Imperial College London

CNS Trauma: imaging

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Overview

Background about traumatic brain injury

Long term consequences

Pathophysiology

Clinical imaging

Advanced imaging of brain connectivity

Traumatic Brain Injury (TBI)

TBI is the leading cause of disability in people under 40.

Peak age group is 15-25 years

Annual number of TBI in the UK: 150,000-Minor
10,000-Moderate
4,500-Severe

Lifetime cost: $3 million (severe); $941,000 (moderate); $85,000 (mild)

Mild TBI is the most common neurological condition in the US.

WHO: major public health problem / need for long-term treatment

Outcome

Outcome is unpredictable:

At 5-7 yr follow up…
25% died
29% had improved
25% had deteriorated

Whitnall JNNP '06

Cognitive and neuropsychiatric factors are critical to good outcome

Long-term consequences

Cognitive

Memory
Attention
Concentration
Distractability

Physical

Weakness
Visual loss
Anosmia
Vestibular

Endocrine/Sleep

Lethargy
Weight change
Mood disturbance
…

Neuropsychiatric

Mood change
Anxiety
Emotional lability
PTSD

Epilepsy

Generalised
Focal

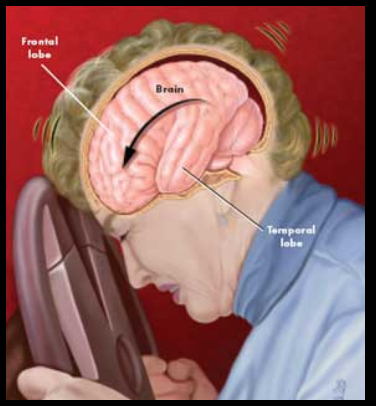
Chronic pain

Headache
Complex regional pain syndromes
Neuromuscular spasticity

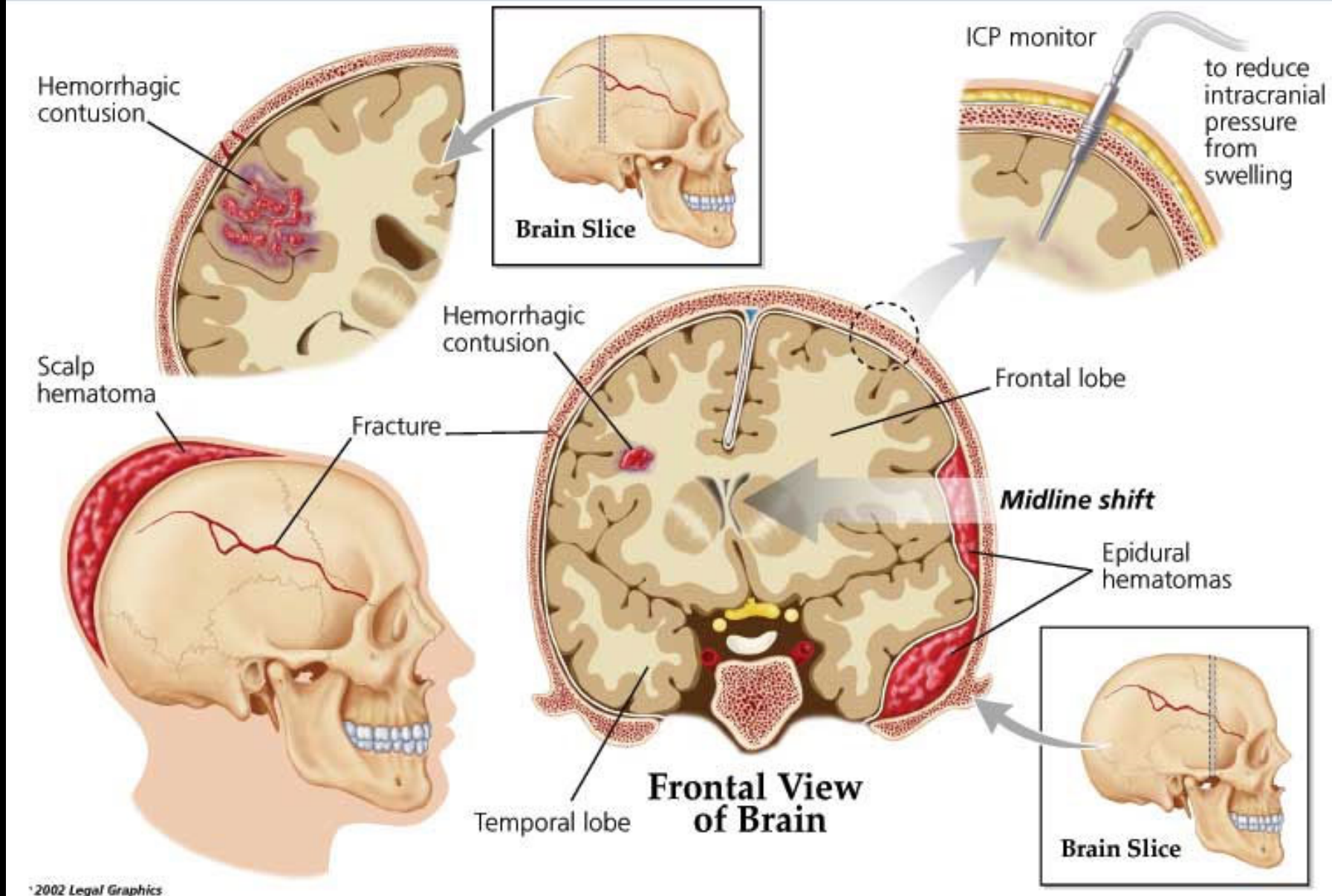
Traumatic brain injury - pathophysiology

Types of brain injury

- Two broad types of brain injuries:
  - Focal damage
    - Fractures
    - Intracerebral contusions
    - Bleeding
      - subarachnoid haemorrhage
      - extradural and subdural haematomas
      - Intracerebral haemorrhage
  - Diffuse axonal injury (DAI)
    - Damage to the connections between regions

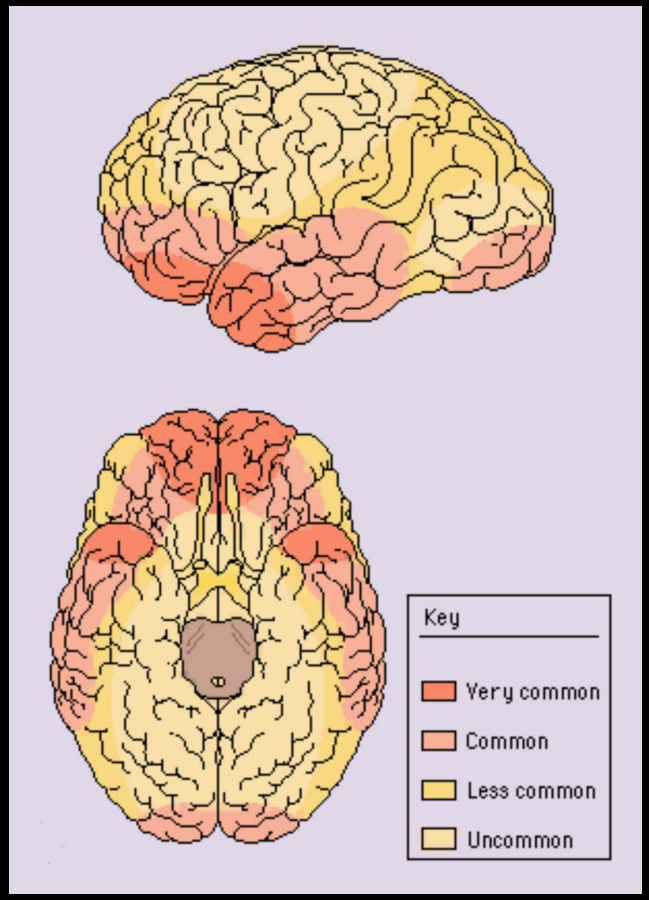


Traumatic Brain Injury



Pathophysiology: contusions & axonal injury

Contusion location



Love & Ellison: Neuropathology

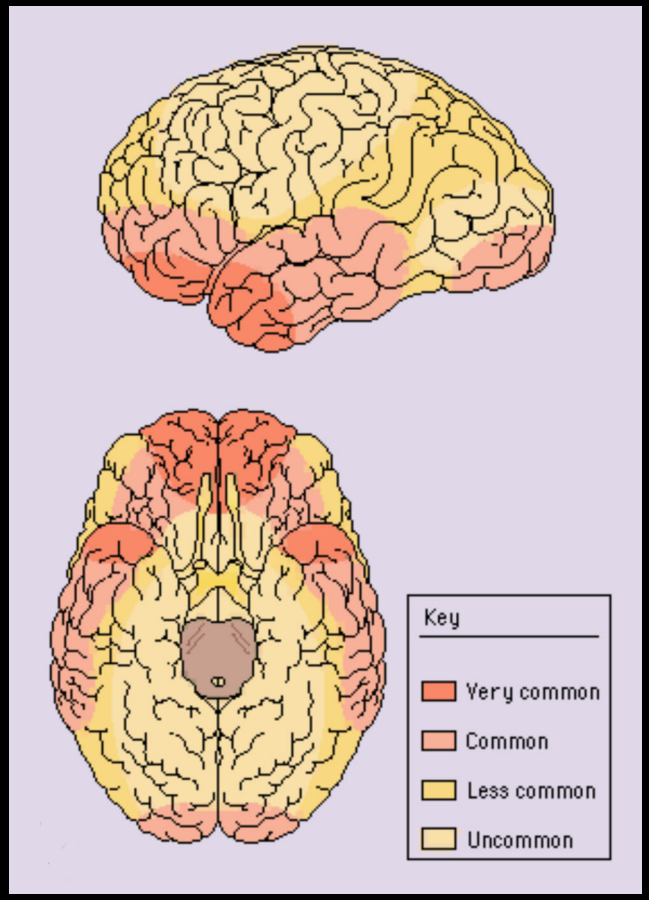
Pathophysiology

Diffuse axonal injury (DAI)

- Scattered or multifocal axonal change in subcortical WM, corpus callosum and brainstem
- Mechanical tearing/shearing of axon in severe cases
- More commonly abnormality within the axolemma
- Leads to axon transport impairment, swelling and downstream detachment
- In large myelinated axons probably secondary to calcium influx and protein activation
- Occurs over hours to days

Pathophysiology: contusions & axonal injury

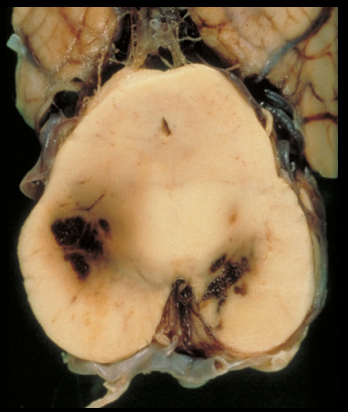
Contusion location



Love & Ellison: Neuropathology

Diffuse axonal injury

Grade 2: lesions in corpus callosum



Grade 3: lesions also in brainstem

Adams et al '85

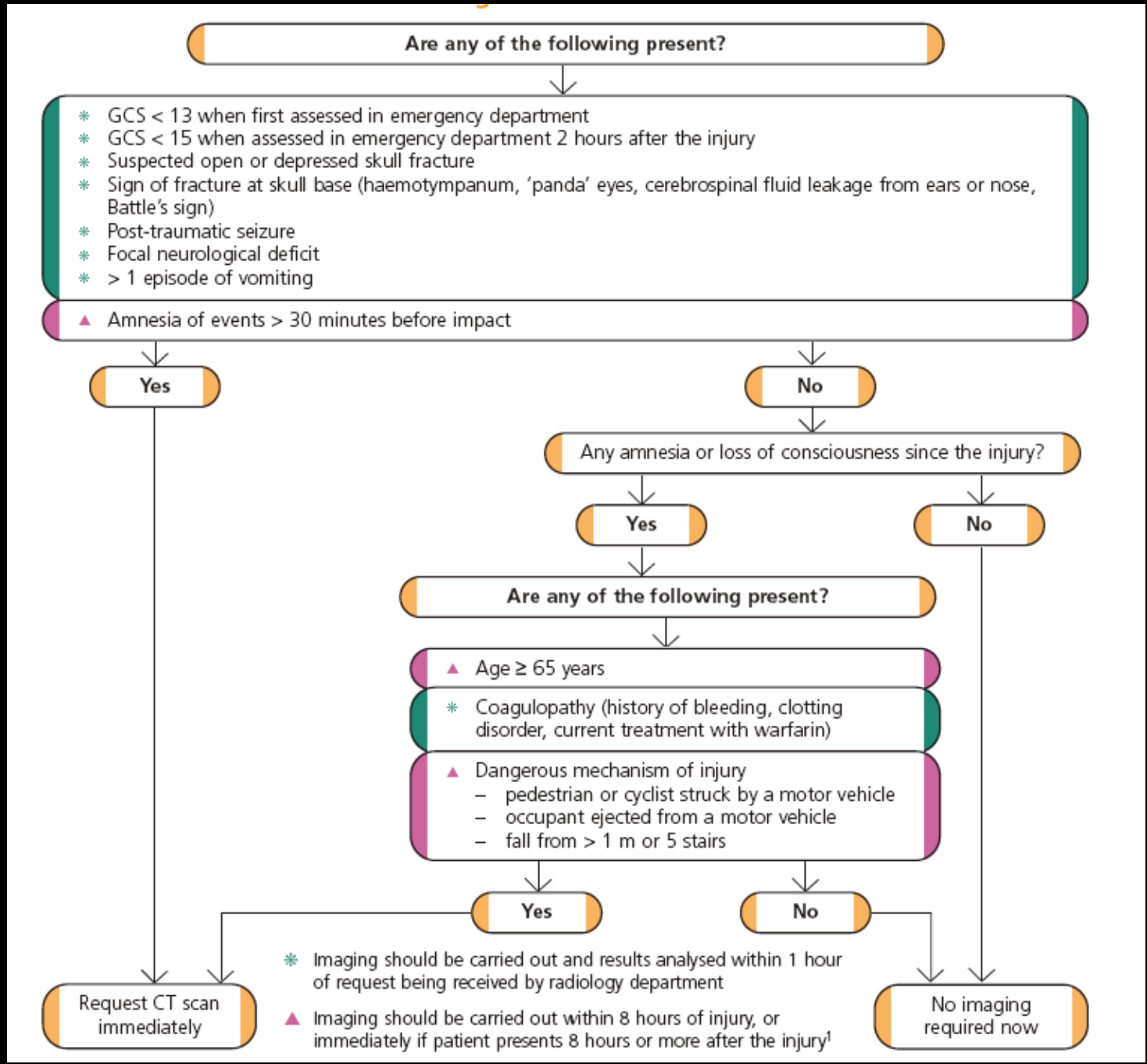
28 year old man brought in from a nightclub after being hit by a bouncer. He fell and hit hit head on the pavement. You are the A+E SHO, what do you do?

Imaging

Acute – CT

Chronic – CT or MRI

Initial assessment – NICE guidelines


Are any of the following present?
GCS < 13 when first assessed in emergency department
GCS < 15 when assessed in emergency department 2 hours after the injury
Suspected open or depressed skull fracture
Sign of fracture at skull base (haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from ears or nose, Battle's sign)
Post-traumatic seizure
Focal neurological deficit
> 1 episode of vomiting
Amnesia of events > 30 minutes before impact
Yes
No
Any amnesia or loss of consciousness since the injury?
Yes
No
Are any of the following present?
Age ≥ 65 years
Coagulopathy (history of bleeding, clotting disorder, current treatment with warfarin)
Dangerous mechanism of injury
– pedestrian or cyclist struck by a motor vehicle
– occupant ejected from a motor vehicle
– fall from > 1 m or 5 stairs
Yes
No
Request CT scan immediately
No imaging required now
Imaging should be carried out and results analysed within 1 hour of request being received by radiology department
Imaging should be carried out within 8 hours of injury, or immediately if patient presents 8 hours or more after the injury[1]

When to involve the neurosurgeon

- Discuss the care of all patients with new, surgically significant abnormalities on imaging with a neurosurgeon (definition of 'surgically significant' to be developed by local neurosurgical unit and agreed with referring hospitals).
- Regardless of imaging, other reasons for discussing a patient's care plan with a neurosurgeon include:
  - persisting coma (GCS ≤ 8) after initial resuscitation
  - unexplained confusion for more than 4 hours
  - deterioration in GCS after admission (pay greater attention to motor response deterioration)
  - progressive focal neurological signs
  - seizure without full recovery
  - definite or suspected penetrating injury
  - cerebrospinal fluid leak.

Traumatic brain injury – CT imaging

CT

- First CT in Hounsfield's EMI lab in London – 1972 (Nobel in 1979)
- X-rays pass in multiple directions through 'object'
- 3-D reconstruction based on the differential attenuation the beams
- Relative degree of attenuation expressed in Hounsfield units
- Water = zero
  - CSF = 3
  - White matter = 30
  - Gray matter = 38
  - Fresh blood = 81
  - Bone >1000

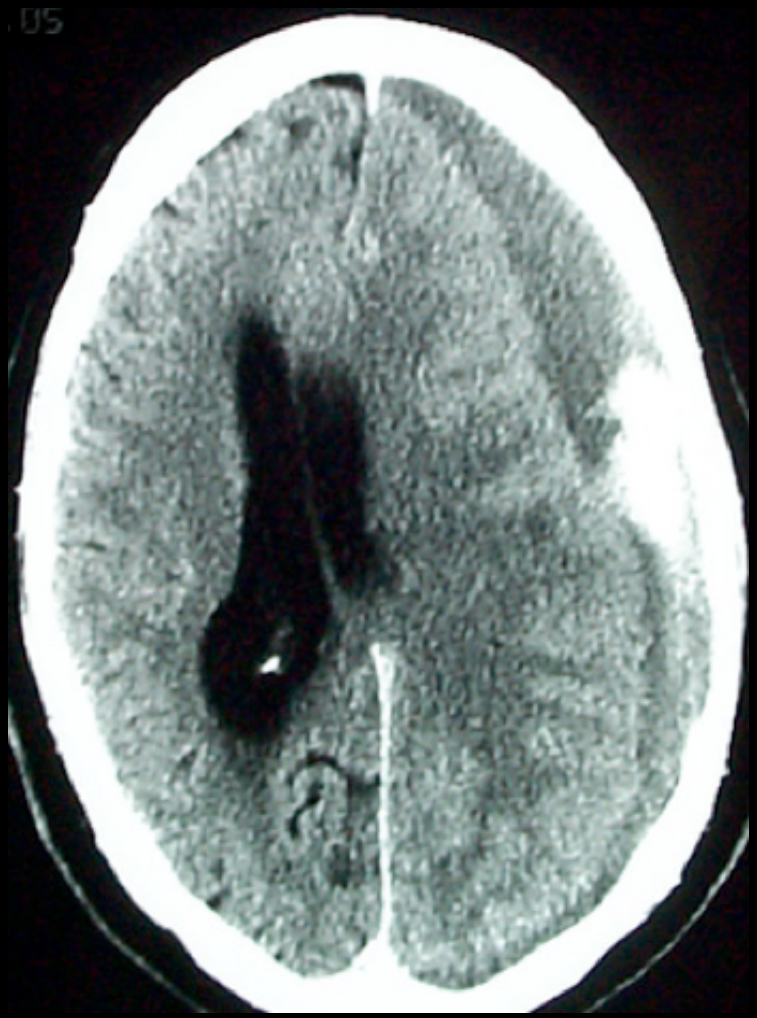
Advantages of CT

- Quick
- Cheap
- Better than MR at identifying
  - Blood
  - Bony abnormalities
  - Calcification
- Less claustrophobia
- But relatively poor resolution and limited by artefacts

CT: traumatic brain injury

Extradural

Acute on chronic subdural



CT examples

CT examples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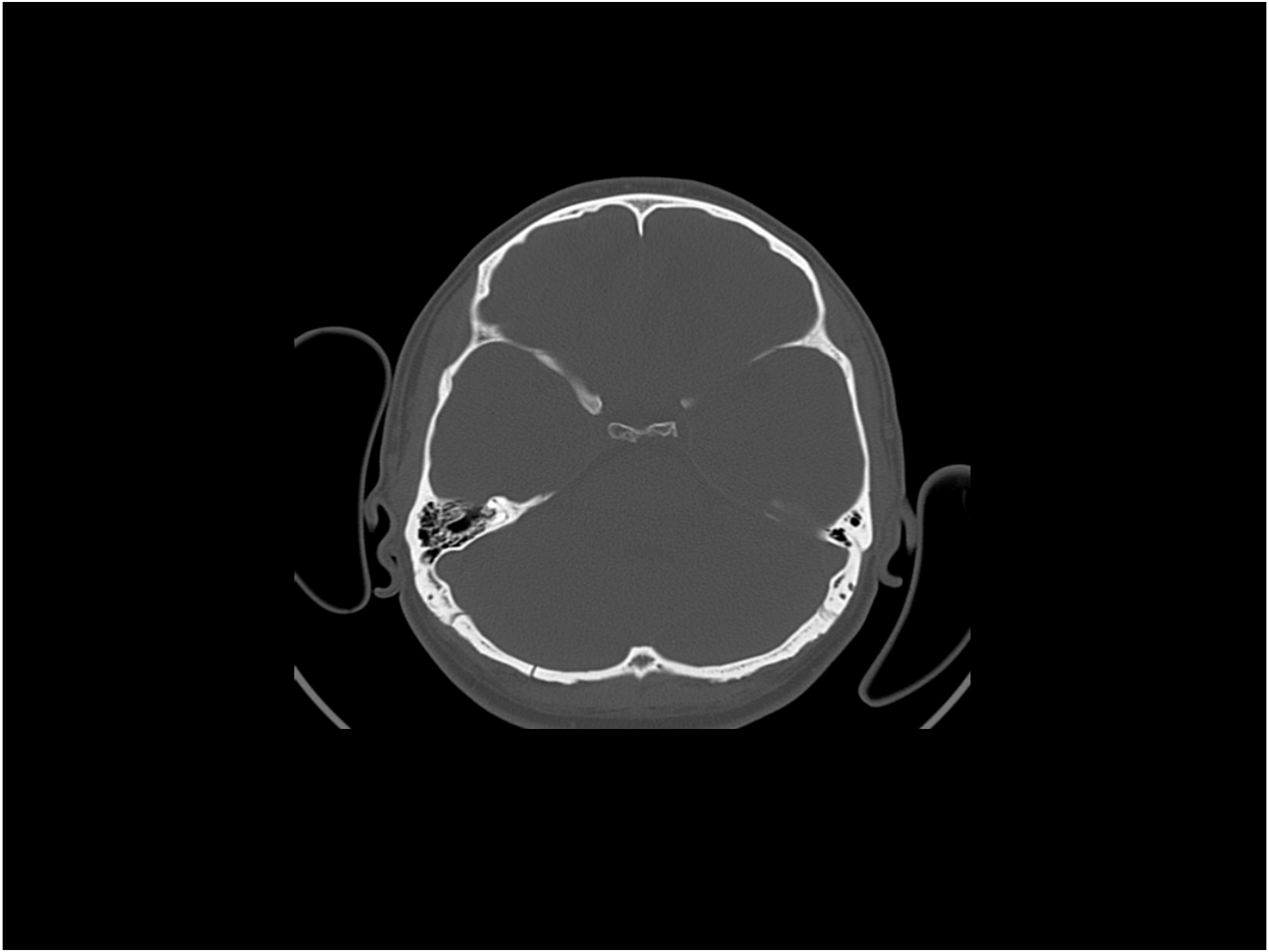
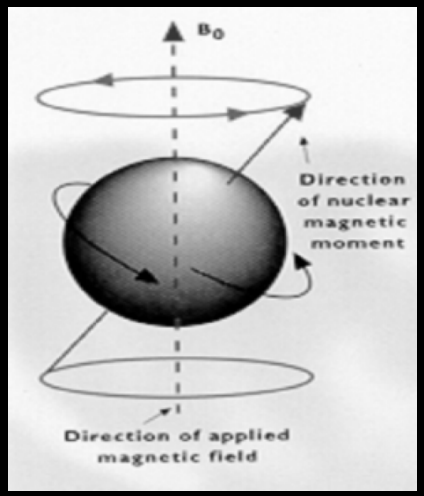


Traumatic brain injury – magnetic resonance imaging

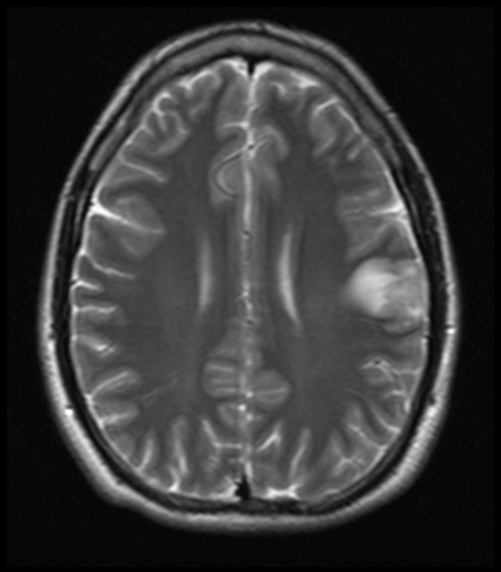
MRI – principles

- Signal results from interplay of tissues and applied magnetic fields.
- Most MR imaging based on proton imaging of the hydrogen nucleus.
- Hydrogen nuclei are aligned by the presence of a primary static magnetic field within the scanner.
- Within the magnetic field the hydrogen nucleus precesses at its own unique resonant frequency (Lamor frequency).
- Applying an RF pulse at the this frequency knocks the hydrogen nucleus out of alignment, before it gradually relaxes back to original position.
- T1 and T2 relate to different measures of this relaxation time.
- RF receiver coils receive the magnetic changes associated with these changes and convert to electric current



MRI – Sequences

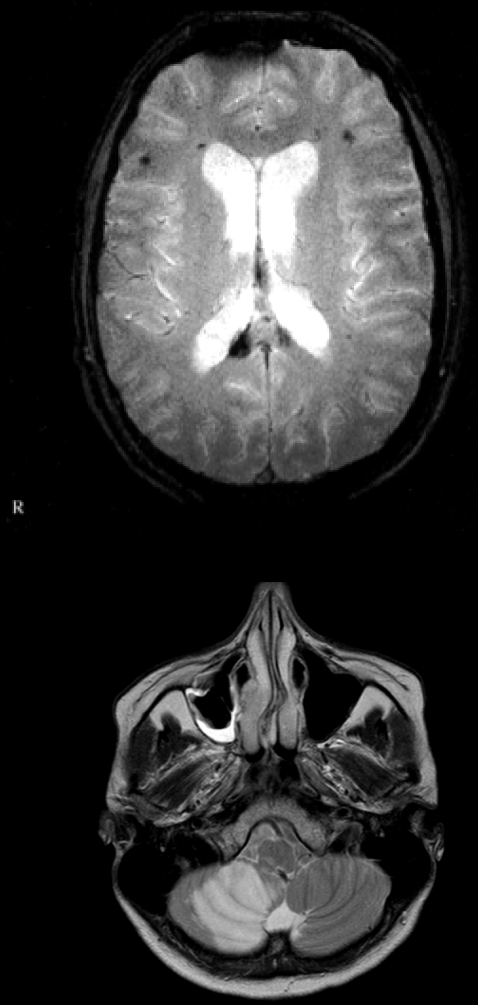
- **Conventional spin echo**
  - Has varying T1 and T2 relaxation effects
- T1
  - Good tissue discrimination
  - Used in conjunction with gadolinium
  - Dark CSF
  - Bright fat
  - Dark lesions
- T2
  - Sensitive to the presence of increased water
  - Visualises oedema
  - Bright CSF
  - Dark (suppressed fat)
  - Bright lesions



MRI – Sequences

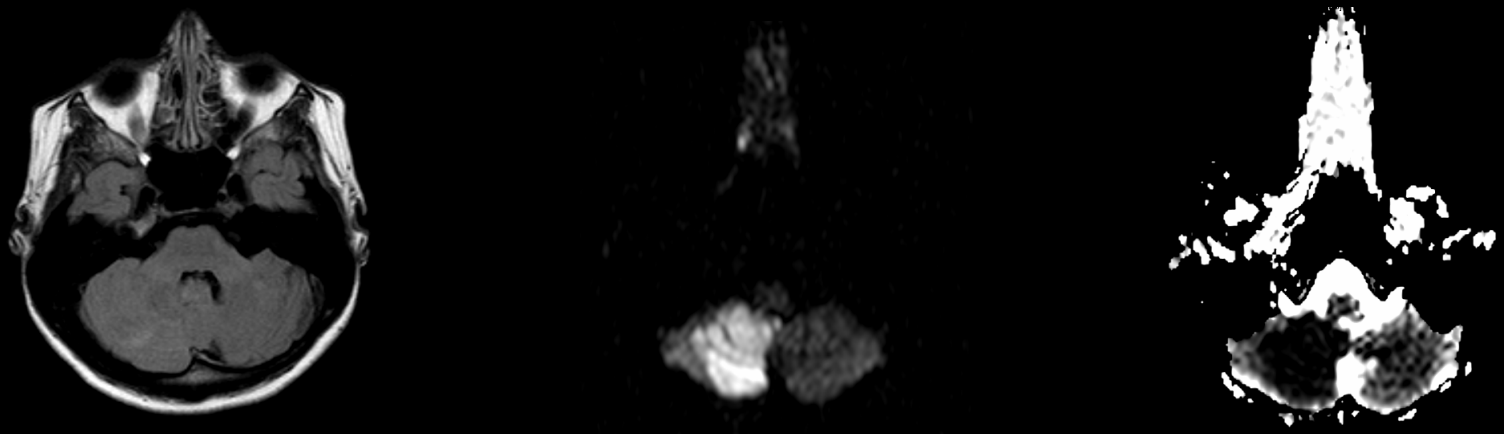
- **Gradient echo imaging (T2*)**
  - Increased susceptibility to magnetic field inhomogeneities
  - Blood, iron, calcium and manganese produce artefact
  - This can be useful to detect clinically

- **Flair**
  - Fluid attenuation inversion recovery
  - Developed at The Hammersmith
  - T2 weighted contrast with a dark CSF
  - Changes the dynamic range of the image
  - Better a delineating pathology, particularly around the ventricles



MRI – Sequences

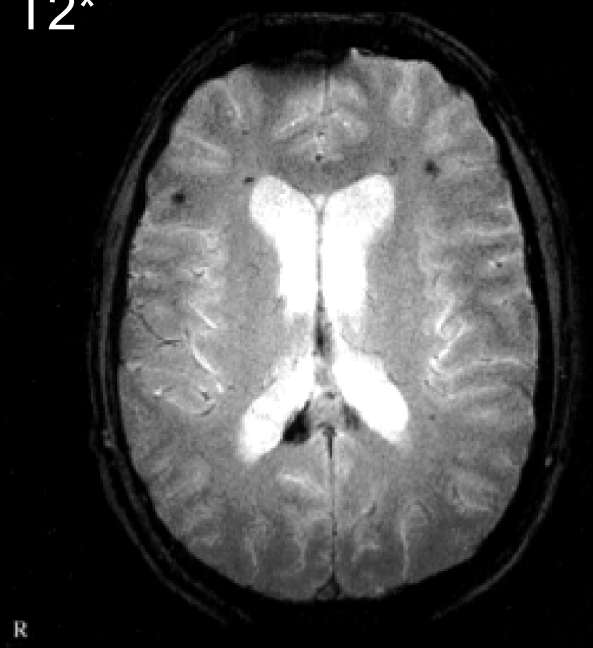
- **Diffusion weighted imaging (DWI)**
  - Imaging of water diffusion
  - As cells swell e.g. after an infarct diffusion reduces
  - This results in high signal on DWI …
  - And low on the apparent diffusion coefficient map (ADC)
  - DWI will demonstrate cerebral ischaemia within minutes of irreversible damage
  - Differentiates from TIAs



MRI imaging : microbleeds

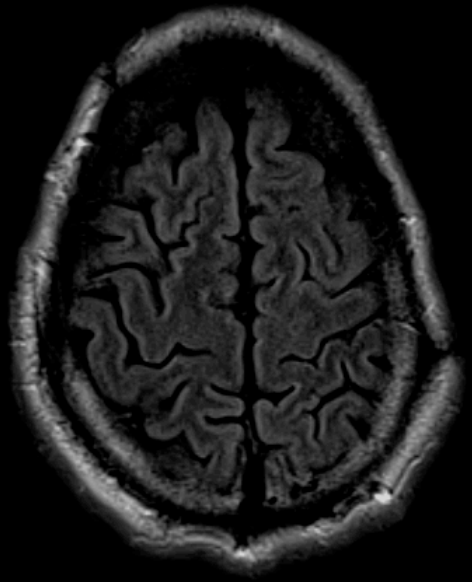
- Microbleeds on Gradient echo imaging are an MR marker of diffuse axonal imaging.

T2*

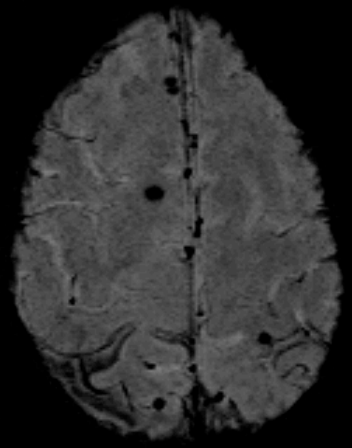


Imaging microbleeds

Flair



T2



T2*, gradient echo
(Susceptibility weighted imaging)

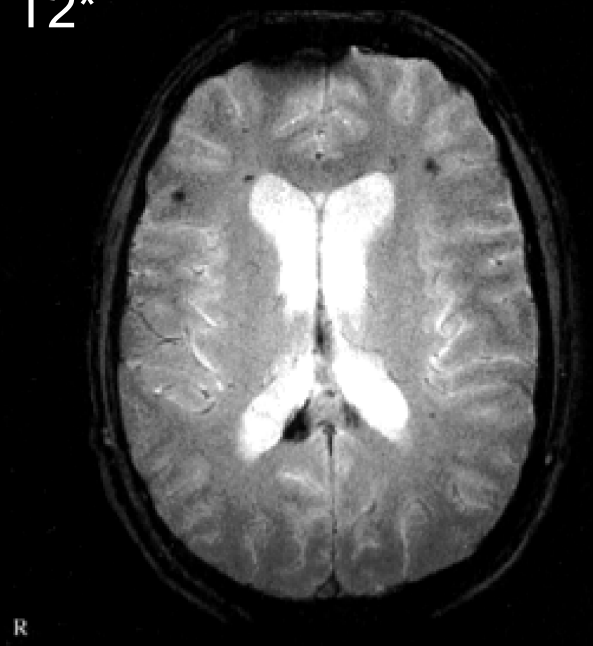
Diffuse axonal injury following TBI

- In isolation this results in cognitive impairment, particularly in memory and executive function.

  (Scheid et al Arch Neurol 07)

- Often associated with impairments of consciousness.

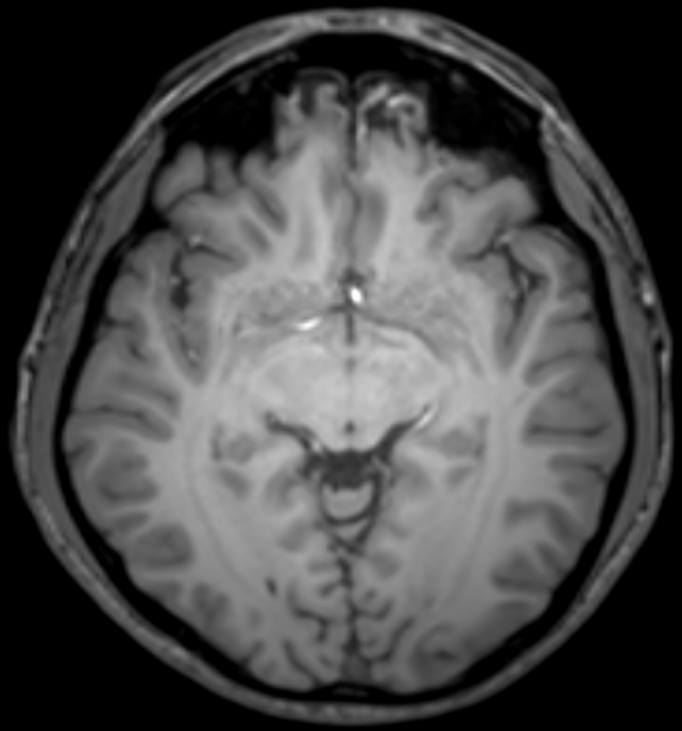
T2*

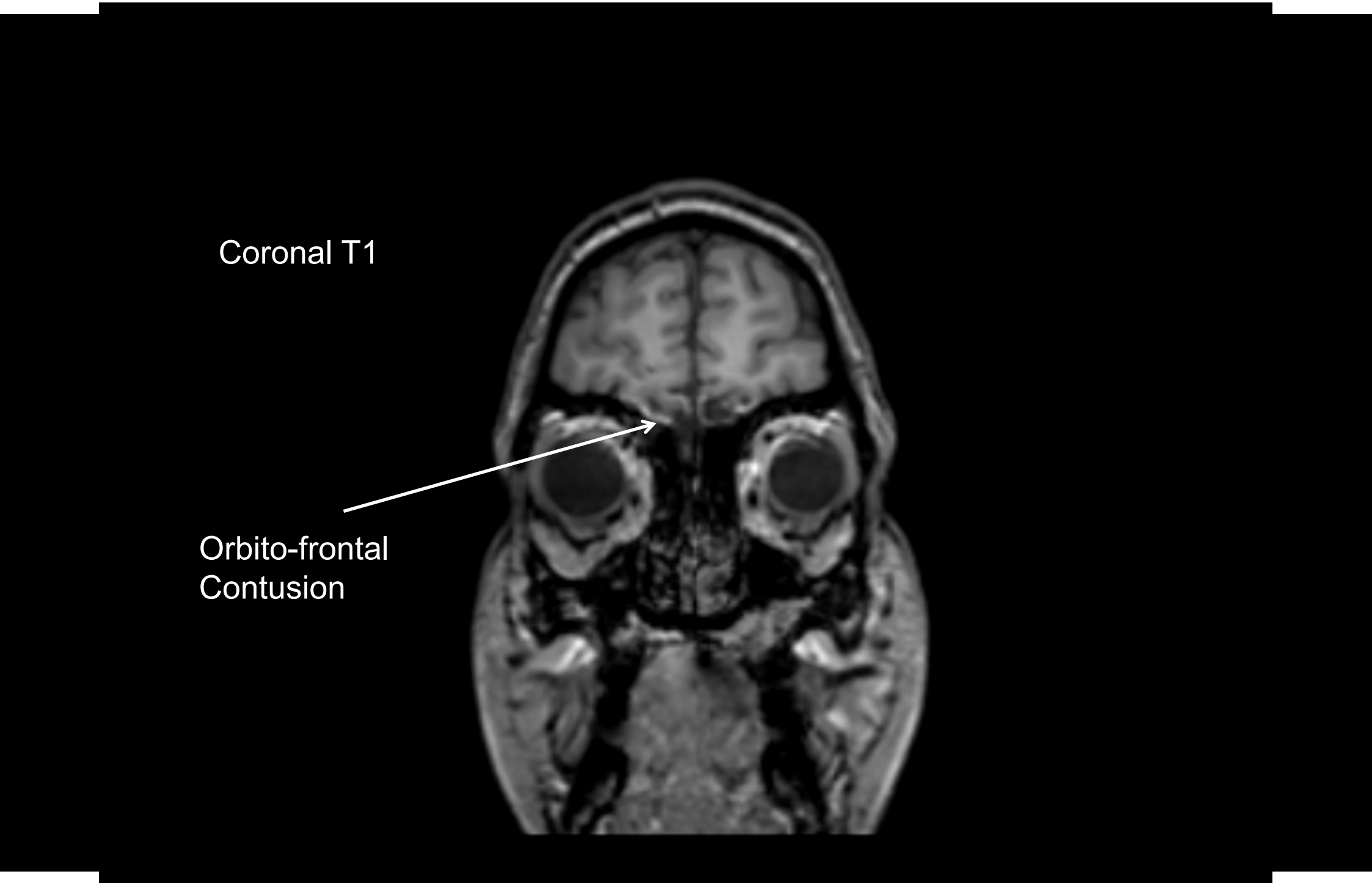


28 year old man brought in from a nightclub after being hit by a bouncer. He fell and hit hit head on the pavement. You are the A+E SHO, what do you do?

He is referred to the TBI follow-up clinic from A+E. You see him as an SPR. He is now complaining of memory impairment, difficulty concentrating and uncontrollable bursts of anger. How do you proceed?

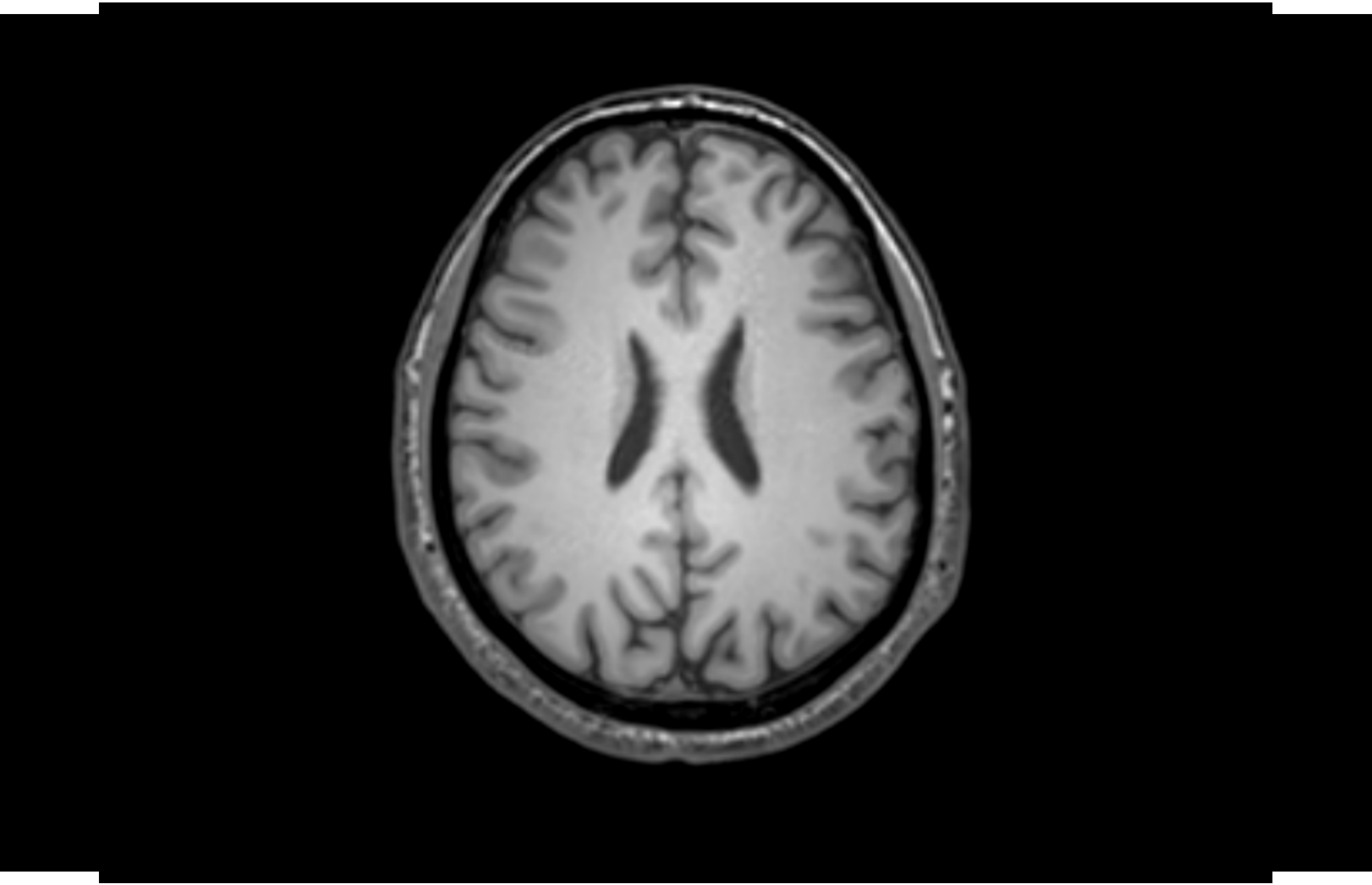
Axial T1

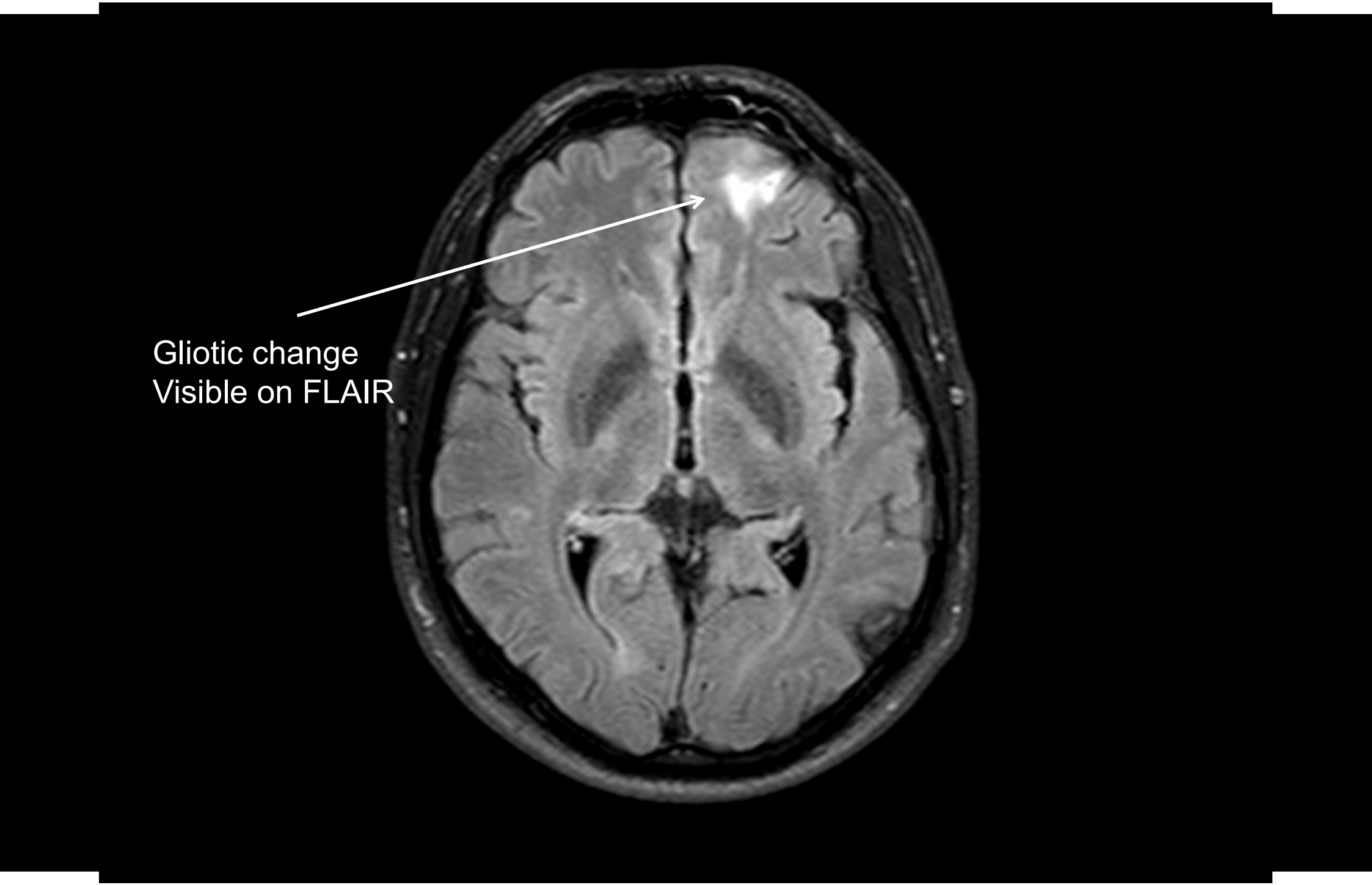



Coronal T1
Orbito-frontal
Contusion

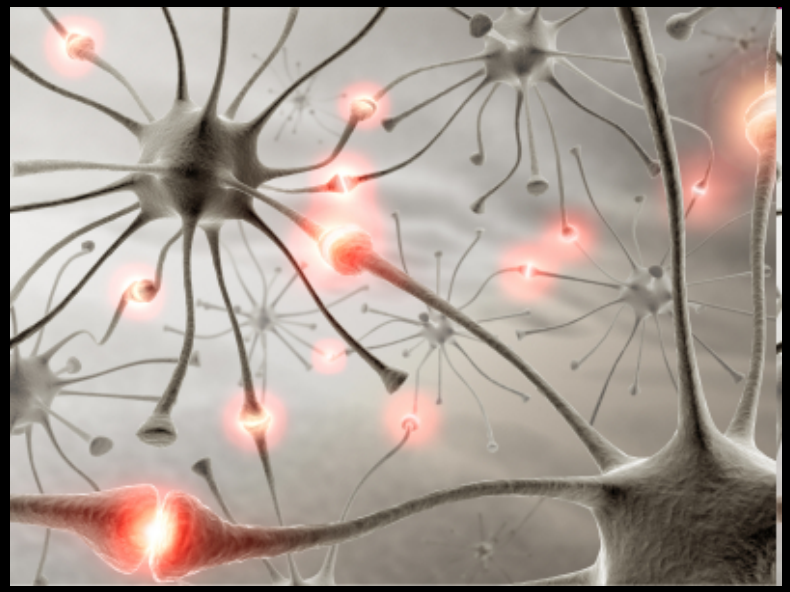

Microbleeds on
Gradient echo imaging

23 year old man hit over the head with a bottle.




Gliotic change
Visible on FLAIR

Epilepsy

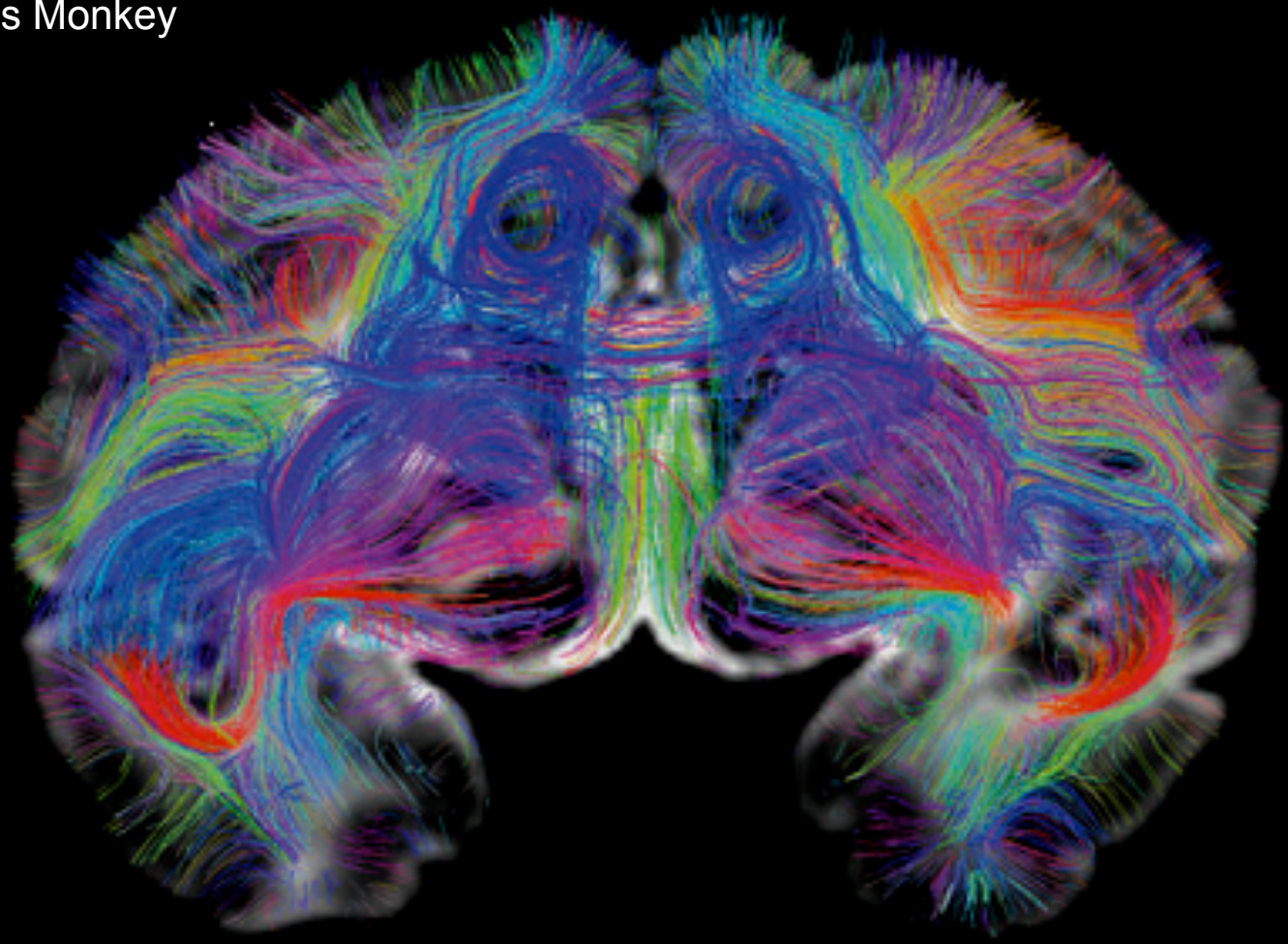


Consider epilepsy with transient neurological abnormalities. This may be altered consciousness but also focal motor/sensory abnormalities

Traumatic brain injury – imaging brain connectivity

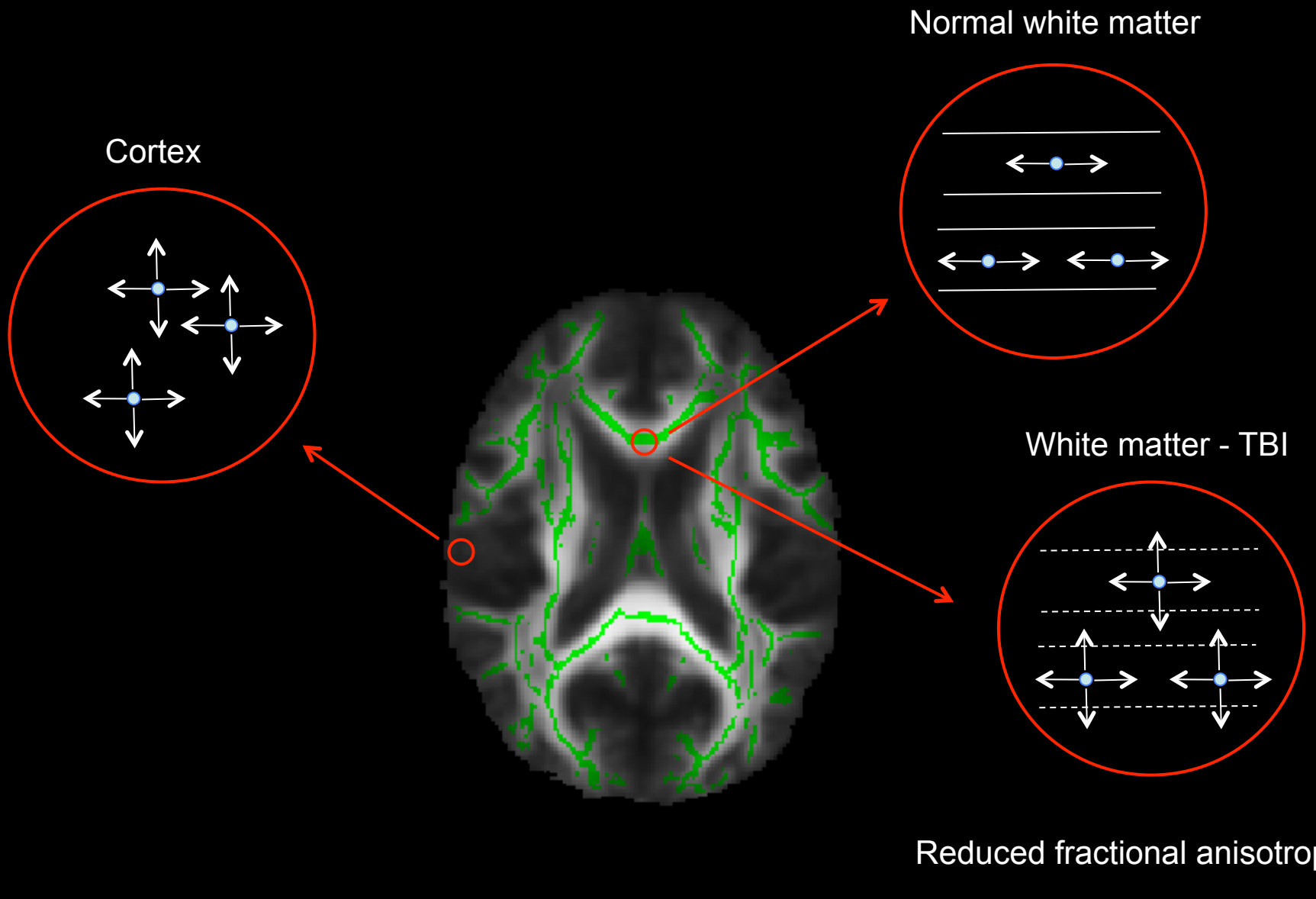
White matter integrity

Rhesus Monkey



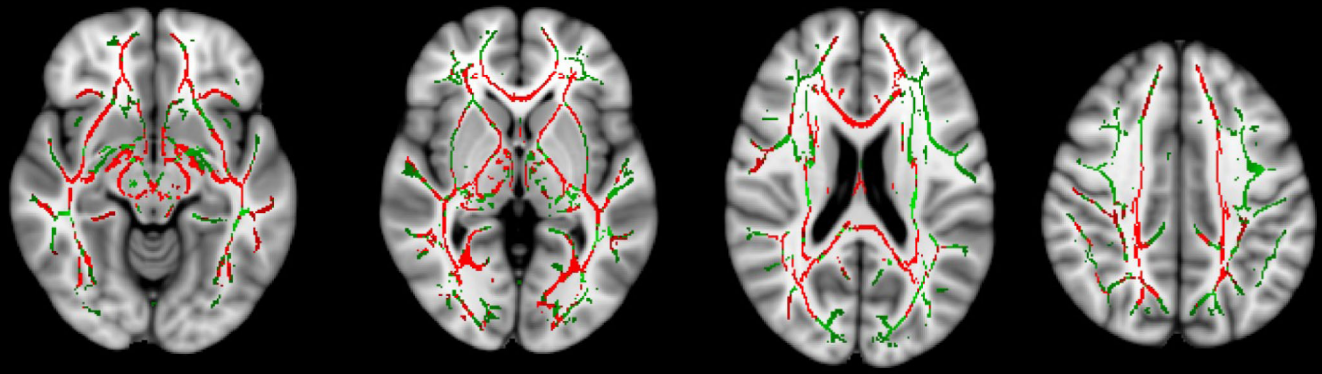
Schmahmann et al Brain ‘07

Diffusion tensor imaging – studying white matter

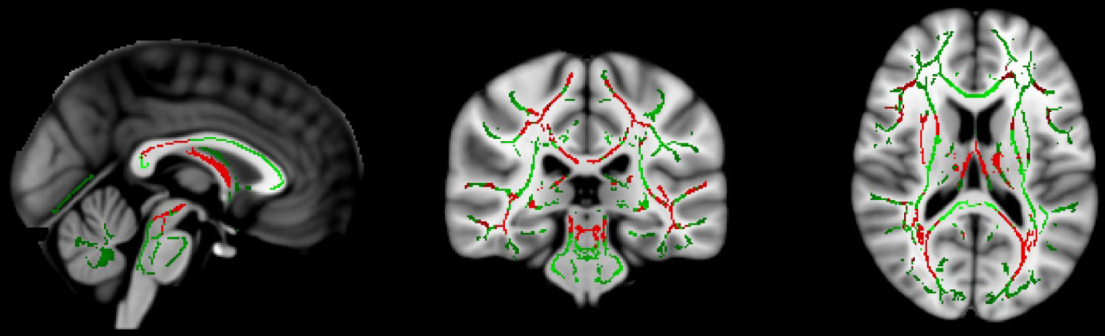


White matter disruption in TBI patients

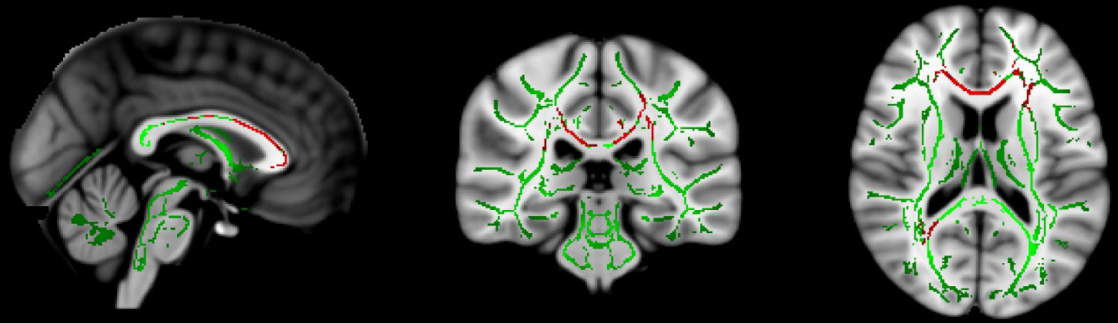
high mean diffusivity



Greater disruption in patients with microbleeds



Disruption is also present in the absence of microbleeds



Predictors of outcome – initial assessment

Initial GCS not a good predictor of outcome

Classification purely based on GCS is problematic:

- 80% mild (14-15), 10% moderate (9-13), 10% severe (3-8)

Cognitive status is important

- Post-traumatic amnesia is a good predictor of long-term outcome

Imaging findings should be included in the assessment

- Lesion volume/location explains a surprisingly small amount of the variance
- Diffuse axonal injury is important: image microbleeds, in future may image brain connectivity routinely