Case 1.

A previously well 22-year-old man presents to hospital with weakness and sensory disturbance.

Whilst on an evening out with friends he developed altered sensation in right hand. Within minutes this altered sensation affected the entire right arm and shortly after this involved left arm as well. Within 15 minutes he noticed weakness in his right hand and could no longer hold his beer. Within 30 minutes his legs were weak and he could no longer bear his own weight and had to be helped out of club into a taxi by support of two friends. His symptoms persisted into the next morning and he also became aware of an altered sensation down his left leg which went up into his thorax as well as urinary frequency.

Otherwise well. No comorbidity.

Cranial Nerves

	R	L
I	N	N
II	N	N
III, IV, VI	N	N
V	N	N
VII	N	N
VIII	N	N
IX,X,XI	N	N
XII	N	N

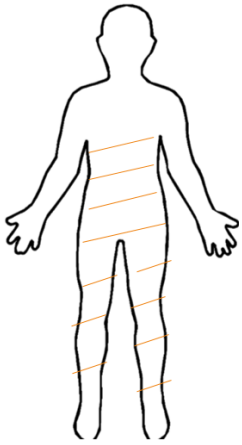
	R	L
Cerebellar	N	N
Pin Prick	Reduced	Reduced
JPS	N	N
Vib	N	N

Upper Limb

	R	L
Tone	N	N
Sh Ab	5	5
EF	5	5
EE	5	5
WE	5	5
FE	4	4
FDI	3	3
APB	3	3
Biceps	++	++
Triceps	++	++
Supinator	++	++

Lower Limb

	R	L
Tone	N	N
HF	3	4
HE	5	5
KF	3	4
KE	5	5
APF	3	3
ADF	5	5
AI	5	5
Knee	++	++
Ankle	++	++
Plantar	↑	↑

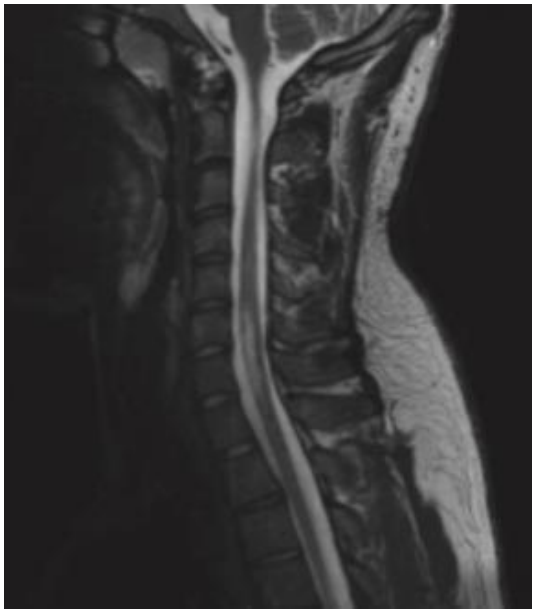


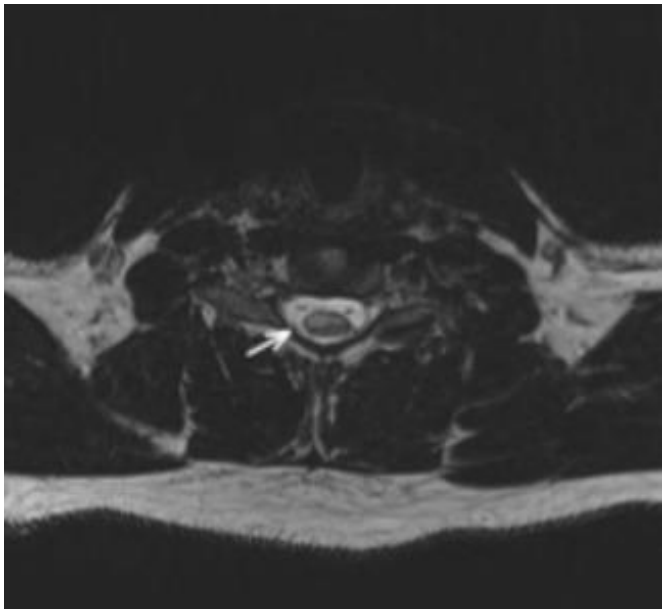
Stop and think 1.

1. ***Based on the examination findings are you able to localise the site of the lesion?***
2. ***What are the potential aetiologies for pathology at this site in the nervous system? How does the tempo of symptoms help you to narrow this down?***
3. ***What investigations would you perform?***

FBC	Units		Normal Range
Hb	g/L	124	120 – 160
MCV	fL	78	76-96
WC	10^9/L	8	4 – 11
PLT	10^9/L	160	140 – 450
B12	ng/mL	400	200 – 900
Folate	ng/mL	5	2-20
U&E			
Na	mmol/L	134	133 – 146
K	mmol/L	4.5	3.5 – 5.3
Urea	mmol/L	6	2.5 – 7.8
Creatinine	umol/L	90	0 – 110
LFT			
ALT	u/L	14	11 – 55
Alk Phosphatase	u/L	35	30 – 130
Bilirubin	umol/L	7	0 – 21
Albumin	g/L	45	35 – 50
INR		1.0	1.0
Bone Profile			
Calcium	mmol/L	2.18	2.18 – 2.62
Phosphate	mmol/L	0.8	0.8 – 1.5

CSF			
Opening pressure	cmH20	16	14 – 20
WC		4	<5
RC		0	<5
Protein	g/L	0.61	0.2 – 0.4
Glucose	Mmol	4	60 – 100% serum
Serum glucose	Mmol/L	5	4 – 7.8





MRI cerivcal spine demonstrating increased signal around C7/C8. On axial imagining this signal change is more prominent in anterior cord.

Diagnosis:

Anterior spinal cord infarct presumed secondary to cocaine use.

Case 2.

A previously healthy 17-year-old male patient presented to hospital with a 2-week history of sensory disturbance and limb weakness. He first noted distal lower limb paresthesia and a week later developed progressive weakness of his lower and then upper limbs. He subsequently developed bladder dysfunction with urinary frequency and urgency. Otherwise fit and well with no medical comorbidity. Patient admitted to frequent use of nitrous oxide in form of 'whippits'. In the 48 hours preceding symptom onset used >40 canisters

Cranial Nerves

	R	L
I	N	N
II	N	N
III, IV, VI	N	N
V	N	N
VII	N	N
VIII	N	N
IX,X,XI	N	N
XII	N	N

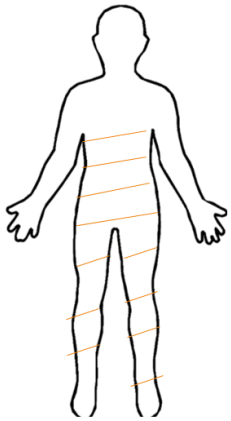
	R	L
Cerebellar	N	N
Pin Prick	N	N
JPS	Reduced	Reduced
Vib	Reduced	Reduced

Upper Limb

	R	L
Tone	Spasticity	Spasticity
Sh Ab	4	4
EF	5	5
EE	4	4
WE	3	3
FE	4	4
FDI	5	5
APB	5	5
Biceps	+++	+++
Triceps	+++	+++
Supinator	+++	+++

Lower Limb

	R	L
Tone	N	N
HF	3	4
HE	5	5
KF	3	4
KE	5	5
APF	3	3
ADF	5	5
AI	5	5
Knee	+++	+++
Ankle	+++	+++
Plantar	↑	↑



Stop and think

1. **What are the main differences with the previous case? How does this help us localise the site of pathology?**
2. **What are the possible aetiologies and what investigations would you request?**

FBC	Units		Normal Range
Hb	g/L	124	120 – 160
MCV	fL	78	76-96
WC	10*9/L	8	4 – 11
PLT	10*9/L	160	140 – 450
B12	ng/mL	233	200 – 900
Folate	ng/mL	5	2-20
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Na	mmol/L	134	133 – 146
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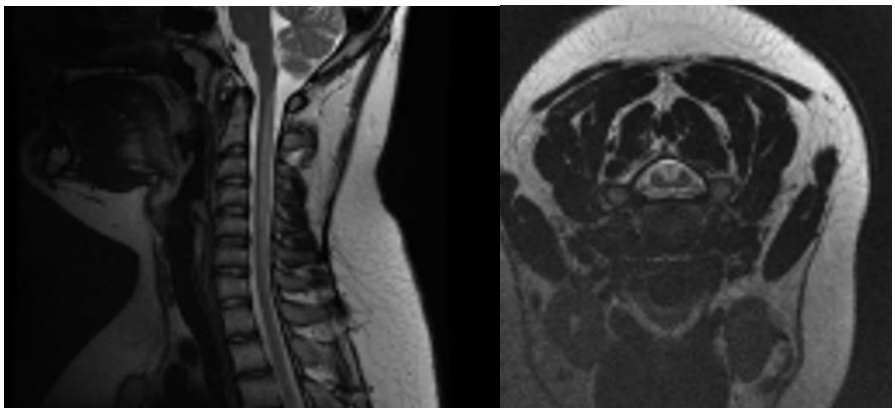
Bilirubin	umol/L	7	0 – 21
Albumin	g/L	45	35 – 50
INR		1.0	1.0
Bone Profile			
Calcium	mmol/L	2.18	2.18 – 2.62
Phosphate	mmol/L	0.8	0.8 – 1.5

MMA	1.94 umol/L ↑	0.00–0.25 umol/L
Homocysteine	42.0 umol/L ↑	5.0–14.0 umol/L

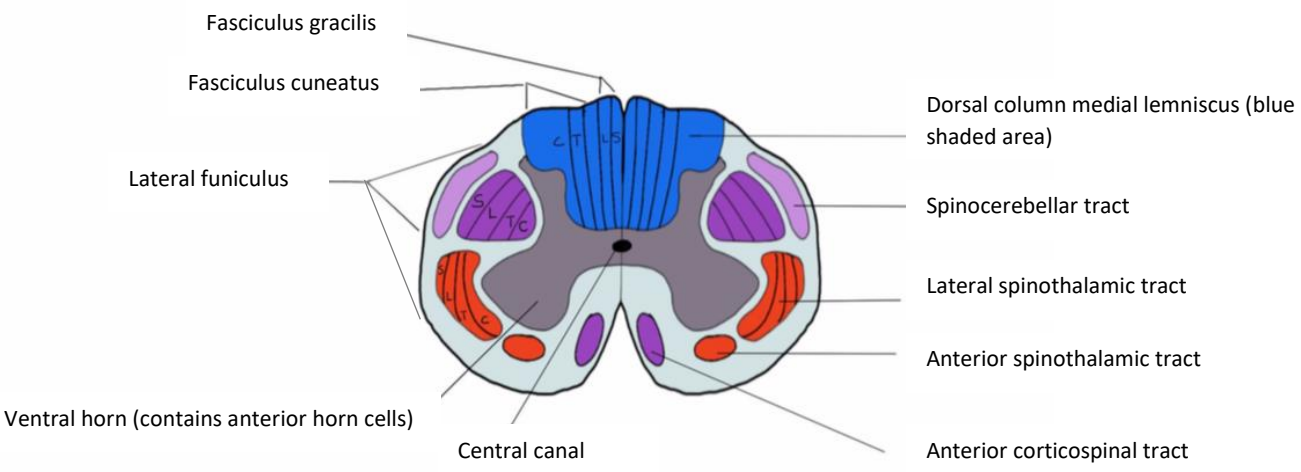
CSF			
Opening pressure	cmH20	14	14 – 20
WC		3	<5
RC		0	<5
Protein	g/L	0.34	0.2 – 0.4
Glucose	Mmol	4	60 – 100% serum
Serum glucose	Mmol/L	5	4 – 7.8



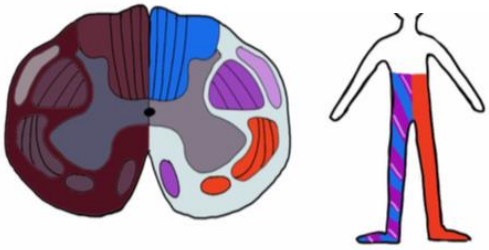
MRI cervical spine demonstrating increased signal throughout entire cervical spine, on axial imagining this increased signal is located in posterior cord involving dorsal columns.

Diagnosis: Posterior cord syndrome secondary to 'functional B12 deficiency' secondary to nitrous oxide use.

Spinal Cord Syndromes

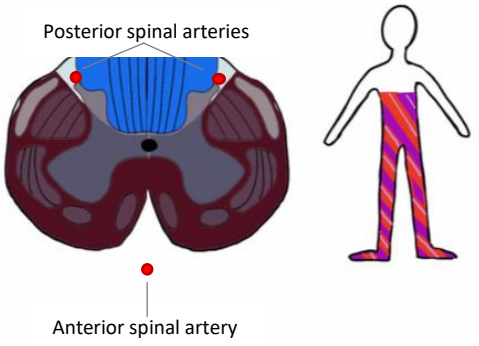


Somatotopic organisation shown C= Cervical, T= Thoracic, L=Lumbar, S= Sacral



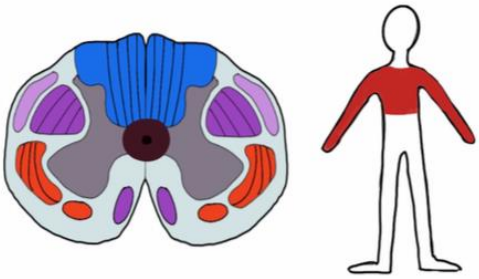
Brown-Séquard Syndrome

'Hemi-section' or lesion of half of the cord causes weakness, loss of vibration and proprioception ipsilateral and contralateral loss of pain and temperature sensation distal to the lesion. This occurs because the spinothalamic tract crosses at the level of entry to the spinal cord, whereas the corticospinal tract and dorsal columns cross in the medulla



Anterior cord syndrome

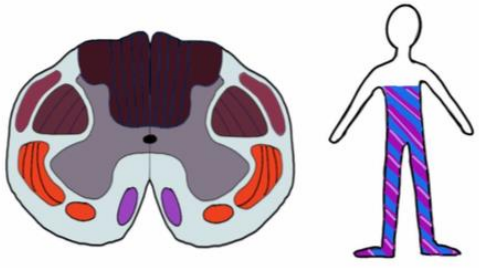
Vascular supply to the anterior cord is via the anterior spinal artery. Occlusion of this results in an anterior cord syndrome, causing symmetrical loss of pain, temperature and weakness distal to the lesion due to impairment of spinothalamic and corticospinal tracts respectively. Dorsal column function (vibration and proprioception) is preserved due to different



Central cord syndrome

A central fluid filled cavity expands outwards, initially causing disruption to the crossing spinothalamic tracts. This most commonly occurs in the cervical cord, causing a 'cape like' distribution of pain and temperature sensation loss across the upper chest and arms. As it expands it affects the anterior horn cells and corticospinal tracts causing weakness.

Expansion of the central canal is known as hydromyelia; if



Subacute combined degeneration of the cord

Impairment of the posterior and lateral columns (most commonly due to B12 deficiency) causes loss of vibration, proprioception, ataxia and weakness as a result of impairment of the dorsal column, and later, spinocerebellar and corticospinal tracts.

***'Dissociated sensory loss'**- when a sensory modality such as proprioception is impaired and pain and temperature are preserved (and vice versa) this is referred to as dissociated sensory loss. This clinical finding is a useful marker for spinal cord pathology.*