

Spinal Cord Injury

History of Presenting Illness

SEVERE PAIN

SWELLING + BRUISING AT BACK OR NECK

DIFFICULTY MOVING LEGS AND/OR ARMS

Differential Diagnoses

The limbs you are attempting to move may simply be **FRACTURED**.

THE SWELLING and BRUISING may be the result of traumatic soft tissue injury

Your **SEVERE PAIN** is consistent with your history of violent impact and could be related to the **OTHER INJURIES**

OTHERWISE ALWAYS ASSUME SPINAL CORD INJURY IN ANY MVA

Pertinent Findings on History (Hx)

- **WHAT HAPPENED** is a fair question when addressing an MVA victim who cant move
- Drug + Alcohol history is essential
(not just medicolegally, as the pt. may get unpleasant withdrawal symptoms)
- Eyewitness accounts of the impact: exactly how they fell / hit
- Allergies, medications;

Findings on Examination (Ex)

- Glasgow Coma Scale →
- Temperature, Blood Pressure; Pulse Rate:

Hypothermia (but skin = warm and dry)

hypotension **without** compensatory tachycardia?

Urine output preserved? = Spinal Shock!

Induced BY INJURY ABOVE T6

Pulse Oximetry to make sure the coma your patient is about to slip into is not due to anoxia

- **Is Trachea mid-line?**
- **Percuss** to check for pneumothorax

Abdominal examination: Looking for palpable, enlarged bladder (paralysis of detrusor muscle)

Neurological Examination

TONE + REFLEXES:

Flaccid = Lower Motor Neuron Lesion

Rigid = Upper Motor Neuron Lesion

MOVEMENT: any at all?...

Check for flickers of movement: better prognosis if present

SENSATION:

Map out area of loss to determine the level of injury

Follow dermatome map, its surer than the myotome tests

Eye Opening	E
spontaneous	4
to speech	3
to pain	2
no response	1
Best Motor Response	M
To Verbal Command:	
obeys	6
To Painful Stimulus:	
localizes pain	5
flexion-withdrawal	4
flexion-abnormal	3
extension	2
no response	1
Best Verbal Response	V
oriented and converses	5
disoriented and converses	4
inappropriate words	3
incomprehensible sounds	2
no response	1

E + M + V = 3 to 15

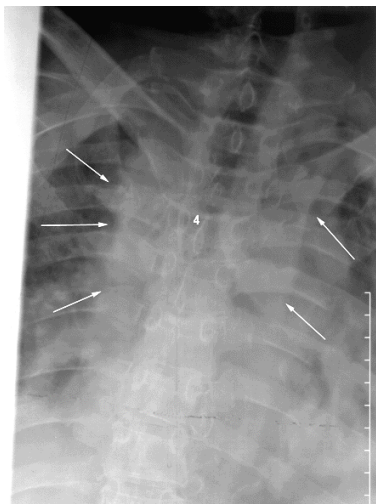
- 90% less than or equal to 8 are in coma
- Greater than or equal to 9 not in coma
- **8 is the critical score**
- Less than or equal to 8 at 6 hours - 50% die
- 9-11 = moderate severity
- Greater than or equal to 12 = minor injury

Coma is defined as:

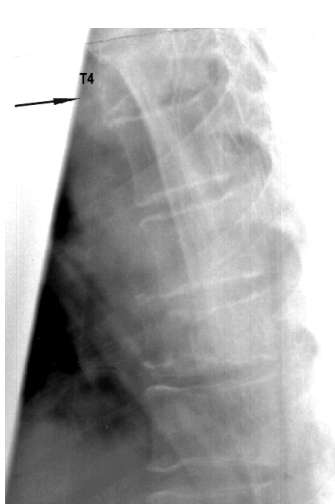
- (1) not opening eyes,
- (2) not obeying commands, and
- (3) not uttering understandable words.

Tests and Investigations : TRAUMA SERIES :

Chest X-ray: Cervical and Thoracic



ABOVE: Thoracic X-ray of a T4 fracture surrounded by haematoma (arrows)
:BUT the fracture itself is hard to see



ABOVE: Lateral Thoracic X-ray of a T4 fracture (arrow): see the collapsed T3 impacted into the T4 vertebra

- To see the fracture site; important for **Anteroposterior, lateral, and special (odontoid and neuroforaminal)**
 - The fracture will look like a **collapsed or shortened vertebral body**
 - The fracture site will be surrounded by an **area of opaque-ish (blood-filled) haemorrhage**
- patient does not have to move much; just wheel them into the X-ray room (there's usually one in every Emergency Room)
You are also looking for **Pneumothorax, haemothorax, pericardial effusions, rib fractures etc.**

Radiography can

~miss~

facet fractures!

It's a shit modality anyway, don't rely on it for diagnosis

CROSS-SECTIONAL STUDIES:

Much more information than the plain radiograph



← CT:

needed to determine extent of injury:

- **spinal canal compression by pieces of fractured vertebra,**
- **oedema-**

← opposite is a CT scan of a T4 fracture and slight spinal canal narrowing

MRI: even better,

but rarely available;

thus more useful for **checking progress of recovery**, eg.

- is a syrinx developing?
- Is there appropriate bone healing?

Soft tissue definition is also good enough to look for causes of

NON-TRAUMATIC SC INJURY, eg. spinal infarct, vascular malformation, etc

ECG: !! important; after acute SC injury you often get **"Autonomic Dysreflexia"**

= hypotension, bradycardia, and, rarely, cardiac arrest

THUS:

- evaluate bradycardia
- look for arrhythmias
- (eg, most commonly supraventricular tachyarrhythmias)
- look for ST-T wave abnormalities indicative of ischemia

SPIROMETRY is needed to assess respiratory function, which may be impaired following a C-spine fracture (C3, 4, 5 keep the diaphragm alive ... via the phrenic nerve)

URINE ANALYSIS

first, visually: is the patient's urine full of frank blood?

They may be bleeding in the pelvis somewhere

FULL BLOOD COUNT:

Mainly interested in **Haemoglobin**; i.e. how much blood has been lost

BLOOD ALCOHOL

Mainly medicolegal reasons; the police may need to know if alcohol was involved as a causative factor in the MVA.

Plus if the patient is an alcoholic it will be harder to deal with them during their long recovery, as they begin to withdraw from their addiction and become agitated and uncooperative

How is this diagnosis made ? ...Immediately!

With any major trauma, especially to the head or neck and with whiplash injury, spinal injury should be immediately suspected.

Management

Aims of early management:

- **minimise secondary damage** to the spinal cord
- **prevent complications** from SC dysfunction

1) First Aid Management must include immobilisation of the unstable spine

whilst paying attention to the **airway, breathing and any associated injuries** that may not be evident due to loss of pain sensation below the spinal cord injury level.

2) Admission (immediate) Management

1. **Replace fluids and blood → !!! ONLY !!! IF THE PATIENT REQUIRES IT**

2. **Artificial ventilation** if the pt cannot breathe

3. **Nasogastric intubation to aspirate stomach contents** (!! Paralytic ileus results from loss of sympathetic innervation, and foul things creep up the GIT into the stomach; thus → need to aspirate)

4. **Urethral catheter to drain the bladder** (normally it will distend painlessly until 150% full, and then empty spontaneously until 25% full)

5. **Give histamine (H₂) receptor antagonists**

to reduce stress ulceration in the stomach

6. **Give subcutaneous heparin** } ← Trauma, paralysis and bed rest are risk factors for

7. **Use TED stockings** }

thromboembolism (10% of SCIs develop PE in hospital)

8. **Regular turning (3-hrly) and sheep skin bed cover** to prevent pressure sores

9. **Position the paralysed limbs** to prevent muscle shortening and contractures

3) Short Term Management

1. **empty bowel with enemas and manual evacuation**

2. **feed through nasogastric tube (nil by mouth)**

3. **SURGICAL MANAGEMENT (→ FIXATION ←)**

of spinal fracture and spinal canal narrowing

4. **post-operative analgesia (Patient-Controlled Analgesia, PCA)**

4) Long Term Management

1. **Social worker** to discuss legal assistance and counselling needs

2. **Physiotherapy** to prevent contractures

3. **Occupational therapy** to discuss job and accommodation

4. **Clinical psychologist / psychiatrist** to counsel for grief, etc.

5. **Baclofen** to manage muscle spasms and spasticity

6. **Wheel chair or calliper training + devices to assist driving**

5) Follow-up

REGULAR URINALYSIS → watch for hydronephrosis / pyelonephritis (from bladder paralysis)

REGULAR PHYSIOTHERAPY → watch for contracture + spasticity

REGULAR PHONE FOLLOW-UP → monitor emotional well-being (high suicide risk)

PROGNOSIS

Usually **lifespan is greatly reduced**

*Prognosis is **BETTER** if:*

- *You have some flickers of movement in the lower limbs after the accident*
- *You are younger than 50 (91% of you will regain ambulation, instead of only 41% of the oldies)*

***MOST IMPORTANT PREDICTOR OF CLINICAL OUTCOME:
RETENTION OF SACRAL SENSATION***

~Especially pinprick~

for 72 hours to 1 week after injury

= BEST PROGNOSIS EVER !!

GENERALLY: most people regain one level of motor function within 6 months.

Improvement after that is rare but not unheard of

MORTALITY:

SCI patients die of

- **Pneumonia**
 - **Pulmonary emboli**
 - **Septicaemia**
 - **Renal Failure**
- (in order of frequency)

!! DEPRESSION KILLS SPINAL CORD INJURY VICTIMS !!

*The **suicide rate** among individuals with SCI is nearly **5 times higher than in the general population***

(lower for men than for women)

BUT!! You must AVOID TRICYCLIC ANTIDEPRESSANTS

Because their anticholinergic effects will exacerbate bladder problems

Epidemiology

Yearly incidence in Australia = 14.5 cases per million

60% are in cervical region; 60% are INCOMPLETE (not fully severed cord)

50% are from MVAs, 20% from contact sports

Basic Sciences: PHYSIOLOGY OF SPINAL SHOCK

a state in which there is loss of sensory and motor function below the level of spinal cord injury

marked by absence of somatic and autonomic reflexes (flaccidity) below the level of cord injury.

damage to the spinal cord results in **disruption of tonic descending excitatory influences from the brain.**

Removal of this excitatory input is then believed to produce an overall depression of reflex activity.

However, this explanation does not account for the observations that the net effect of descending spinal pathways is inhibitory and that **experimental spinal cord transection leads to an increase in reflex activity.**

**Hypothermia (but skin = warm and dry)
hypotension without compensatory tachycardia?**

Urine output preserved?

= Spinal Shock!

Induced BY INJURY ABOVE T6

Changes associated with loss of reflex activity:

Loss of somatic reflexes leads to loss of deep tendon reflexes and a flaccid paralysis of the affected limbs (lower limbs in paraplegics and upper and lower limbs in tetraplegics).

Loss of reflex activity in viscera so that the bladder and bowel lose their tone and become flaccid.

This may result in complete absence of bowel activity (paralytic ileus) and bladder tone (bladder distension).

Loss of autonomic reflexes leads to major disturbance within the cardiovascular system.

If the spinal cord injury is above the T6 level there is a substantial loss of sympathetic vascular tone.

loss of sympathetic tone → decrease in vascular resistance, thus pooling of blood in the extremities. This leads to **a dramatic fall in blood pressure (80-100 systolic/50-60 diastolic mm Hg)** even though there has been no change in the circulating blood volume. Despite the dramatic fall in blood pressure **the loss of reflex activity means that there is no compensatory increase in heart rate** which may be still around 60 beats/minute. **The skin also remains warm and dry.** These effects are in contrast with circulatory shock as a result of blood loss (hypovolaemic shock).

THUS: !! DO NOT TREAT SPINAL SHOCK WITH FLUID REPLACEMENT !!

MAKE SURE ITS SPINAL, NOT HYPOVOLEMIC

the fall in blood pressure is due to spinal shock it can often be left untreated.

If severe, treat with inotropic agents such as dopamine, thus increase the pumping action of the heart

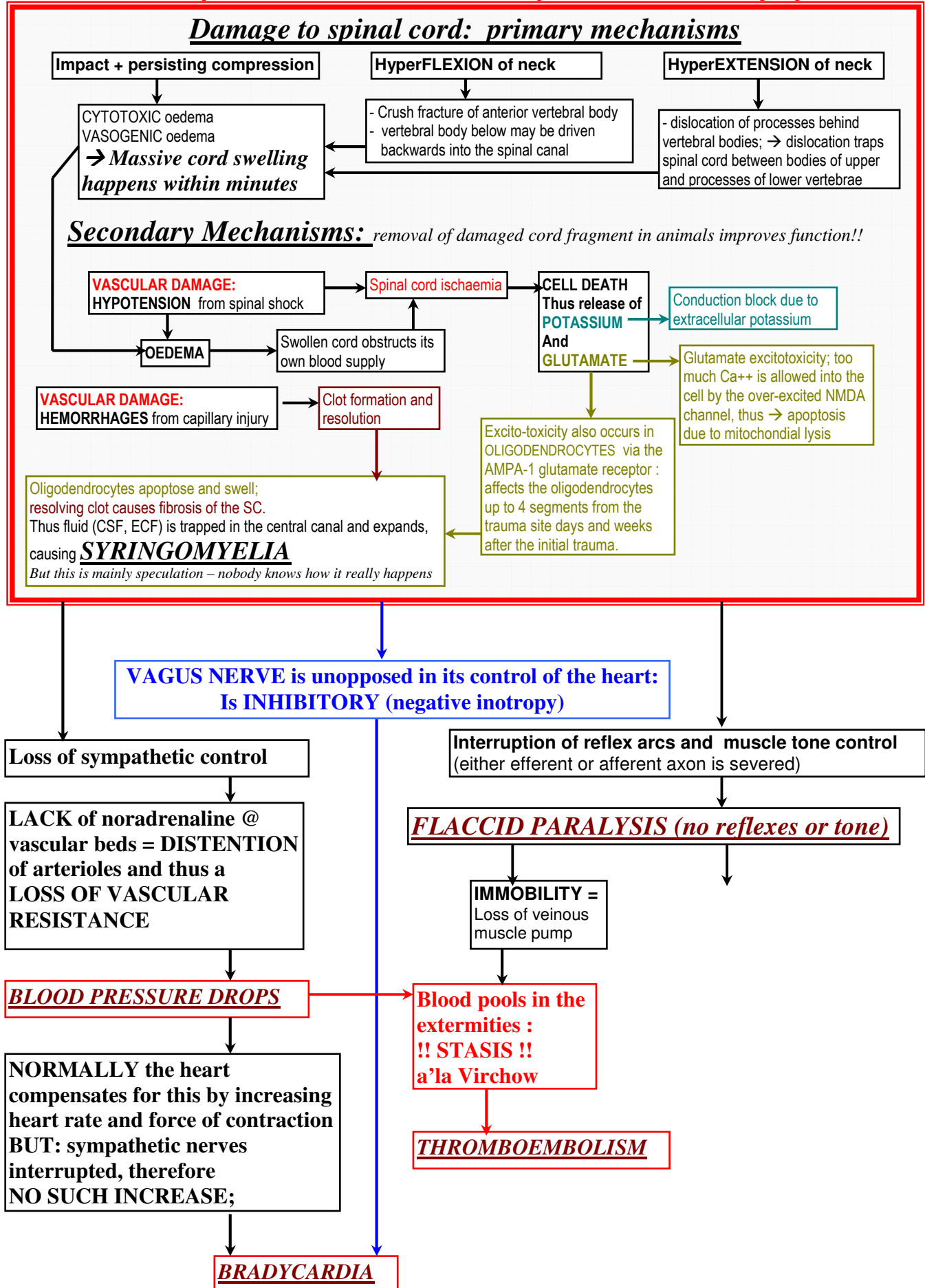
spinal shock can last **from several hours to around 6 weeks**

THEN: reflexes below the level of spinal cord injury become hyperresponsive.

This can lead to **increased tone and hyperreflexia of the limbs and bladder** if they are below the lesion.

If the injury is above T6, a condition called **autonomic dysreflexia** can occur in which nociceptive input below the injury (eg a distended bladder) will produce dramatic and dangerous rises in blood pressure.

Mechanism of Spinal Shock and some Complications of SC injury



Relevant anatomy: segmental organisation of the spinal cord

The spinal cord is:

- **45 cm** in length
- from **C1 to bottom of L1**

- **31 segments:**

- **8 cervical,**
- **12 thoracic**
- **5 lumbar**
- **5 sacral**
- **1 coccygeal.**

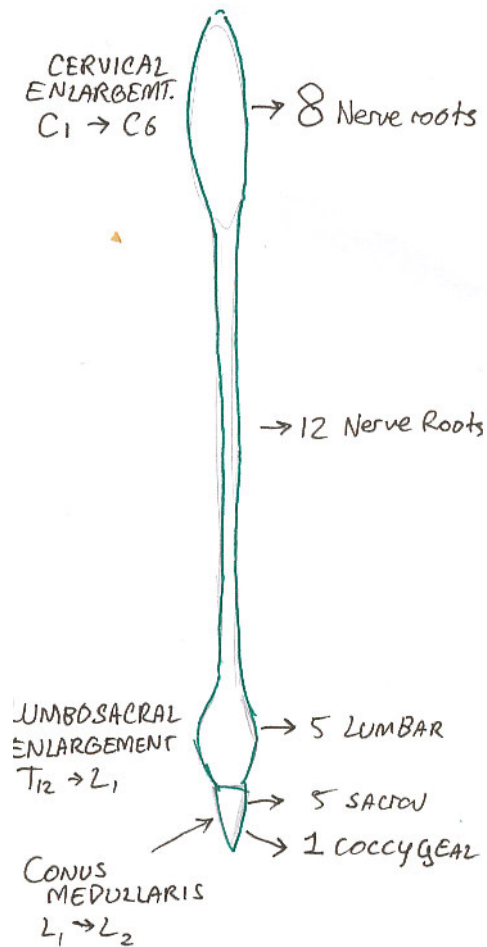
sacral spinal segments
lie opposite lower thoracic vertebrae

- surrounded by **meninges**
pia, arachnoid, dura.

The pia mater closely invests the spinal cord and its nerve roots
subarachnoid space contains cerebrospinal fluid

@ **INF END:** conus medullaris
cauda equina

A fibrous band of pia mater, the **filum terminale**, anchors the spinal cord to the sacrum and coccyx below.



DORSAL HORN:

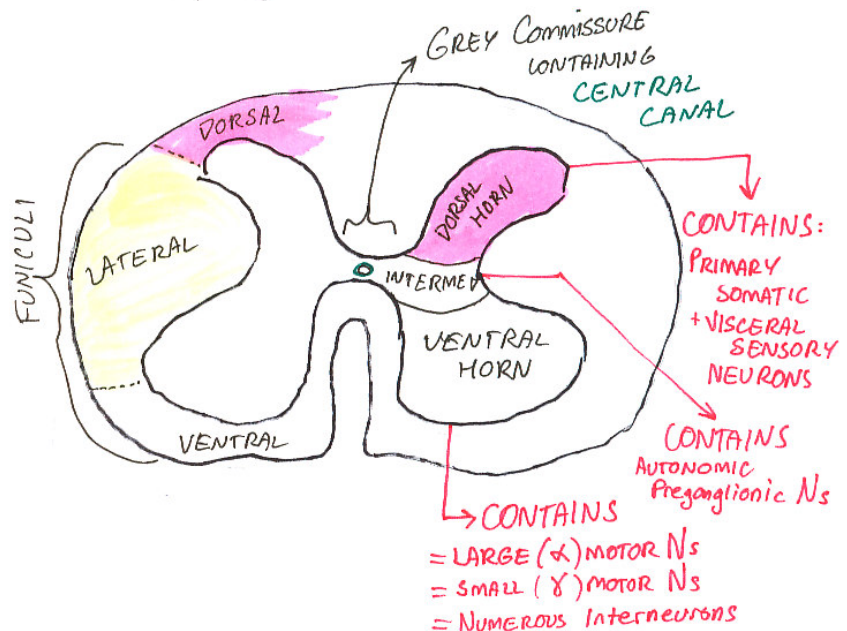
Visceral + somatic
afferent (sensory) neuro

INTERMEDIATE MATTER:

Autonomic NS neurons
(pre-ganglionic)

VENTRAL HORN:

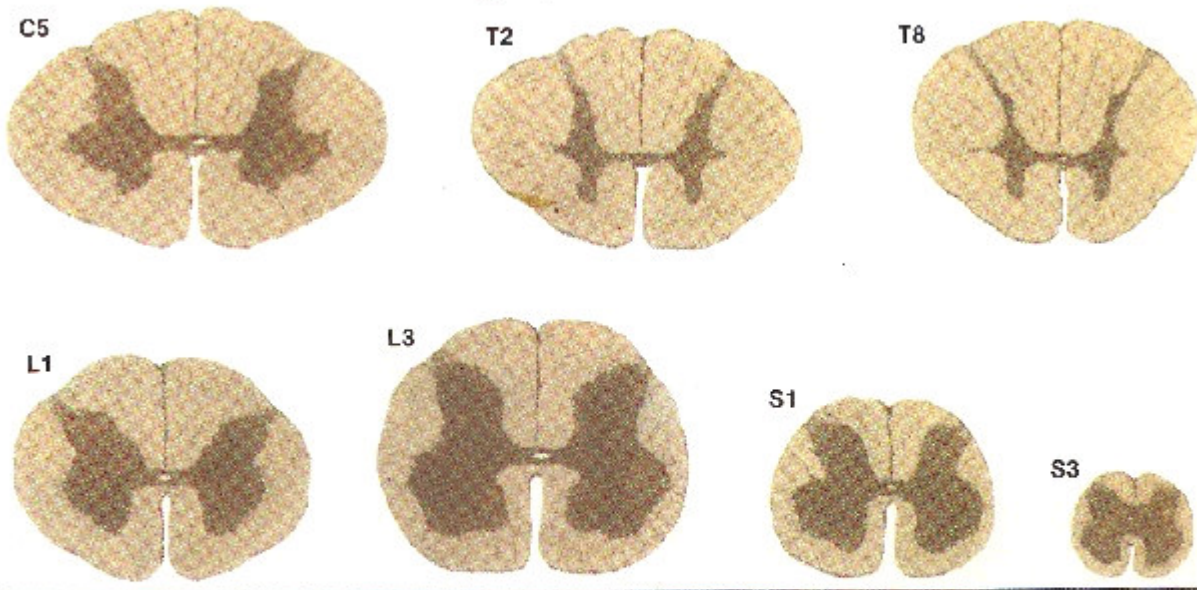
Alpha (large)
and **Gamma (small)**
motor neurons



Laminae I-V are found in the **dorsal horn**,
laminae VIII and IX in the **ventral horn**,
laminae VI, VII and X in the **intermediate grey**.

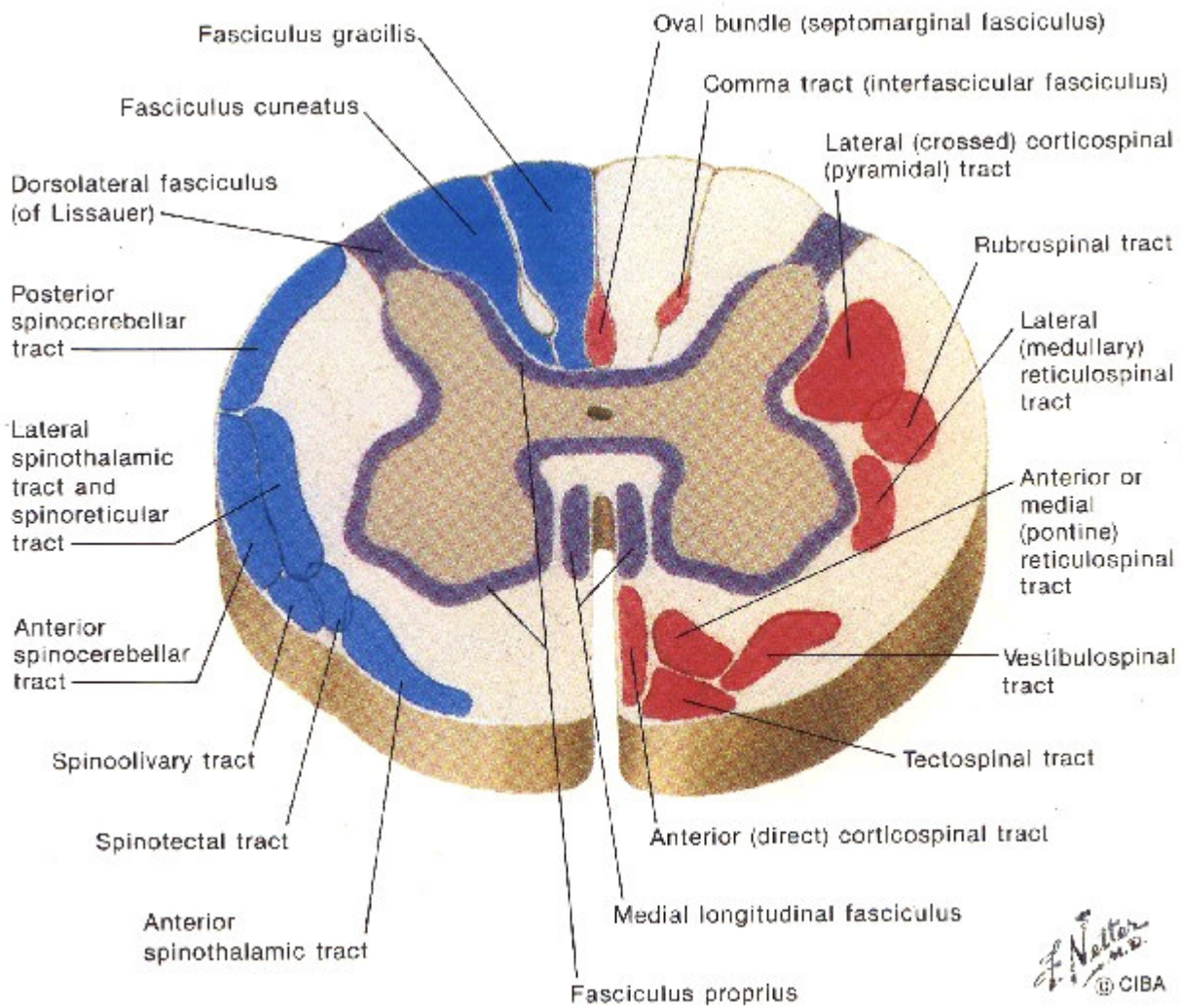
Neurons within specific laminae are targets of both primary afferents and descending supraspinal inputs, and contain the cells of origin of distinct ascending pathways

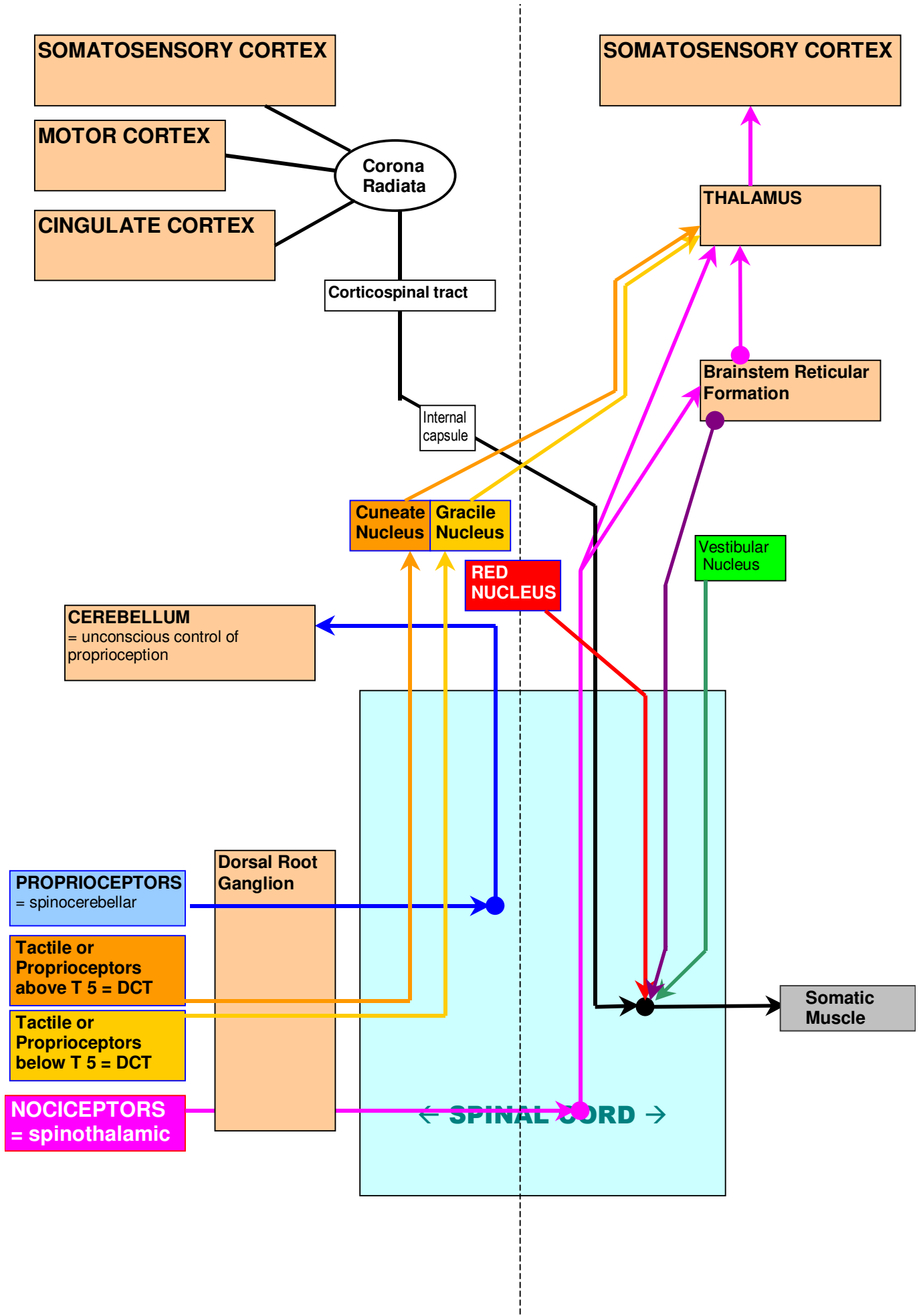
Sections Through Spinal Cord at Various Levels



Principal Fiber Tracts of Spinal Cord (composite)

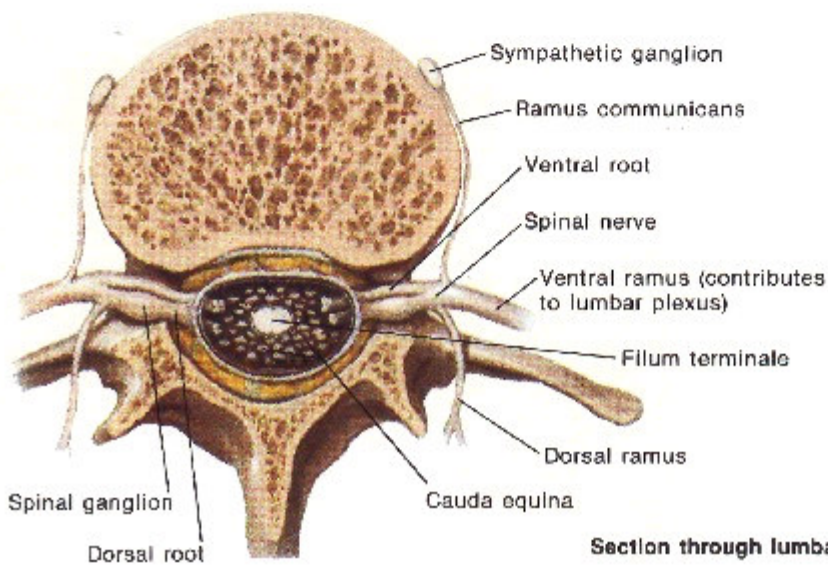
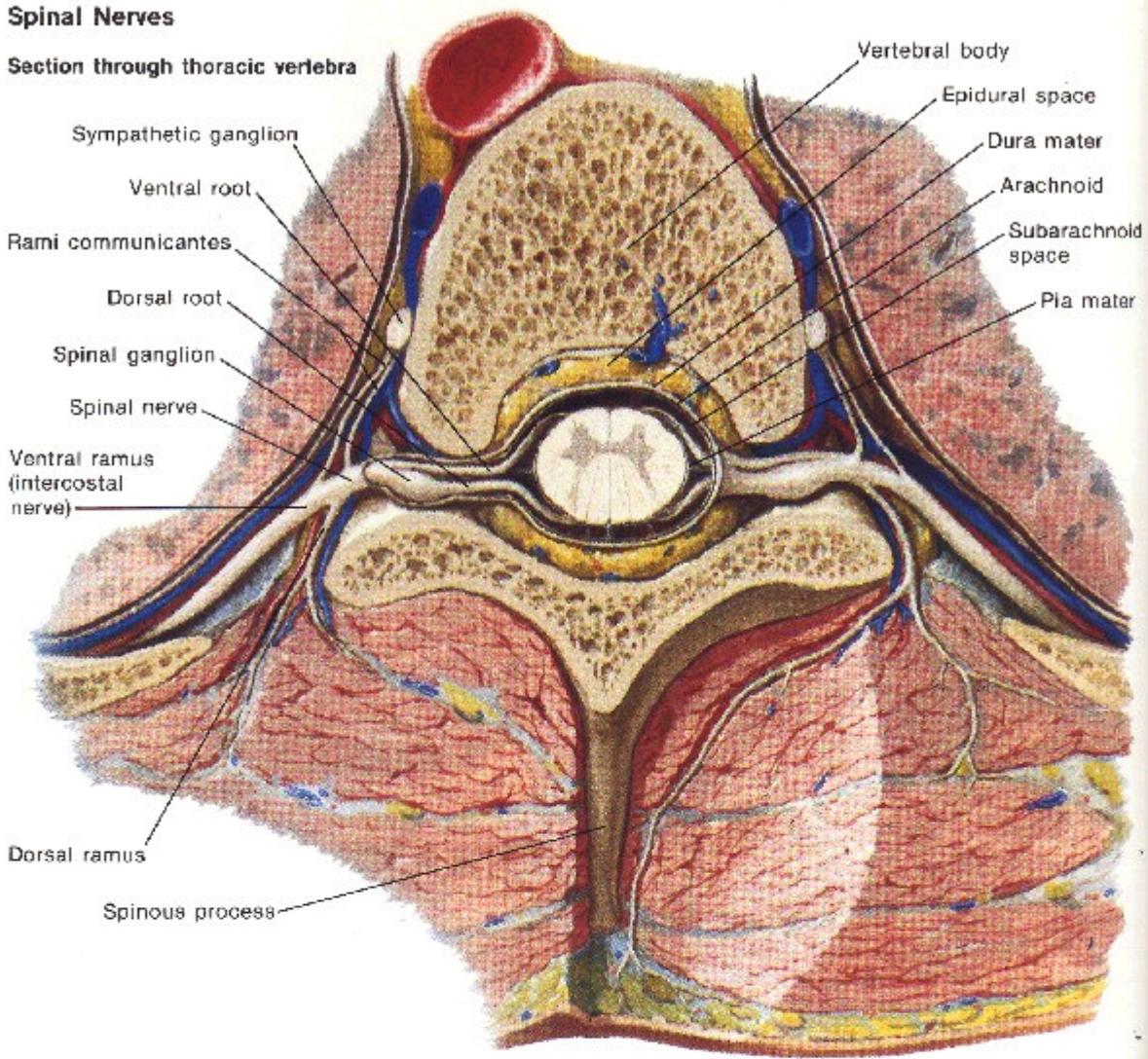
- Ascending pathways
- Descending pathways
- Fibers passing in both directions





Spinal Nerves

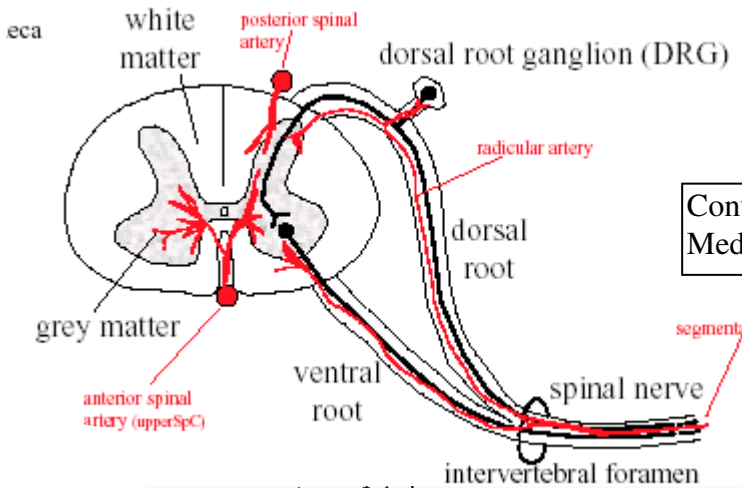
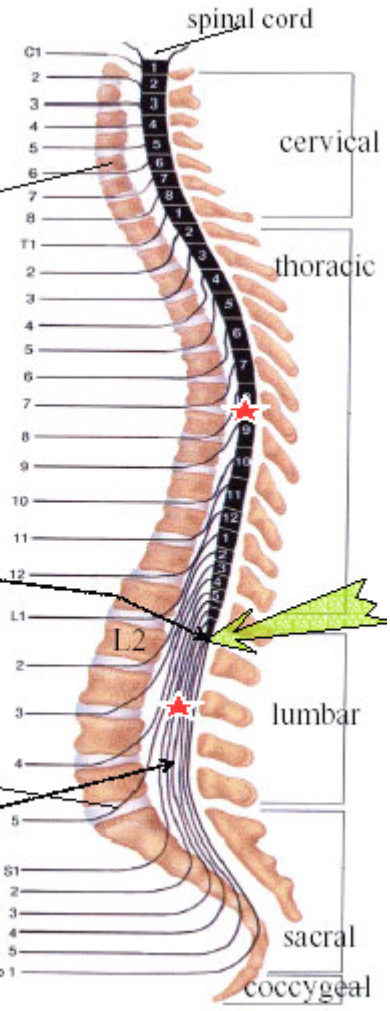
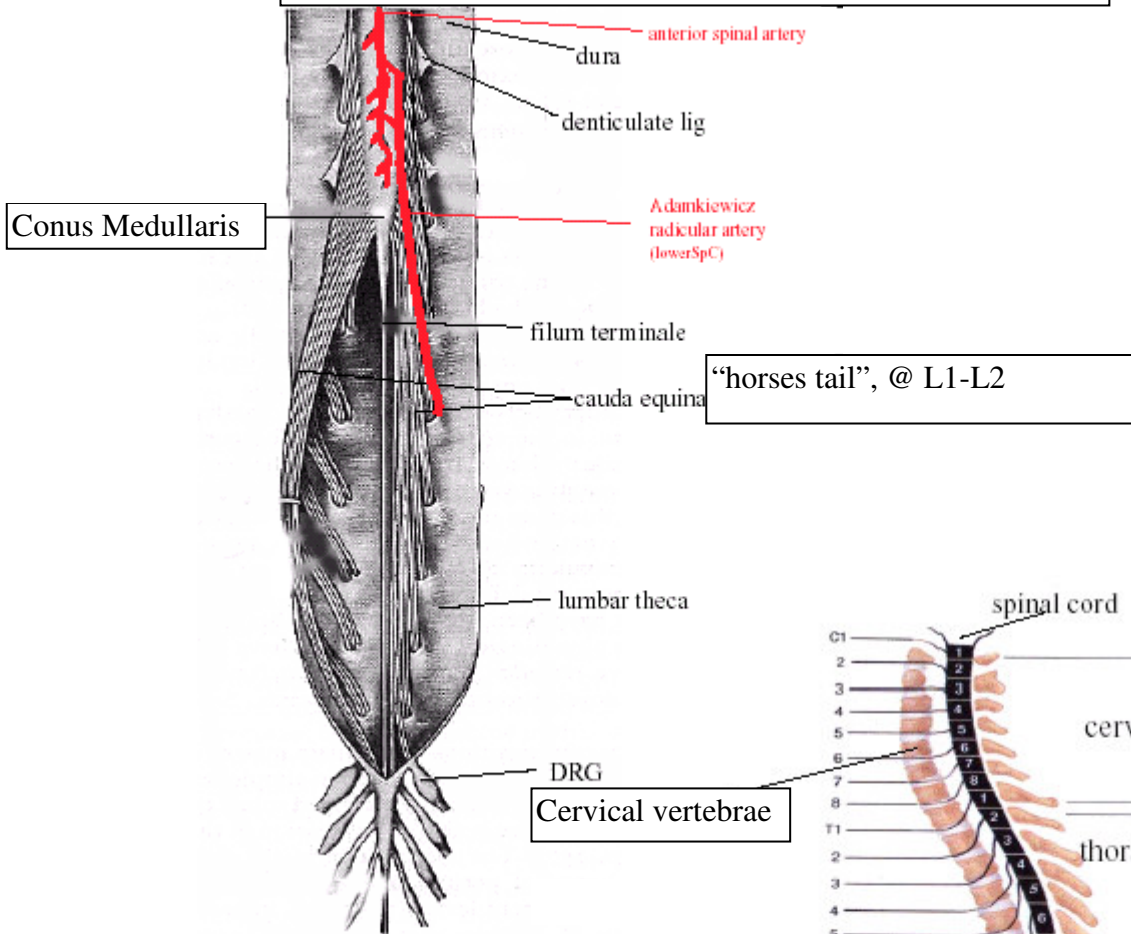
Section through thoracic vertebra



Section through lumbar vertebra

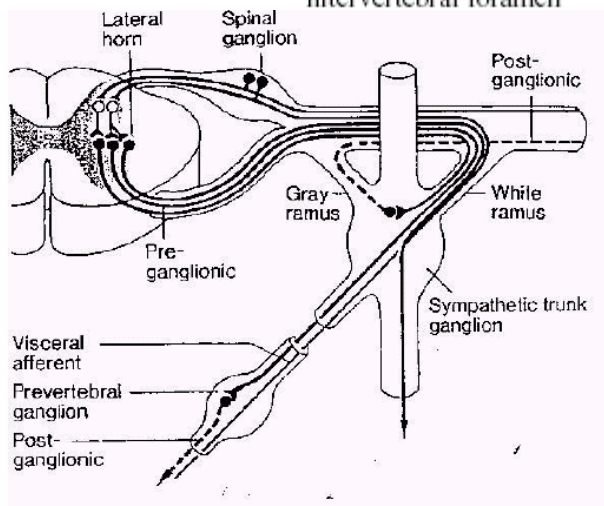
Mitrofanatomy of the Spinal Cord

Major blood supply: Adamkiewicz ("radicular") artery



Conus Medullaris

Cauda Equina



Spinal cord structure and function longitudinal organisation 6.01

Somatic Motor Organization:

Somatic motoneurons are located in Rexed lamina IX.

individual muscle = a separate portion of lamina IX,
usually two adjacent spinal segments.

Recall that the motor segmental supply of a muscle is called a myotome.

2 columns of motorneurons:

MEDIAL

The medial motor column

- throughout the length of the SC
- contains motoneurons innervating the **axial muscles of the body.**

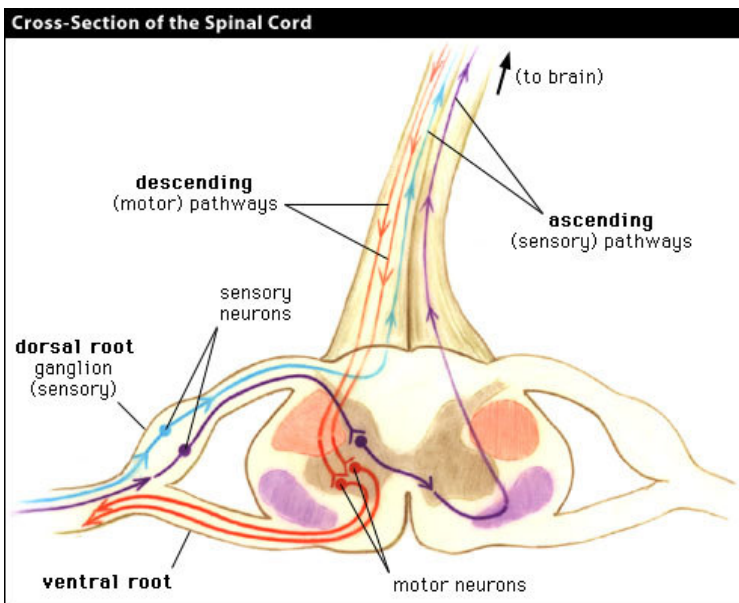
and

LATERAL

The lateral motor column

- **only in cervical and lumbar enlargements**
- contains motoneurons innervating the muscles of the **upper and lower limbs**

Cells of origin of motor pathway = UPPER MOTOR NEURONS
Cells in the ventral horn = LOWER MOTOR NEURONS



Damage to ventral horn

somatic motor neurons results in

- paralysis (absence of movement),
- wasting of denervated muscles,
- absence of muscle tone (flaccidity)
- an absence of reflexes (areflexia).

Damage to descending motor pathways results also in paralysis or paresis (weakness of movement),

however **without muscle wasting,**
with **normal or increased muscle tone**
(*spasticity*)
and **intact or enhanced reflexes** (hyper-reflexia).

THUS:
SPINAL CORD DAMAGE = PARALYSIS.
GREY MATTER DAMAGE: **WASTING + AREFLEXIA**
WHITE MATTER DAMAGE: **SPASTICITY + HYPERREFLEXIA**

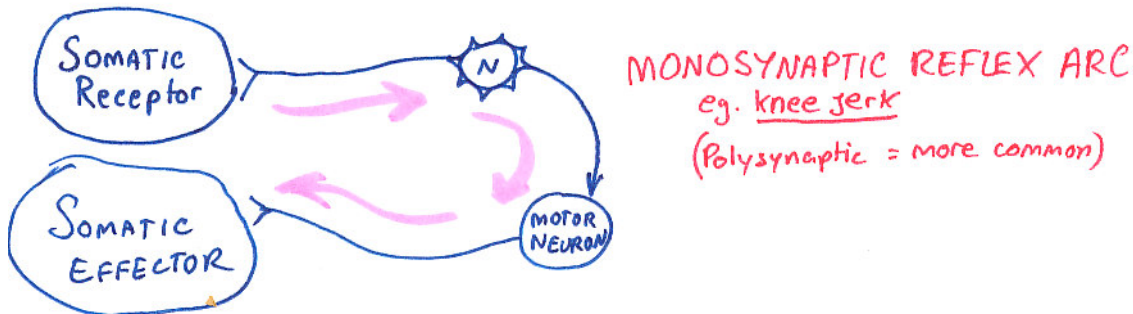
Damage to the spinal cord which involves both the grey and white matter produces both "upper" motoneuronal (white matter related) and "lower" motoneuronal (grey matter related) symptoms. Lower motoneuronal symptoms help to identify the level(s) of segmental damage.

Somatosensory Organization: PRIMARY AFFERENT INPUT PROCESSING

The central processing of primary afferent input involves **three main channels** of communication.

Reflex channels:

The high priority monosynaptic reflex arc is a rather recent evolutionary development. More common are polysynaptic reflex arcs which may involve coordinated activity across a large number of spinal segments, e.g., an unexpected, light touch on the shoulder (C3 dermatome) can evoke a dramatic startle response. The integration of activity across numbers of spinal segments is achieved by intersegmental axons (propriospinal fibres) of spinal interneurons.



Cerebellar channels:

Dorsal spinocerebellar tract neurons receive primary afferent proprioceptive input (deep tissues: muscles, tendons, joints) from the lower limb and "relay" it to the ipsilateral cerebellum.

A comparable tract sending proprioceptive information from the upper limb to the cerebellum arises from neurons in the external cuneate nucleus of the caudal medulla. **There is no conscious awareness of the proprioceptive information which reaches the cerebellum.**

General principles of lemniscal systems:

- 1st order neurons - cells bodies are
- found in periphery (e.g., dorsal root ganglia)
- there is always at least 1 synapse prior to the thalamus
- lemniscal systems are highly organised topographically
 - **FAITHFUL, not PROMISCUOUS**

Lemniscal channels:

Refer to the pathways by which cutaneous and proprioceptive information reaches consciousness. Conscious perception of somatosensation (touch, pressure, pain, temperature, position of limbs in space, more complex perceptions of size, weight, texture) depends on the ascending sensory information reaching a diencephalic target (the thalamus) and then the cerebral cortex. Only two major pathways need to be considered at present.

Dorsal columns (gracile and cuneate tracts):

Primary afferent information (primarily large diameter dorsal root fibres) enters the dorsal columns and ascends ipsilaterally the length of the spinal cord to terminate on second order neurons in the dorsal column nuclei of the medulla. Somatosensory information carried within the gracile and cuneate tracts provides the basis for our ability to perceive limb position and complex tactile sensations (e.g., weight, texture, shape).

Spinothalamic tract (spinal lemniscus):

Primary afferent information (small diameter, often unmyelinated fibres) synapse on second order neurons, located primarily in laminae I or V. The axons of these 2nd order lamina I and V neurons, then both ascend (not more than 1 or 2 segments) and cross to the opposite side to enter the "spinothalamic tract" within the ventral (anterior) part of the lateral column.

The spinothalamic tract is a component of what is sometimes called the anterolateral system. As the tract name implies, the axons ascend through the spinal cord and brain stem to terminate in the thalamus. Somatosensory information carried within the spinothalamic tract provides the basis, at least in part, for our ability to perceive and localise pain and temperature sensation, as well as simple touch.

Behavioural science

Serious spinal cord injury = a devastating change to physical and psychological

- changes in independence,
- social roles
- sexual roles
- vocational roles
- = disrupt the overall premorbid lifestyle.

Affects whole family

More risk of suicide, drug abuse, psychiatric illness

Data from the U.S.A. show **fewer marriages and more divorces**

Long term physical complications

Loss of extrinsic innervation → disturbance of bowel motility and co-ordinated propulsion, necessitating bowel training and optimisation of stool consistency.

Pain and spasticity also frequently interfere with function and rehabilitation and may require physical, pharmacological and psychological interventions.

Rehabilitation

The aim is

- to maximise independence,
- assist psychological adjustment
- reintegrate the individual into the community and workplace.

These outcomes require an **individualised, goal orientated rehabilitation program** provided by a **specialised inter-disciplinary team**.

Want your patient to be physically stronger, mobile and functioning in society

Psychosocial intervention focuses on the individual's and family's understanding of grief, anger, disbelief and helplessness.

Determinants of outcome after injury

Despondency, grieving and chronic pain have been found to be associated with poor adjustment to spinal injury,

Loss of normal sexual function, especially in males, can be a source of distress and reduction in self esteem, further contributing to psychological morbidity.

a good outcome is associated with:

- young age,
- female sex,
- higher educational level,
- the ability to relate well,
- confidence in one's mastery over the environment
- ability to have indulged in risk-taking behaviour.

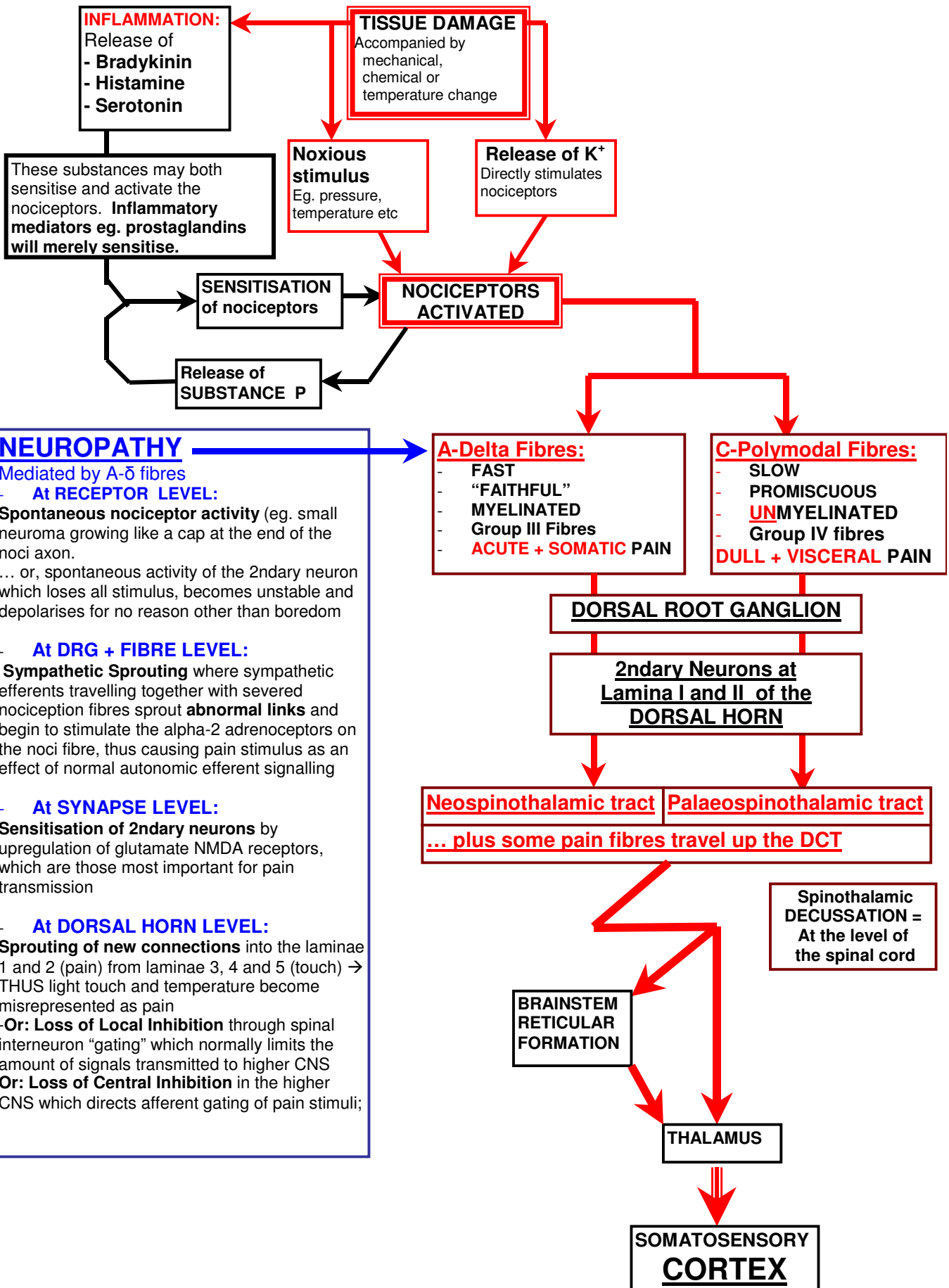
PAIN MANAGEMENT

Chronic Nociceptive:

oral analgesics + anti-inflammatories, opiates, epidural or neurosection

Chronic Neuropathic:

Anticonvulsants and antidepressants (rarely responds even to opiates)



Physiology: AUTONOMIC CONTROL OF CONTINENCE

BLADDER:

The **trigone** (triangular area at the urethra end of the bladder) and its buddy the **internal sphincter** are at all times contracted; for as long as they are, the pee stays put. THE REST of the bladder is the **DETRUSOR** muscle; it contracts to expel urine.

THUS: to urinate:

the **trigone and internal sphincter must relax**, and the **detrusor must contract**
DETRUSOR = innervated by ANS; Parasympathetic = contract, Sympathetic = relax
External sphincter = somatomotor voluntary control. Thank Christ

Continence reflex

Bladder fills = detrusor stretches

Stretch receptors: via visceral afferents **inform the Sacral Spinal Cord**

= REFLEX ARC:

→ inhibition of parasympathetic detrusor
(THUS = no contraction)

→ excitation of external urethral sphincter
via somatic **puddendal nerves**
(THUS turns off the tap)

...Until painful, when it takes avoluntary non-reflexive effort

Micturition reflex

Pontine and sacral micturition centres combine their power to produce

→ relaxation of external urethral sphincter
via inhibition of somatic **puddendal nerve** efferents

→ Contraction of the detrusor via parasympathetic efferents

→ detrusor contracts, thus

→ **shape of bladder changes**

→ **thus internal spincter is pulled open**

THUS, urine flows out: AND

→ **the urethra feeds back to sacrum**; and a reflex arc causes detrusor to contract more (positive feedback loop)

What happens in NEURO DYSFUNCTION?

If all nerves to the bladder are severed, IT KEEP FILLING until the pressure inside overcomes the outflow resistance and then the urine will leak continuously. Eeew.

BUT slowly after an SC injury the sacral micturition centres recover, and are able to control micturition via the sacral detrusor contraction reflex. THIS IS IMPERFECT as the afferent for this reflex must be powerful indeed (filled to ~150% capacity) and the emptying which occurs will be incomplete (~25% left)

BOWEL: two sphincters,

internal (smooth muscle) ← sacral parasympathetics
and external (striated) ← pudendal nerve somatomotor

Strangely, the rectal wall is NOT controlled by the enteric NS

Continence reflex

Rectum fills = its wall is stretched;

THUS: reflex relaxation of internal sphincter,
(via enteric NS in wall of rectum)

Reflex **CONTRACTION of external sphincter**

Reflex **CONTRACTION** of the wall of the rectum
(via visceral afferents running through the pelvic splanchnic nerve)

THIS LASTS A FEW SECONDS;

then the tension on the rectal wall is released and the internal sphincter contracts again.

The actual maintenance of the external sphincter tone is done by voluntary corticospinal neurons

Defaecation reflex

→ **!! Triggered by VOLUNTARY EFFORT !!** ←

visceral afferents from the rectal wall stretch receptors trigger a reflex arc:

→ **CONTRACTION** of descending and sigmoid colon and rectum

→ **RELAXATION** of the external AND internal sphincters

BUT: THAT ALONE AINT GOIN TO DO IT

Also requires increase in intra-abdominal pressure

THUS: one must **VOLUNTARILY**

→ **CONTRACT** the abdominal muscles

→ **CONTRACT** the diaphragm

so... sympathetic nerves do very little in defaecation

What happens in NEURO DYSFUNCTION?

DESTRUCTION OF THE SACRAL CORD = ABOLITION OF DEFAECATION REFLEX

...destruction of the SC anywhere else will mean that the sacral and enteric components will still work- BUT not any of the voluntary abdo-pressure-raising components.

THUS: need to MANUALLY COMPRESS THE ABDOMEN and even open the sphincter

CNS TRAUMA: a'la Roger Pamphlett

Primary injury: focal eg. traumatic impact, or diffuse eg. axonal oedema

FOCAL BRAIN INJURIES: contusions, haematomas, brainstem injuries

Commonest sequelae = ANOSMIA (severed olfactory bulbs)

Contusion:

- a "brain bruise"
- Bleeding from damaged small vessels
- usually the same areas affected, no matter what impact
- (usually seen in base of skull, at the inferior frontal and temporal lobes)
- Coup and contra-coup (at the impact site and opposite to it where the brain bounced)
- Contusions increase in size over hours

Intracerebral haematoma:

- defined as mass of blood greater than 2 cm, not in contact with surface
- Usually as result of skull fracture,
- Follows a short acceleration injury that shears blood vessels deep in the brain
- Half are unconscious on admission, 20% may be lucid
- Bad if its very big: causes mass effect, and thus coma
- May be delayed for some days after injury

Extradural Haematoma

- = bleeding between the skull and dura
- arterial, fast bleeds
- from a skull fracture in 90% adults, 70% kids
- (kids mid meningeal is much looser, while in adults its almost enveloped in bone)
- most commonly from a mid meningeal rupture following temporal bone fracture
- lucid period: "people who talk, walk, then die"

Subdural hematoma

- Due to rupture of bridging veins
- Angular acceleration of the head, (eg. falls, assaults)
- In alcoholics and elderly, subdural space is large, thus may not notice until very late
- Could be due to "burst lobe syndrome" = blood leaking out of damaged brain
- (these ones are unusually unconscious after injury)

Brainstem injury:

- Usually contusion;
- Often from base of skull fractures
- Sudden deceleration = violent rapid neck hyper-extension

Diffuse Axonal injury:

- Long term deceleration/acceleration, eg. MVA
- Rotation forces are important in this
- COMMON CAUSE OF POST-TRAUMATIC COMA when you cant find a macro lesion
- Only macro signs are hemorrhages in corpus callosum and lateral brain stem
- Axons swell microscopically, from 2 hrs after injury until ~ 1 months

Boxer's Brain Damage

- 75% of which is from subdural hematomae
- long-term damage: "punch-drunk syndrome" =
- = neurofibrillary tangles
- = diffuse amyloid plaques
- = looks a lot like dementia

Pathology of brain injury:

- primary cell damage : due to glutamate excitotoxicity
- secondary is due to hypoxia (ruptured vessels), oedema (cytotoxic, vasogenic)
- tertiary is due to mass effect eg. brainstem herniation ..
- or secondary Brainstem hemorrhage
- (PRIMARY CAUSE OF DEATH IN BRAIN TRAUMA)

Outcome of brain injury:

- subdural: 70% mortality, 10% complete recovery
- diffuse axonal : 10% mortality, 70% good recovery
- epilepsy in 5% of brain injuries

Syringomyelia

Signs of the disorder tend to develop slowly, although sudden onset may occur with coughing or straining

May extend into brainstem (syringobulbia)

Has “cape-like distribution” of DISSOCIATED SENSORY LOSS:

Loss of pain and temperature with retention of the other senses; DCT symptoms

Wasting of the small hand muscles

Painless burns

Winging of the scapula

Long Tract signs

Scoliosis