

HEAD INJURY

Dept Neurosurgery

- **INTRODUCTION**
- **PATHOPHYSIOLOGY**
- **CLINICAL CLASSIFICATION**
- **MANAGEMENT**
 - **INVESTIGATIONS**
 - **TREATMENT**

INTRODUCTION

Most head injuries are due to an impact between the head and another object.

Depending on the characteristics of the object (e.g. size, shape and hardness/softness) various resultant injuries appear.

It is not always necessary for the head to come into contact with an object for there to be a head injury - even a significant one for that matter. However, acceleration-deceleration without contact can occur (e.g. "shaken-baby syndrome") which can lead to neuronal damage and subdural haematomas.

DEFINITIONS

✗ CLOSED HEAD INJURY

✚ Head injury without open scalp wounds

✗ OPEN HEAD INJURY

✚ Head injury with open wounds

✗ PENETRATING HEAD INJURY

✚ Head injury with interruption of the dura.

✗ Eg. Knife, axe, Gunshot

PATHOPHYSIOLOGY

There is a primary and a secondary head injury or primary and secondary brain damage.

(A) PRIMARY HEAD INJURY:

A primary head injury is an injury that occurs at the moment of the impact or accident.

The following are results of a primary head injury:

- Concussion

A transient loss of consciousness for usually up to a few minutes, with subsequent recovery and no neurological deficit except amnesia.

- **Brain Contusion**

Area of swelling & diffuse haemorrhages (e.g. a bruise).

Coupe & contre-coupe contusions.

- Diffuse axonal injury**

- Skull Fractures**

Open or closed skull fractures

(B) SECONDARY HEAD INJURY

A secondary head injury or secondary brain damage, is an injury due to something that develops after the impact or accident, and has adverse neurological effects.

Clinically, a patient is initially well (even after a concussion), but later deteriorates.

Causes of Secondary Brain Damage (causes of deterioration)

Intracranial Causes:

- Haematomas - extradural
 - subdural
 - intracerebral**
- Brain oedema or swelling**
- Meningitis/brain infections**
- Obstructive hydrocephalus**

II Extracranial Causes

- Hypoxia: leads to lactic acidosis and cerebral vasodilatation. This congestion leads to raised intracranial pressure.
- Hypercarbia: This also leads to cerebral vasodilatation and raised intracranial pressure.
- Hypotension: this leads to cerebral ischaemia.

Note: $CPP = MAP - ICP$

CPP = Cerebral perfusion pressure

MAP = Mean arterial pressure

ICP = Intracranial pressure

\therefore Avoid \downarrow MAP of \uparrow ICP

CLINICAL CLASSIFICATION OF HEAD INJURY

Glasgow Coma Scale:

E: Eye opening response (1-4)

M: Motor response (1-6) i.e. Best Motor Response

V: Verbal response (1-5)

E:	1	=	No eye opening
	2	=	Opens eyes to pain
	3	=	Opens eyes to sound
	4	=	Spontaneous eye opening

- M: 1 = No motor response
- 2 = Extensor/decerebrate response (rigidity)
- 3 = Flexor/decorticate response (rigidity)
- 4 = Withdrawal response
- 5 = Purposeful response
- 6 = Obeys commands
- V: 1 = No response
- 2 = Makes sounds only (to painful stimulation)
- 3 = Utters words only
- 4 = Says phrases, but confused
- 5 = Well orientated to time, place & person

GCS - used to monitor daily progress, and detect deterioration early.

GCS - also useful in classifying the severity of the head injury (and prognosticating)

e.g. Mild head injury: 13-15

Moderate head injury: 9-12

Severe head injury: 3-8

CT BRAIN



CT BRAIN



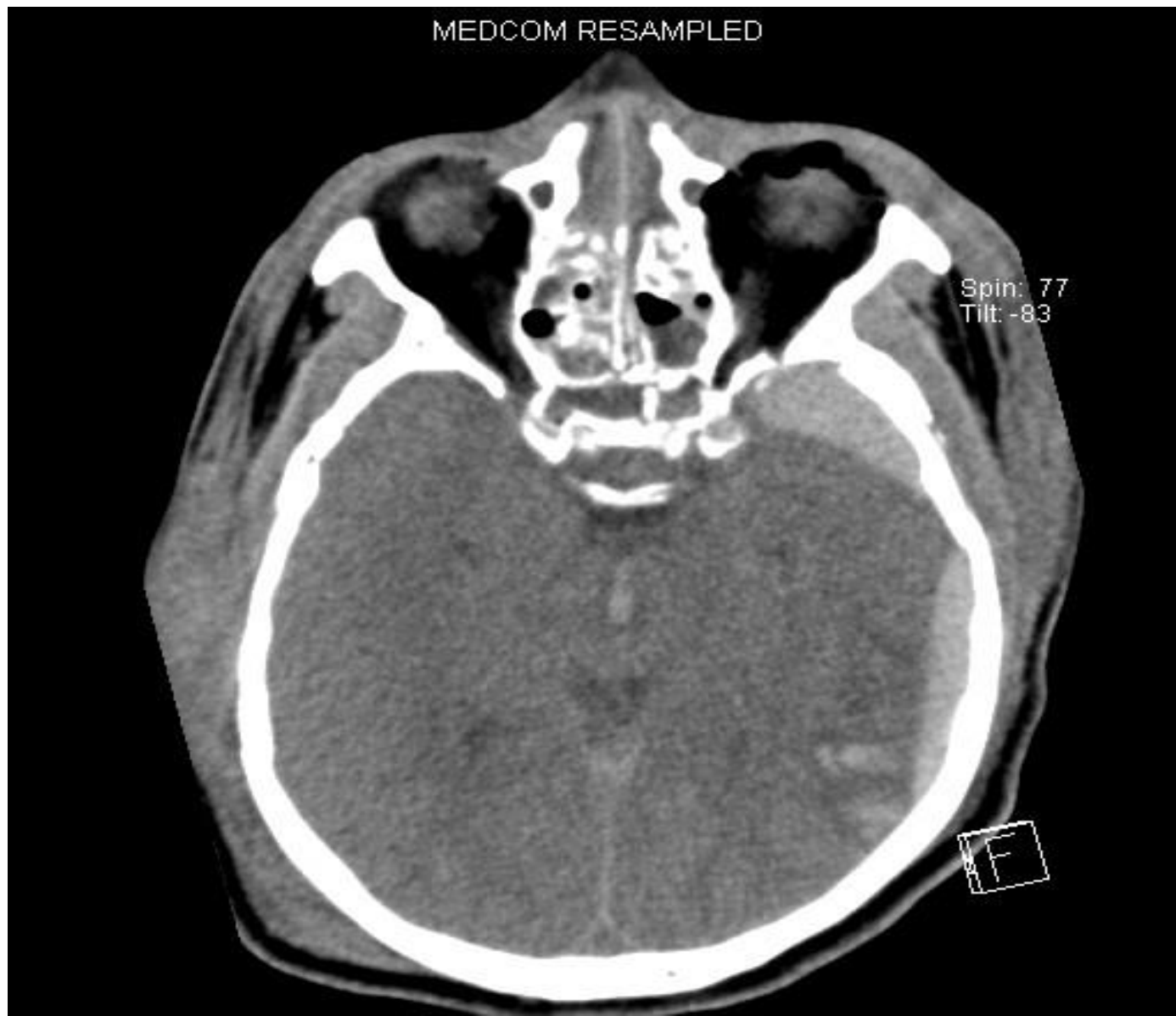
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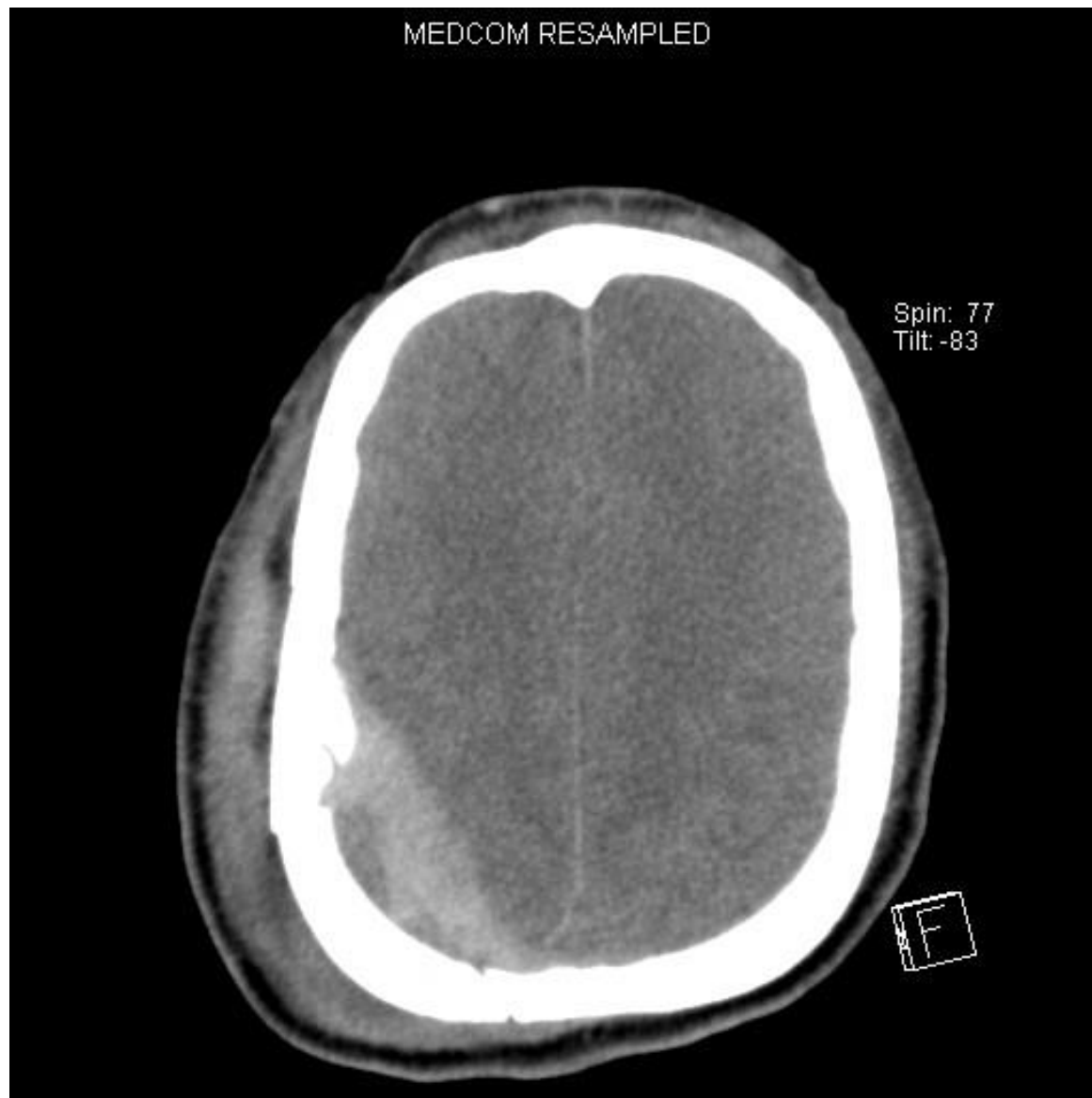
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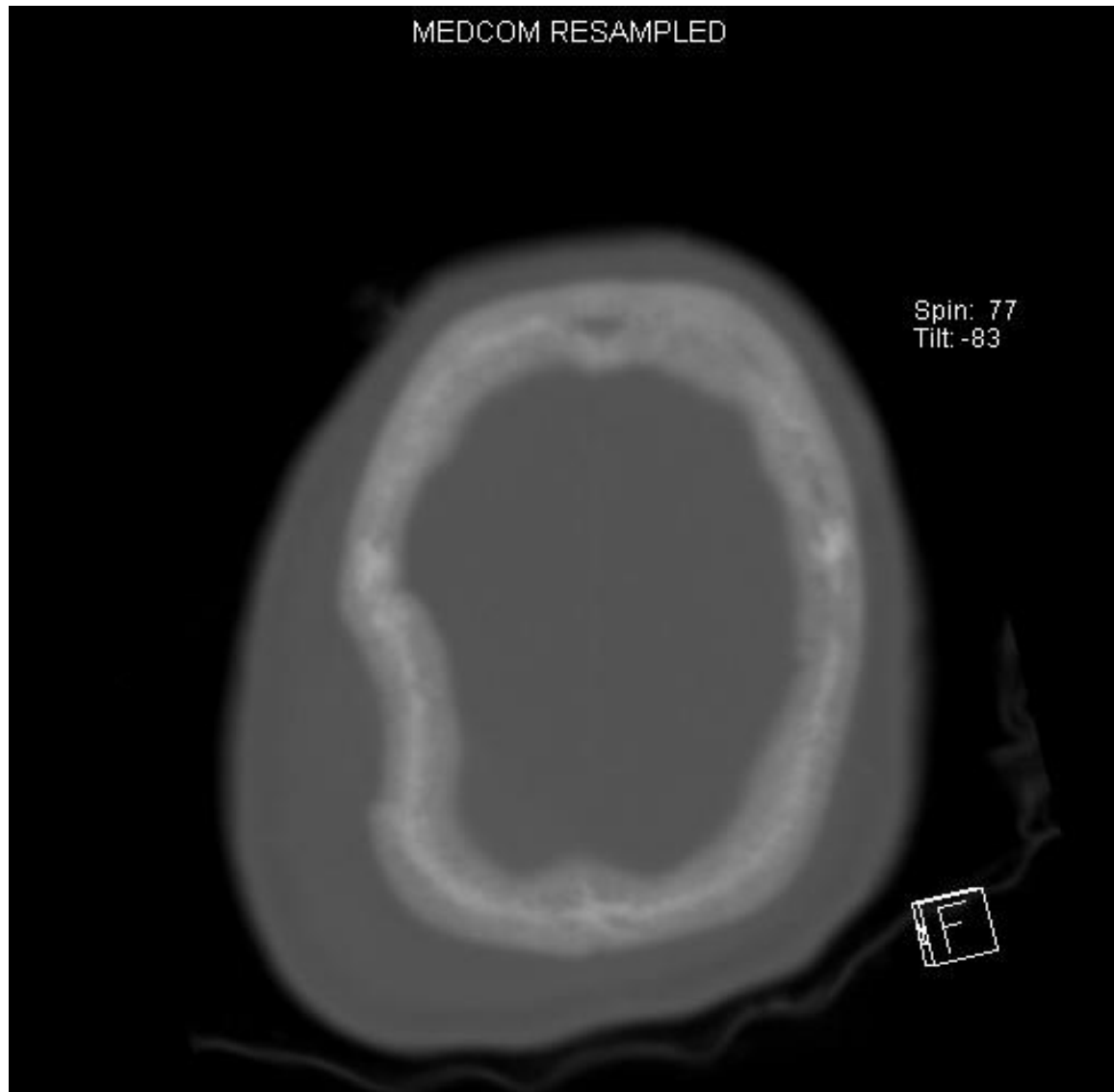
TBI-1



TBI-2



TBI-3





2. What sign is exhibited above?

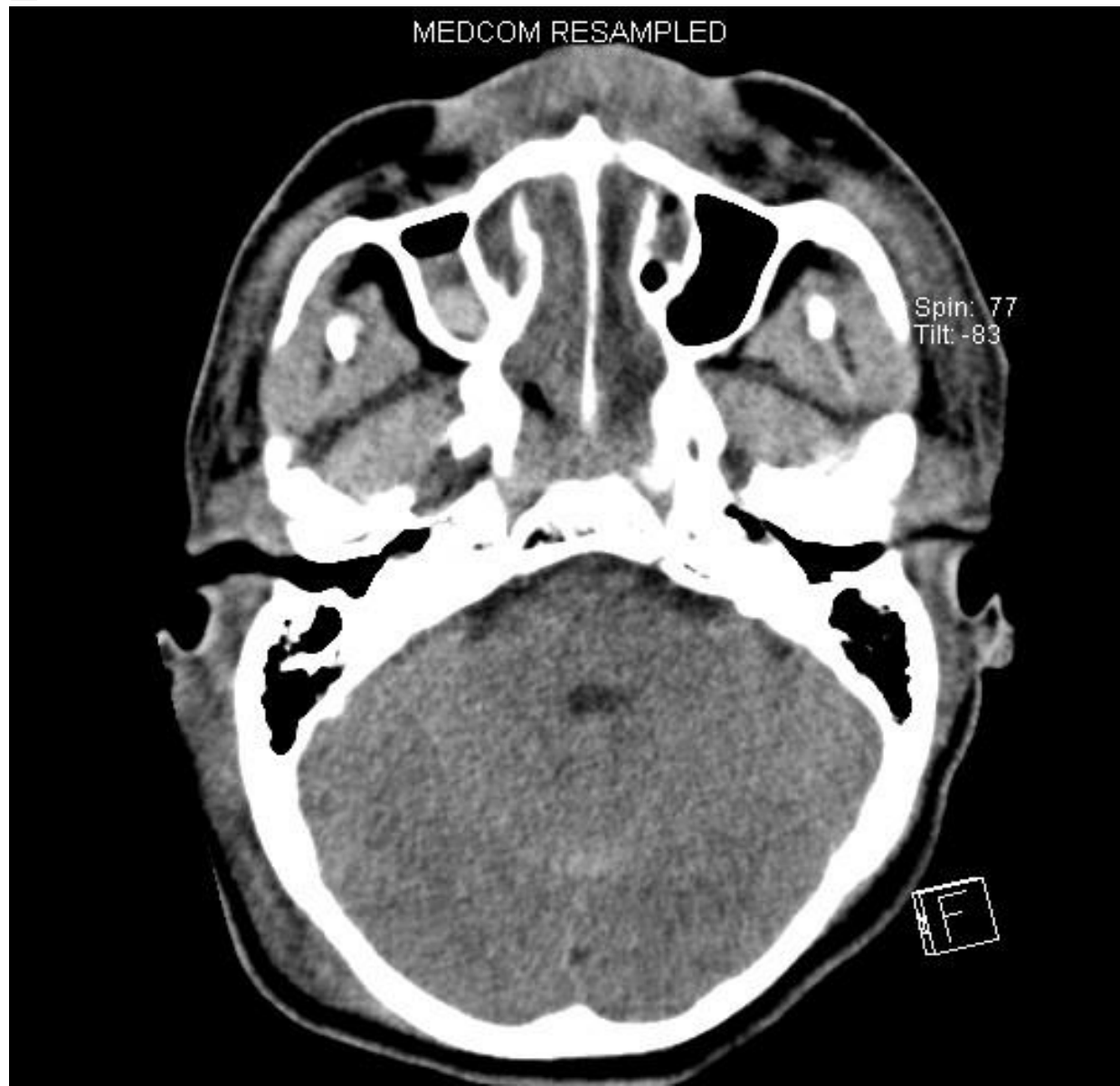
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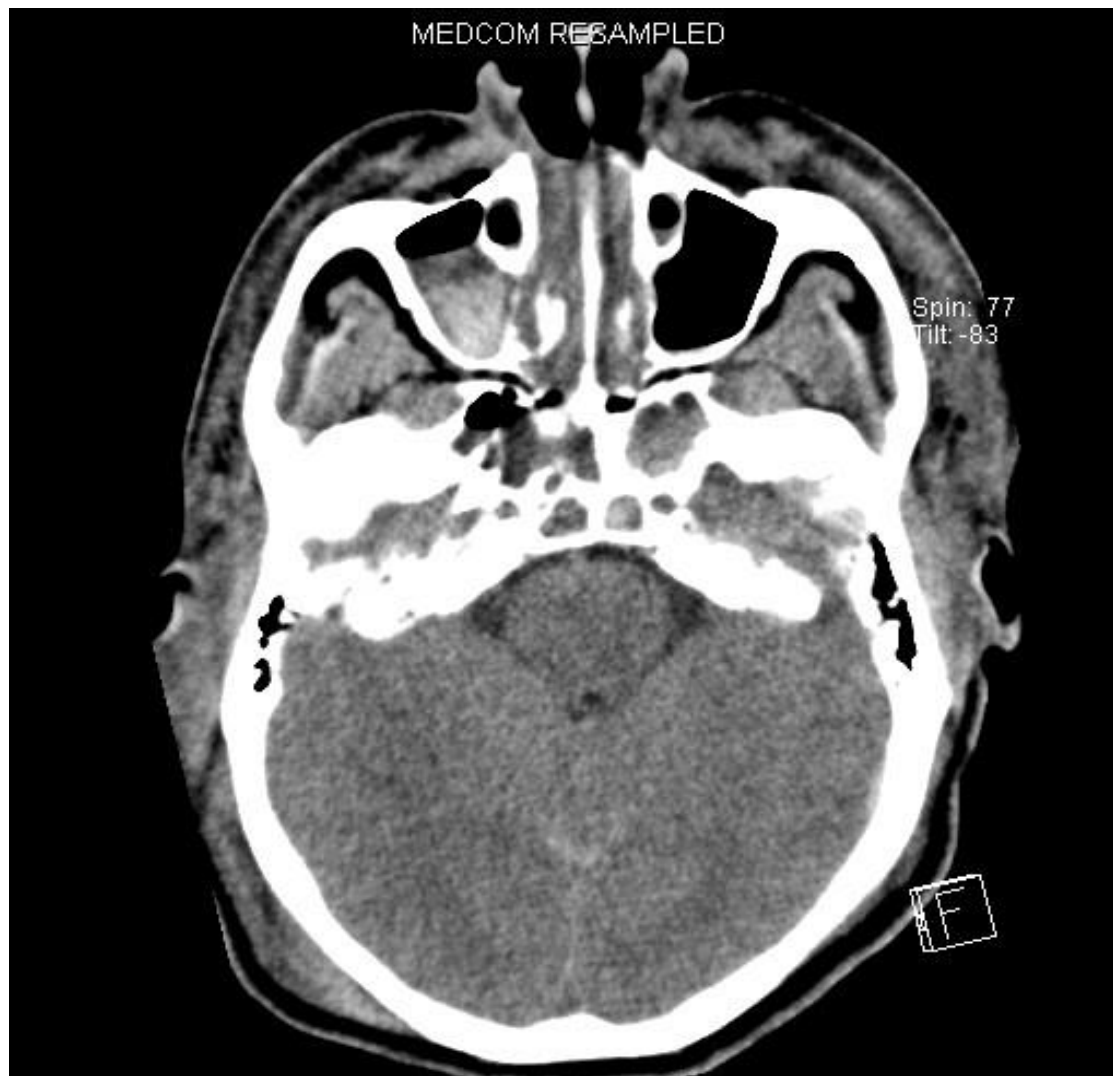
14. What clinical sign is exhibited above?

Watter kliniese teken word hierbo vertoon?

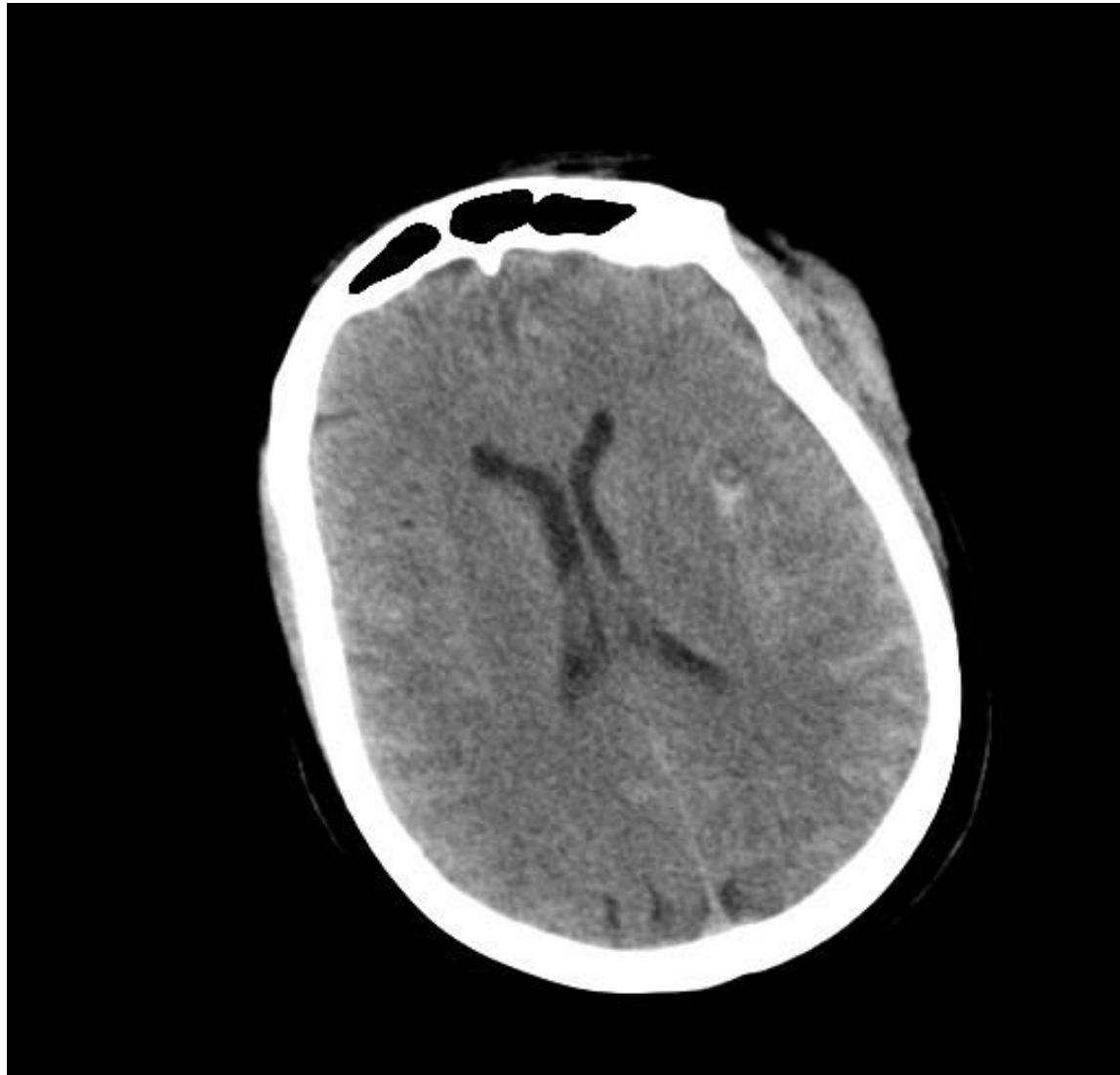
BOSF-1



BOSF-2



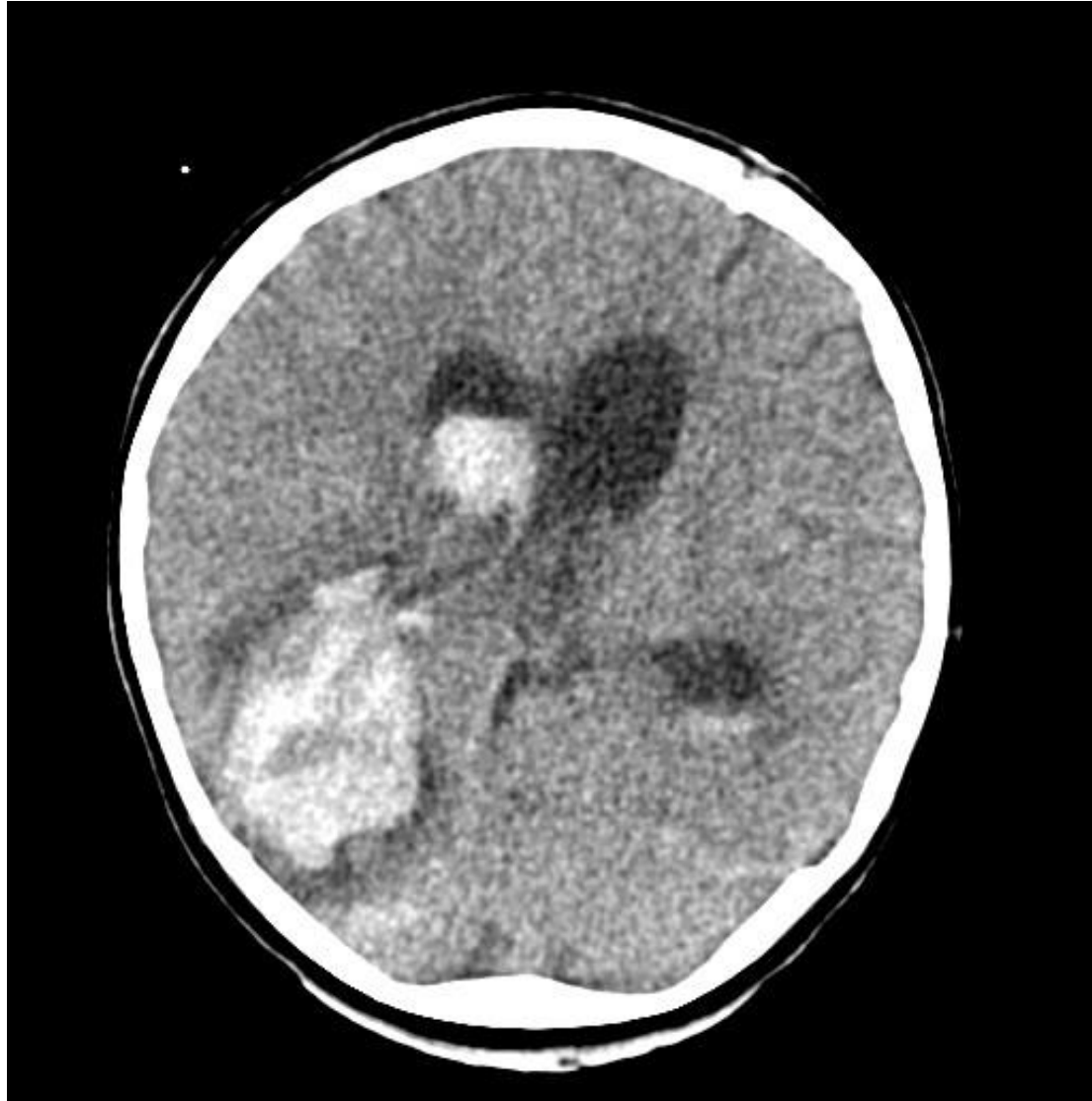
TBI-CNT

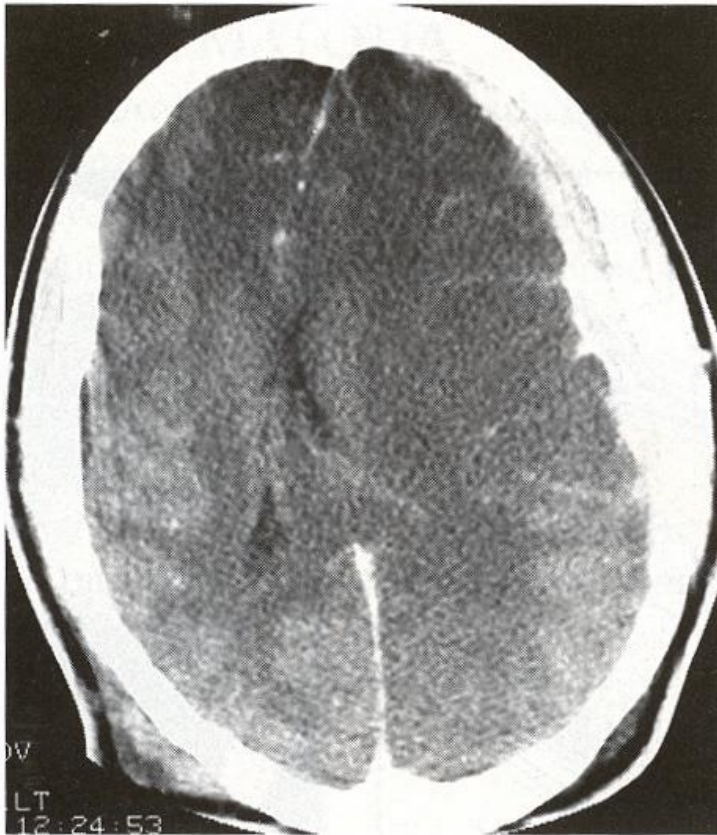


TBI-SAH



TBI-IVH





5. What intracranial bleed is represented on the CT scan?

Watter intrakraniale bloeding word op die RT skandering afgebeeld?



6. What intracranial bleed is represented on the CT scan?

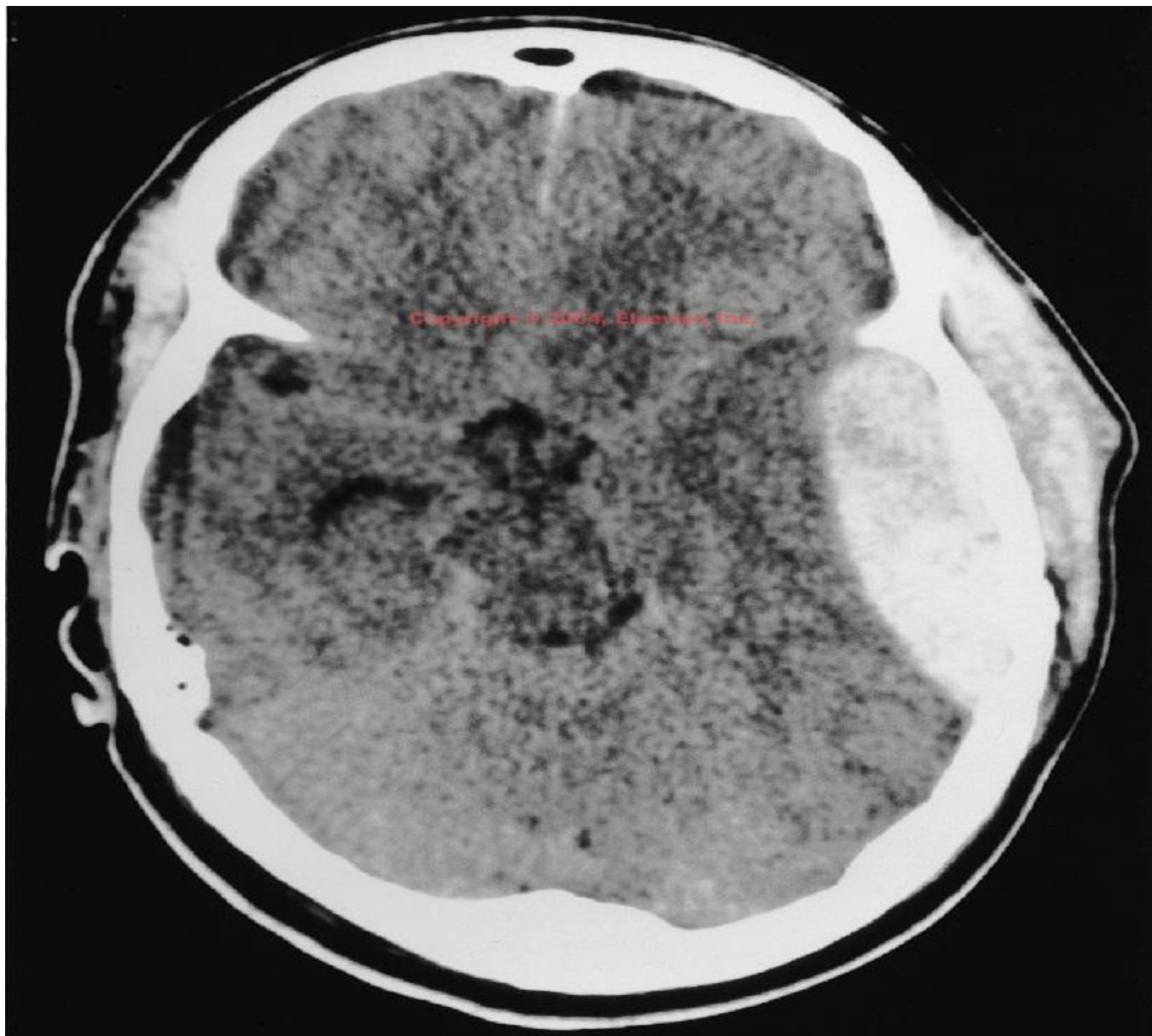
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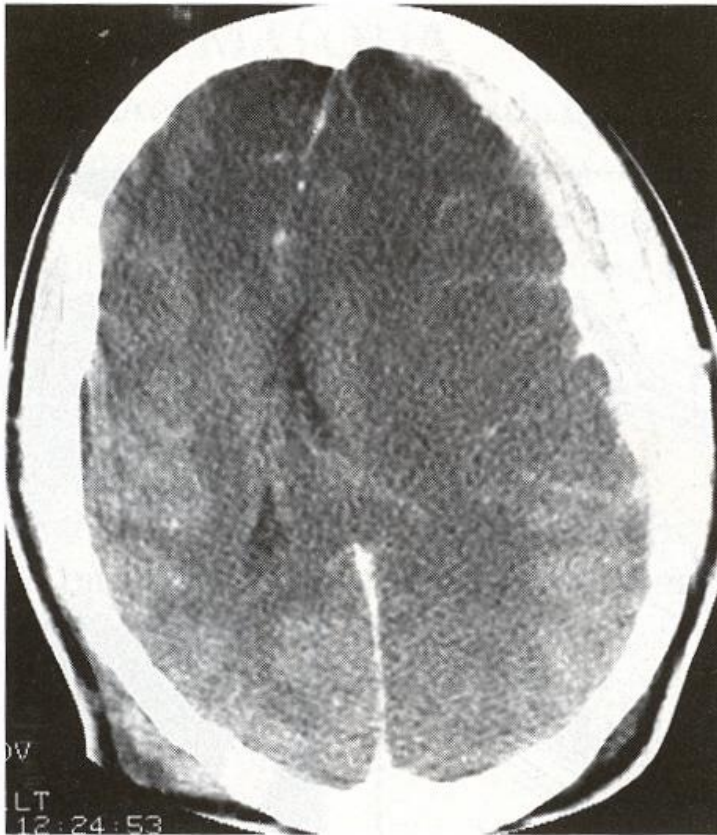
TBI-SDH



TBI-EDH







5. What intracranial bleed is represented on the CT scan?

Watter intrakraniale bloeding word op die RT skandering afgebeeld?

TBI-SKULL FRACTURE









3. What is the diagnosis?

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MANAGEMENT

**This is after resuscitation
(A, B, C....) and stabilisation.**

(A) INVESTIGATIONS

- Skull X-rays: fractures to be elevated \pm antibiotics.**
- CT brain: e.g. intracranial haematomas to be evacuated.**
- MRI scans: most advanced.**

INITIAL MANAGEMENT OF A SEVERE HEAD INJURY — $GCS \leq 8$

✕ RESUSCITATION

+ RESPIRATORY SYSTEM AND C-SPINE PROTECTION

- ✕ Intubate and ventilate to avoid hypoxia and hypercarbia

+ CARDIOVASCULAR SYSTEM

- ✕ IV Fluids – Initially Ringers lactate or Normal saline
- ✕ MAP 80-100mmHg
- ✕ Avoid glucose containing solutions in the first 24hrs unless hypoglycaemic

+ CENTRAL NERVOUS SYSTEM (General Measures to control ICP)

Elevate HOB to 30degrees

Avoid constriction of the jugular veins

Sedation and Analgesia

- ✕ Midazolam (Dormicum)
 - ★ 2 to 4mg/hr
- ✕ Propofol (diprivan)
- ✕ Morphine or fentanyl

Anticonvulsants – phenytoin

- ★ LD: 18mg/kg
- ★ MD 100mg 8hrly

Antibiotic prophylaxis as necessary

Paralysis

- ✕ Pancuronium
- ✕ Vecuronium

ICP monitoring

Indications – See lecture on raised intracranial pressure

Types – Extradural, Subdural, Intracerebral, Intraventricular.

SPECIFIC MEASURES TO REDUCE RAISED INTRACRANIAL PRESSURE

- Ventriculostomy and CSF drainage
- Mannitol and lasix
 - Mannitol is an osmotic diuretic and free radical scavenger
 - Dose: 0.25g/kg 6hrly IV
 - Monitor serum osmolality and maintain $< 320\text{mosm/kg}$ to prevent renal failure.
- Paralysis
- Barbiturates eg. Sodium Pentobarbital
 - May cause hypotension
- Hyperventilation
 - Maintain PCO_2 between 4 and 4.5kpa
- Steroids
 - No longer in use for head injuries.
 - No improvement in outcome
 - Risk of side effects
- Surgical evacuation of the intracranial haematoma
- Decompressive craniectomy

OUTCOME

- PREDICTORS OF OUTCOME
- GLASGOW OUTCOME SCALE
 - 5 Complete recovery
 - 4 Partially disabled
 - 3 Severly disabled
 - 2 Persistent vegetative state
 - 1 Died
- MORTALITY
 - Mild HI 0.4%
 - Moderate HI 4%
 - Severe HI 40%
- MORBIDITY
 - Sequelae or long term complications of a HI

(B) TREATMENT OF SEVERE HEAD INJURY

- Head end elevation ($30^{\circ}\pm$) and head/neck neutral position.
- Intubate for airway protection
- I/V line for fluid therapy, bladder catheter.
- Normalise PaO_2 and PaCO_2
- Mannitol, 1-2g/kg/day for raised intracranial pressure.
- Start feeding on day ≥ 2 n/g feeds.

THANK YOU !!!