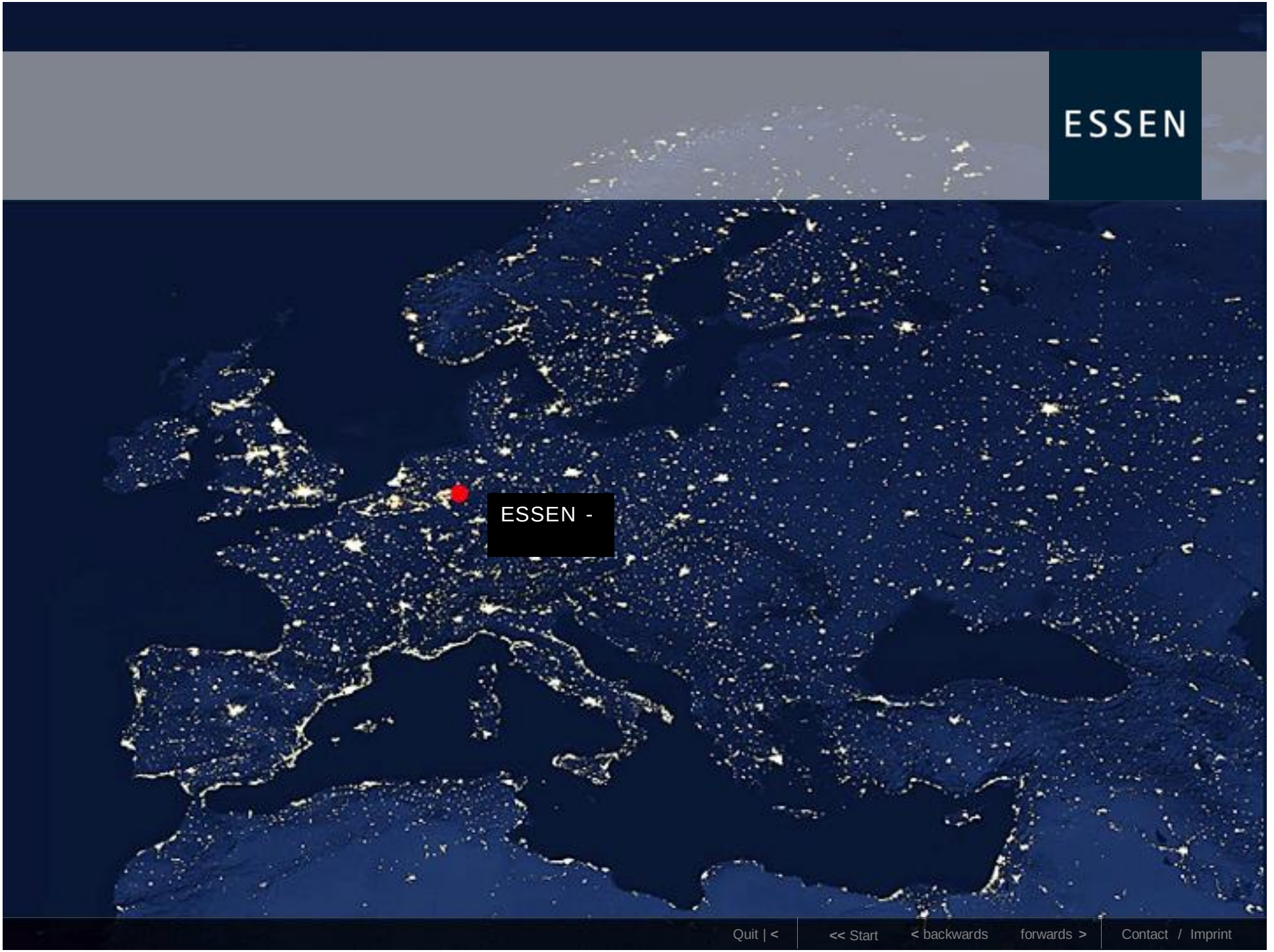
An increase in the amplitude of U waves, which occur at the end of the T wave, are characteristic of hypokalemia.

Therapeutic strategies

- Myopathies: eliminate causative agents, dialysis
- Myasthenic crisis: pyridostigmine, plasmapheresis or IVIG
- AIDP: plasmapheresis or IVIG
- Spinal disorders: emergency surgery, dexamethasone
- Brain stem: revascularization, steroids, drainage

Take home messages:

- Anamnesis and neurologic examination are the basics for decisions
- Distribution of pareses, sensory disturbances and pyramidal tract signs are important hints
- Reflexes are often not very helpful
- Look for autonomic disturbances
- Look for accompanying symptoms (cranial nerves, pain)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