

BLUNT HEAD INJURY WITH DIFFUSE BRAIN DAMAGE OR DIFFUSE TRAUMATIC BRAIN INJURY

*'The very language that one uses to discuss certain types of head injury divulges a number of misconceptions that have been inherited from previous generations of physicians.'*¹

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Richard Hewlett obtained a doctorate in comparative neuroanatomy at UCT, then trained in neuropathology at the Regional Neurological Centre, Frenchay Hospital,

Bristol. He is the author, with the late Professor G S Rutherford, of Correlative Surgical Neuropathology and Imaging. His main interests are the morphological basis of brain MRI and neurotuberculosis.

This article concentrates on those aspects of brain injury that result from blunt force applied to a mobile head. Of particular importance is the concept of acceleration and deceleration and immediate disturbance of consciousness.

AETIO-PATHOGENESIS

Forces involved in closed head injury, described as numerous and complex,² are principally the result of dynamic mechanical loading (Fig. 1).

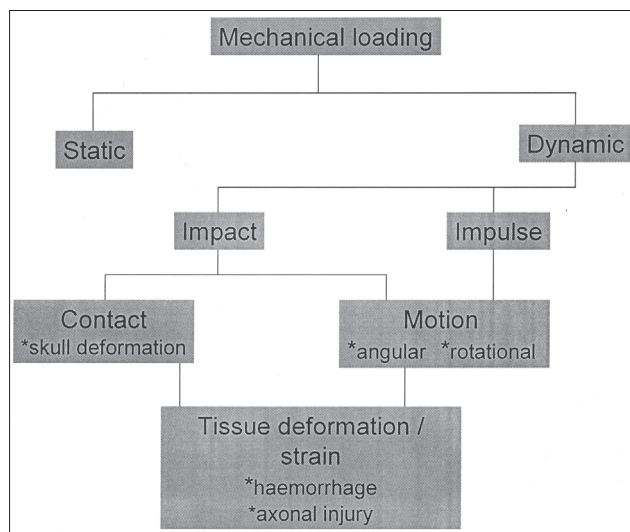


Fig 1. Aetio-pathogenesis of head injury (after Graham et al.²).

The usual type of impact to the resting, movable head, e.g. application of a blunt object, results in focal injury at the site of skull deformation, a lesion still widely referred to as coup contusion (percussion-concussion of Denny-Brown). Combinations of inertia and impact resulting from instantaneous deceleration of the moving head (variations of Denny-Brown's acceleration-concussion) cause a complex of contusions and shear deformation, with associated tissue disruption or tears. The Penn Machine,³ a device that combines inertial acceleration with rotation of the head (Fig. 2), was used experimentally to induce concussion and axonal injury in a consistent association. This important contribution to 20th century neuropathology demonstrated the quantitative relationship between traumatic coma and

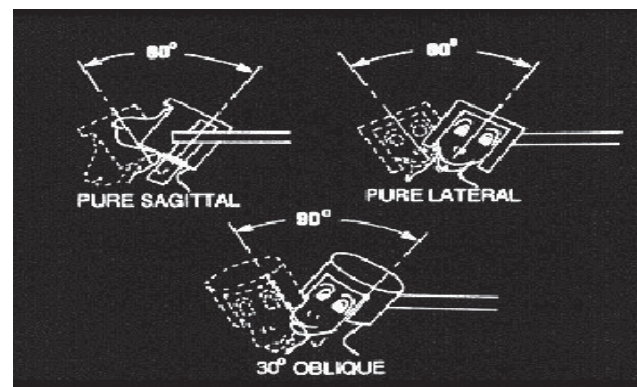


Fig. 2. Penn machine (after Gennarelli et al.³). The piston arm and helmet produce acceleration, deceleration and rotation, with consistent graded axonal injury.

Blunt head injury is associated with a variety of haemorrhagic lesions, which when imaged show a combination of topography and morphology that is so consistent that it can provide a reasonably certain cause for the anatomical diagnosis.

axonal injury. The Penn machine has also shown that, besides magnitude, the critical factors in the generation of diffuse axonal injury (DAI) are the rate of onset and the duration of brain acceleration, as in cases where the impact is cushioned by deformable or energy-absorbing surfaces.

PATHOMORPHOLOGY

Macroscopic haemorrhagic stigmata

Blunt head injury is associated with a variety of haemorrhagic lesions, whose topography and morphology often combine so consistently as to provide reasonably secure aetio-pathogenetic inferences to the anatomic diagnosis. Although focal vascular disruption is common to all types of blunt head injury, conventional terminology distinguishes between impingement and tensile mechanisms, referred to as contusion injury and tissue tear haemorrhage (TTH) respectively.

The morphological characteristics of contusion injuries are dependent on how they were caused and where they are anatomically. Lesions over the lateral convexity of the brain caused by focal inbending of the skull represent the site of impact (coup), and are typically composed of petechiae confined to the cortical gyral crown, often without pial disruption (Fig. 3 A-E).

Depending on factors such as the nature of the applied force and how long after injury they are examined, the microhaemorrhages may coalesce to form rounded haematomas up to a centimetre in diameter.

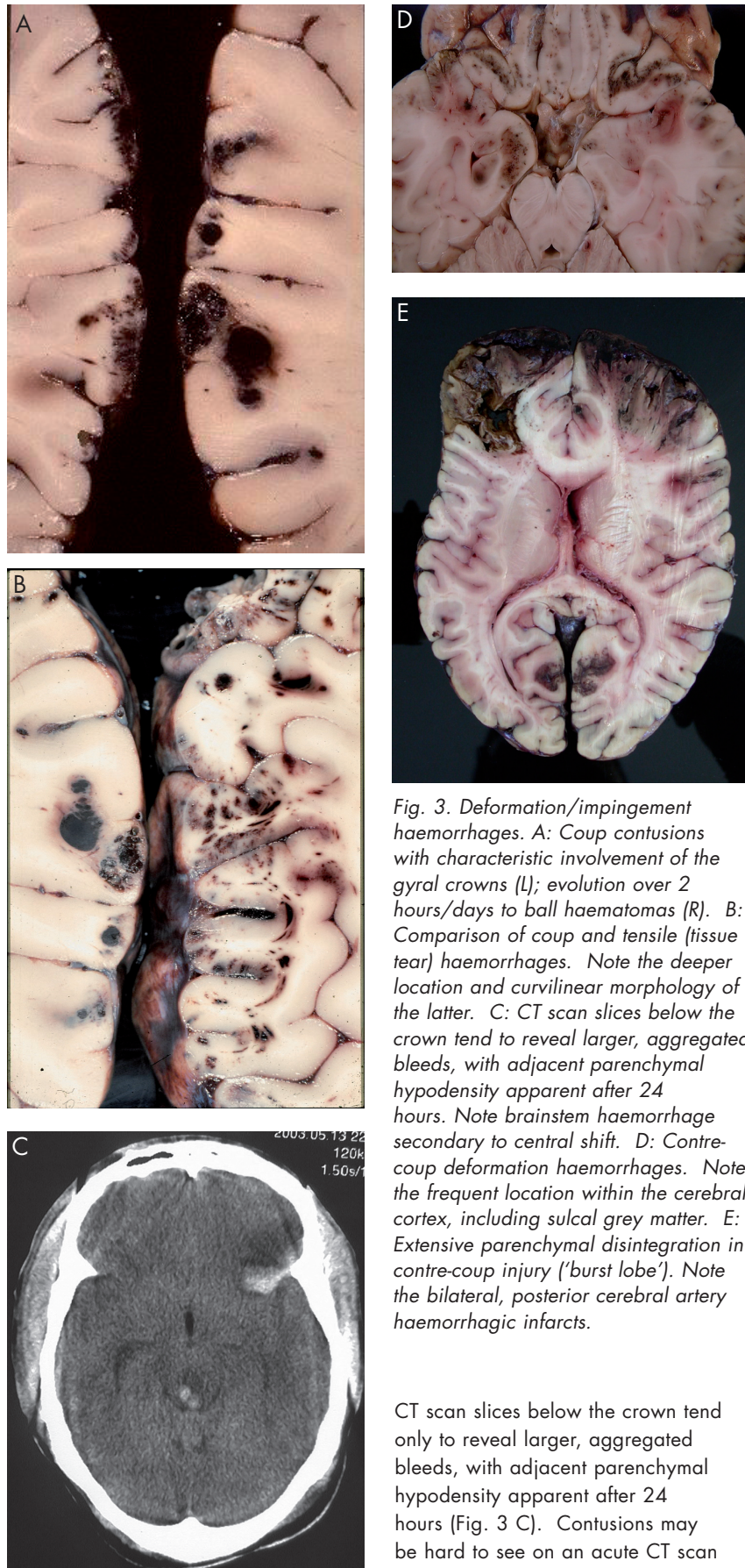


Fig. 3. Deformation/impingement haemorrhages. A: Coup contusions with characteristic involvement of the gyral crowns (L); evolution over 2 hours/days to ball haematomas (R). B: Comparison of coup and tensile (tissue tear) haemorrhages. Note the deeper location and curvilinear morphology of the latter. C: CT scan slices below the crown tend to reveal larger, aggregated bleeds, with adjacent parenchymal hypodensity apparent after 24 hours. Note brainstem haemorrhage secondary to central shift. D: Contre-coup deformation haemorrhages. Note the frequent location within the cerebral cortex, including sulcal grey matter. E: Extensive parenchymal disintegration in contre-coup injury ('burst lobe'). Note the bilateral, posterior cerebral artery haemorrhagic infarcts.

CT scan slices below the crown tend only to reveal larger, aggregated bleeds, with adjacent parenchymal hypodensity apparent after 24 hours (Fig. 3 C). Contusions may be hard to see on an acute CT scan performed soon after injury and are frequently missed. Focal superficial

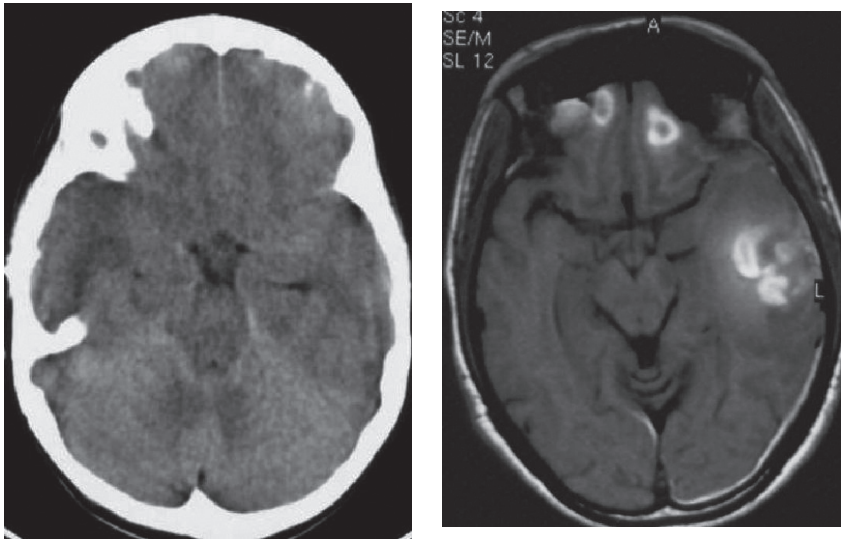


Fig. 4. Contre-coup contusions. CT (L) and T1-weighted MR scan (R): Peripherally hyperintense lesions representing blood, together with characteristic topography, are much more readily identified on MR.

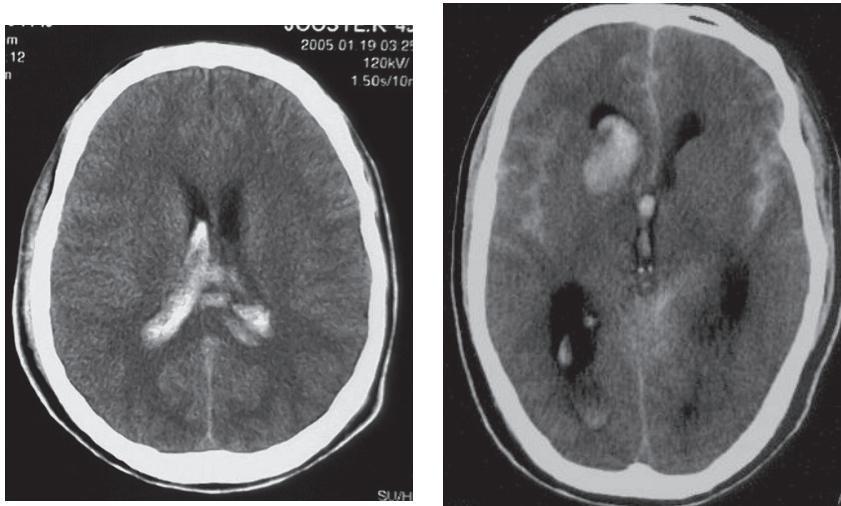


Fig. 5. Acute callosal and ganglionic tissue tear haemorrhages: Non-contrast CT. Note characteristic ventricular clot (L), and ventricular dilatation and subarachnoid blood (R). CSF hyperdensity will disappear over a few days as clot lyses.

haemorrhages occurring away from, or even opposite, the site of impact (contre-coup contusions), which are caused by acceleration-deceleration injury, represent a combination of impingement and shear deformation of the brain on bony margins and protruberances of the skull base, or the dural free margins, and petechiae are frequently located within the cerebral cortex (Fig. 3 B, D). These frontotemporal, basal cortical lesions may also be associated with laceration, sometimes with extensive meningoparenchymal disruption (referred to as a burst lobe) (Fig. 3 E). Polar and perisylvian lesions, because

of their grossly haemorrhagic nature, are much more readily identified on CT, and are even more characteristic on MRI where topography is so accurately defined (Fig. 4).

TTHs are now defined as topographically and morphologically distinctive focal lesions. These are invariably associated with the type of brain deformation caused by acceleration-deceleration, concussive, or immediate impact loss of consciousness and diffuse axonal injury.

Within 24 hours of injury a CT scan can depict most types of TTH accurately, and this mode of imaging now provides the principal collateral for the diagnosis of DAI (Fig. 5), including this diagnosis by default, i.e. in the presence of a normal scan.

Diffuse vascular injury

This term is used in the context of widespread, deep parenchymal haemorrhages, usually accompanied by brain swelling despite, invariably, short survival. We have observed an unusual form of diffuse white matter haemorrhages displaying perivascular morphology (Fig. 6).

Diffuse axonal injury

Properly referred to as diffuse, traumatic axonal injury (DTAI), this entity is defined at light microscopy level as the presence of eosinophilic, silver-positive axonal swellings or spheroids, elliptical in the long axis

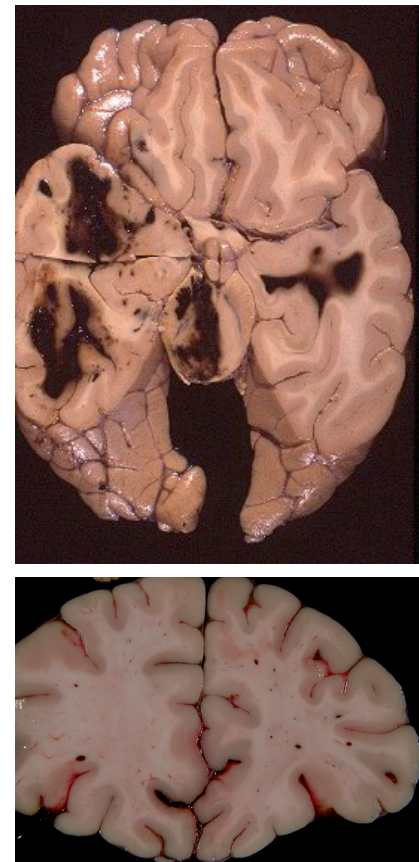


Fig. 6. Diffuse vascular injury: both confluent and focal morphological types appear to be associated with very short survival, although still usually accompanied by brain swelling.

of the nerve fibre, and displaying characteristic topography involving the deep frontal lobes, corpus callosum, putamen-internal capsule, caudal mid-brain decussation and cerebellar white matter. Although widespread (therefore multifocal or diffuse), injury concentration is usually asymmetrical, with features determined by the direction of head and brain movement.

Reaction to axonal damage (usually with microhaemorrhage) starts within a day or two, and depending the degree of severity continues over a period of 3 - 8 weeks.

Grading traumatic axonal injury

Sparse, dispersed, axonal spheroid formation (seen on microscopic preparations) is known to occur in minor head injury with a normal Glasgow Coma Scale (GCS); in these cases pathognomonic TTHs are said not to be observed (although there may be other types of focal haemorrhage), and the brain may even be macroscopically normal. Increasingly severe DTAI is accompanied by microscopic and macroscopic haemorrhage, especially callosal TTH, and when combined with dorsolateral mid-brain haemorrhage, such patients are invariably unconscious from impact.⁴ We have observed fornix-septal rupture in a patient who presented with a lucid interval, suggesting that this type of central haemorrhage may be acquired with purely sagittal injury. Interestingly, delayed onset of coma on the MRI basis of DTAI has also been described.

DTAI in infantile, non-accidental injury

Axonal spheroid formation appears more difficult to identify in this category of patients, and amyloid precursor protein (APP) or beta A4 immunostaining is said to be essential.⁵ In infancy, the time course of this reaction is uncertain.

Neuronal injury

Most cases of diffuse traumatic brain injury (DTBI) display numbers of both dark and eosinophilic

nerve cells, whose significance is hard to assess. Both global and regional hypoperfusion may occur as specific early events in severe traumatic brain injury, but there may be selective involvement of the hippocampus independent of hypoxia. Neurodegenerative disease is now accepted as a complication of DTBI, being linked to the ApoE phenotype,⁶ and the issue of post-traumatic Alzheimer-type dementia is likely to become increasingly important. In a case studied at Tygerberg Academic Hospital, the early onset of dementia in a young, post-traumatic coma patient was remarkable because of the complete absence of neuronal alterations in routine preparations.

Brain swelling

Initially, DTAI was identified in a cohort of traumatic coma patients whose brains did not exhibit swelling and it has been proposed that primary brain swelling without focal haemorrhage is an epiphenomenon. The early onset of brain swelling after acceleration-deceleration injury (with or without a lucid interval), not associated with global hypoxaemia-ischaemia, is usually seen in young patients, especially children and infants, and its initial pathogenesis is uncertain, possibly representing cytotoxic swelling. Therefore, in patients with varying grades of pure DTAI, it can apparently occur as an independent process and is the mechanism of death.

Injury regression

At Tygerberg Academic Hospital in the Western Cape, a significant number of cases of head injury come to postmortem weeks or even months after the event. Although pneumonia and metabolic derangement supervene, most of these patients have remained in a state of altered consciousness, many have undergone surgery, and diffuse brain injury is the primary cause of death. The causal relationship of DTAI to the clinical condition of protracted coma or the vegetative state has to be distinguished from the effects of brain swelling and multifocal pressure-related infarcts

located in the basal cortex, deep grey matter and brainstem.

THE FORENSIC NEUROPATHOLOGICAL EXAMINATION

The autopsy should only be commenced after examination of the clinical details and images; useful and essential CT details include soft-tissue injury, fracture/penetration, lucency of the air sinuses, parenchymal haemorrhage and air, state of the ventricles and presence or absence of brain swelling and shift. Posterior dissection (Fig. 7) is essential for the proper identification of tonsillar displacement, and large, basal, subarachnoid haemorrhages should be washed away to disclose the circle in detail.

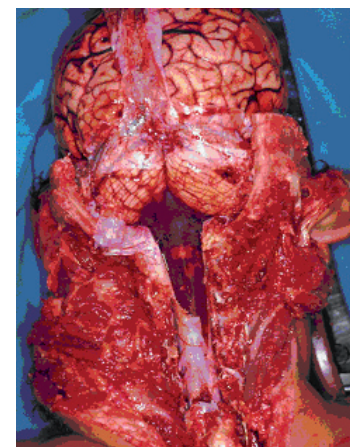


Fig. 7. Posterior dissection showing removal of occipital bone and posterior spinal arches.

The brain should be suspended from the dura immediately after removal, and not left lying on the table.

Similarly, the images and notes must be available at the braincut, which must be performed in the axial plane as a routine, without prior removal of the stem. A routine number of blocks are required in cases of DTAI and hypoxic injury, and the anatomical diagnosis should be entered, even provisionally, on the fixed brain report. Cases of interest (especially protracted coma) should be retained for discussion with the neurosurgeons and neuroradiologists, who will want to see the folder.

At any teaching hospital, a significant number of CNS cases coming to forensic autopsy are non-traumatic, especially spontaneous intracranial haemorrhage, infarct, infection and seizures. Occult hydrocephalus is really only discovered in the context of sudden unexpected death, together with an increasing number of HIV-associated lesions. These cases now constitute an important – even unique – source of neuropathological material available to academic departments.

Acknowledgement

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References available on request.

IN A NUTSHELL

Mechanisms are complex and variable but include acceleration-deceleration and rotation of the head.

The clinical state of concussion/coma is correlative.

The macroscopic stigmata (imaging/autopsy) are haemorrhagic and evanescent, but brain may be macroscopically normal.

The superficial macroscopic lesions are contusions; deep lesions are tissue tears.

The microscopic lesions are microhaemorrhages, axonal spheroids and macrophage aggregates; lesions are evanescent.

The distribution of microlesions is variable, with favoured sites, but should be widespread or 'diffuse' in diffuse brain injury.

Diffuse microlesions can occur independently of brain swelling and vice versa.

Axonal spheroids are not specific; ischaemia is the commonest cause of these lesions.

CT/MR is diagnostic, with gradient echo an essential technique, especially for follow-up/medico-legal purposes.

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