2.2

CT IN HEAD INJURIES

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INTRODUCTION

Trauma constitutes the most frequent cause of death and permanent disability in the western world (3, 20). For example, each year in the United States of America alone, some 2 million incidents of head injury occur (9), representing the most frequent cause of death among children and young adults (3, 20). Annual mortality from head injuries is estimated to be 25 per 100,000 population and is four times higher in males than females (2, 4). In the United States, 20-50% of head injuries are the result of road traffic accidents, 20-40% of firearm injuries, and assault or falls in the remainder (2, 4). Falls are particularly dangerous in children under ten years of age and the elderly, representing approximately 75% of all head injuries in the preschool population (2, 4).

Head injuries can be classified as open or closed, depending on whether or not the dura mater is intact (11, 12, 26). Closed cranial traumas are the more frequent of the two types and produce violent accelerations of the brain tissue, which are responsible for tissue contusion and vascular laceration at both the site of the direct trauma as well as on the opposite side. Among other injuries, skull fractures, extracerebral intracranial haemorrhages and parenchymal lesions are typically observed alone or in varying combinations. Open head trauma includes lesions of the skull such as depressed fractures associated with interruptions of the dura mater, and intracranial lesions secondary to the action of penetrating foreign bodies or dislocated calvarial fracture fragments.

Brain lesions can also be divided into primary and secondary categories according to how closely they are linked to the traumatic event (3, 20, 22, 26). Primary lesions include skull fractures, extraaxial haemorrhages (e.g., epidural and subdural haematomas and subarachnoid haemorrhages) and intraaxial lesions (e.g., diffuse axonal injury [DAI]), cortical and deep grey matter contusion [bland or haemorrhagic], and intraventricular haemorrhage) (Tab. 2.2). Secondary lesions are caused by pathological processes that arise from the brain's response to the trauma, as a complication of the primary traumatic brain lesions or as neurological involvement associated with systemic extracranial trauma (Tab. 2.3). In general, the secondary types of trauma are more clinically devastating than are the primary brain injuries. They are generally caused by compression of the brain, cranial nerves and blood vessels against the bony cranial structures and the non-expandable dural margins. Secondary brain injuries include cerebral herniations, diffuse cerebral oedema, infarction and secondary haemorrhages (3, 20, 22, 26).

Tab. 2.2 - Primary traumatic cranioencephalic lesions.

1) Fractures
2) Intraaxial lesions
a) diffuse axonal injury (damage)
b) cortical contusion
c) intraparenchymal haematoma
d) intraventricular haemorrhage
e) lesions of the deep grey matter
f) brainstem lesions
3) Extraaxial lesions
a) epidural haematoma
b) subdural haematoma
c) subarachnoid haematoma
d) subdural hygroma
e) vascular lesions

Tab. 2.3 - Secondary traumatic cranioencephalic lesions

1) Cerebral herniation
2) Diffuse cerebral oedema
3) Posttraumatic ischaemia
4) Posttraumatic infarction

In the study of acute head injuries, CT currently represents the diagnostic methodology of choice (3, 7, 20, 22, 28). Its ability in distinguishing between minor differences in tissue density, its high spatial definition, capacity to demonstrate even very subtle or critically positioned (e.g., base of the skull, temporal bone) fractures, fast scanning times and compatibility with life support devices all contribute to making CT the most suitable technique in acute phase head injury evaluation (3, 7, 20, 22, 29).

TECHNIQUES

Despite the speed with which CT must be performed in emergency conditions, head injuries warrant the observance of certain technical and methodological parameters that ensure diagnostic efficiency and the reproducibility of the results in the event of serial evaluations (14, 22, 29).

Caution is required when positioning the patient on the CT bed, taking care that any tubes and electrode cables are sufficiently long to enable suitable scan table indexing during the scan. Particular care must be observed in positioning the patient's head in the supine position: the chin must be extended in order to prevent breathing difficulties. And, to prevent a potential cervical spinal cord injury resulting from a suspected or occult unstable spinal column fracture-dislocation, immobilization of the head and cervical spine should be undertaken.

The CT examination begins with a projection digital scanogram that includes the cervical spine. This serves to define the scanning limits for an evaluation of skull vertex fractures, gross subluxations and fractures of the cervical spinal column, gross soft tissue alterations and for the recognition of any radioopaque foreign bodies. The CT scan gantry should be oriented along the orbitomeatal plane, or alternatively along the so-called horizontal "German plane" parallel to the hard palate, and should be directed in such a way as to avoid if possible sectioning through any metal foreign bodies (e.g., dental fillings, etc.) that might be likely to cause beam-hardening artefacts (5, 7, 22, 29). The examination continues with contiguous 5 mm-thick axial slices through the structures of the posterior fossa and then concludes with a stacked series of 10 mm-thick axial slices through the cranial vertex to cover the supratentorial structures (5, 7, 22, 29).

For small lesions or in order to visualize fractures at the base of the skull, the acquisition of 2-3 mm sections is recommended. Coronal scans may be required at a later point beyond the emergency period in order to identify or exclude fractures at the base of the skull, the bony orbit and the petrous bone, and for visualization of parenchymal or bony lesions adjacent to the vertex. It should also be pointed out that coronal scans require optimal patient cooperation and should only be performed when it is absolutely certain that there are no fractures/instability of the cervical spine or at the atlanto-occipital junction.

When an otorrhagia or acute paralysis of the facial nerve suggests a fracture of the temporal

bone, the examination must be completed with a detailed study of the petrous bone using thin slices (1-2 mm), a reduced field of view (FOV) and utilizing bone reconstruction algorithms (14, 24, 29).

Generally speaking the examination should be filmed/reviewed using window and level values that permit an optimal viewing of the cerebral parenchyma (e.g., 100 H.U. window and 40-50 H.U. level), and those that also enable visualization of the bony structures (e.g., 2000-3000 H.U. window and 500-700 H.U. level). In certain cases, especially for the visualization of thin epidural or subdural haemorrhages, it is advisable to film the examination with window values equal to 200-350 H.U. and level values equal to 40-80 H.U. in order to distinguish the mildly hyperdense blood collection from the adjacent bony hyperdensity (9, 15, 22, 28).

The intravenous administration of iodine contrast medium does not significantly improve the diagnostic sensitivity of the examination in cases of cranial trauma; in fact, it can conceal intracranial haemorrhage, causing minor blood collections to be overlooked. The administration of contrast agents is also usually avoided as it can produce renal injury or even potentially worsen brain function in patients with traumatic cerebral lesions. However, the utilization of contrast media can be useful in certain limited cases when better definition of the dural venous sinuses is desired, better visualization of chronic isodense subdural haematomas is necessary, and above all, in order to document simultaneous underlying pathology such as neoplasia or AVM's. Of course, intravascular contrast media are required for performing CT angiography in cases where there is a suspicion of posttraumatic intracranial vascular pathology (e.g., vascular lacerations, arteriovenous fistulae and posttraumatic pseudo-aneurysms). In all of these cases, MR may be more sensitive and is sometimes preferable to CT.

Modern CT appliances with spiral (helical) scanning capability greatly reduce the time required for scanning, these units acquiring the entire scan volume in just a few seconds; when acquired in sufficiently thin sections (e.g., 1-2 mm) these scans can then subsequently be reconstructed in different spatial planes (1, 10, 22, 24). Helical scans are most useful in cases where scanning time must be reduced to a minimum, such as in children or in non-cooperative patients. It can also be very helpful when there is a suspicion of lesions of the orbit or the facial skeleton in which it is possible to quickly obtain high resolution two- or three-dimensional reconstructions (1, 10, 24).

It is useful, however, to stress that spiral CT examinations can sometimes produce images that simulate non-existent pathology, such as minor subdural haematomas, in part due to the partial volume averaging effects along the z-axis of scanning. This kind of artefact appears principally along sloping surfaces such as the skull-brain interface over the frontoparietal convexity (1, 10, 22).

Another type of artefact associated with spiral CT that can simulate a chronic subdural haematoma is the so-called "stairstep" artefact that produces images with hypodense peripheral areas, especially at the skull vertex (1, 10, 22). Such problems can be partly or completely resolved by reducing the collimation, the reconstruction interval, the scan bed's index speed and/or the tube rotation speed (22). Recent research shows that traditional, non-helical CT is superior to spiral CT with regard to the signal/noise ratio, in visualizing the interface between white and grey matter and in observing low contrast, small, complex structures (e.g., the internal capsule) (1).

In summary, the use of spiral CT in cases of cranial trauma should be restricted to those cases in which scanning time must be as brief as possible, instances in which a three-dimensional study of the bony structures is required or when CT angiography of intracranial circulation is necessary.

SEMEIOTICS

PRIMARY TRAUMATIC LESIONS

Skull fractures

Skull fractures associated with cranial trauma are common and are discovered in approx-

imately 60% of all cases (20). The presence of a fracture is not necessarily significant with regard to the clinical severity of the trauma, nor does the absence of a fracture exclude the possibility of severe brain damage; records show that in 25-35% of patients with severe trauma and underlying brain injury, no fracture is present. For this reason, many practitioners feel that x-rays aimed at diagnosing skull fractures are superfluous with reference to patient care (16, 20, 25). In addition to the parietal, frontal and occipital areas, skull fractures can affect the vertex or base of the skull. And, depending on the intensity of the force applied, they can be classified as linear fractures usually caused by lesser forces (Fig. 2.1), and comminuted and depressed fractures typically caused by greater forces (Fig. 2.2) (16, 17, 22, 25, 29). Fractures of the skull base can be caused by superficial or penetrating trauma, the caudal extension of a fracture of the calvaria, or alternatively, the upward transmission of traumatic forces along the vertebral column. Although CT is sensitive in detecting these types of lesions, thin, linear fractures and those oblique/parallel to the plane of section can go unnoticed (16, 17, 25). However, the practical importance of fracture recognition is secondary, as efforts are principally concentrated on the search for associated intracranial lesions. Linear fractures are often associated with epidural and subdural haematomas, whereas depressed fractures are more frequently associated with focal parenchymal lesions adjacent to the bony lesions (20).

In more severe cases, such as in open head injuries that are often consequences of knife or firearm wounds, fractures may be associated with a laceration of the dura mater and a penetration of bone fragments, air or foreign bodies into the cranial cavity (3, 20, 27). In the study of such open head injuries, CT permits the accurate localization of foreign bodies or bone fragments in large part due to their relative high density (Fig. 2.3). CT also provides a detailed picture of the spatial characteristics of depressed fractures (Fig. 2.4) and at the same time, documents any related cerebral lesions.

Lesions of the dura mater at the base of the skull, associated with fractures of the adjacent bone, can be complicated by a CSF fistula formation, with consequent oto- and/or rhinorrhea (Fig. 2.5). In such cases, the site of the fistula can be suggested by means of a detailed study of the bone using high resolution CT. This study should be performed employing axial slices to study fractures of the posterior wall of the frontal sinuses and the petrous bone, and coronal acquisitions to study ethmoid fractures and also to supplement the evaluation of petrous fractures.

In addition, fractures of the paranasal sinuses, the mastoid cells or the middle ear may result in the introduction of air into the subarachnoid spaces or the cerebral ventricles (pneumocephalus) (8, 27). These conditions can occasionally behave as space-occupying lesions (e.g., pneumatocele formation) when a valve mechanism is formed at the dural laceration resulting in a considerable increase in the volume of intracranial air, and therefore will require treatment (Fig. 2.6).

Intraaxial lesions

Diffuse axonal injury

Diffuse axonal injury (DAI) is one of the most common primary brain lesions associated with severe trauma and accounts for approximately 48% of all traumatic intraaxial injuries (3). DAI typically results from abrupt accelerations or decelerations or when violent rotational forces obtain causing a differential inertia between grey and white matter, between the cerebral hemispheric connecting structures or within the diencephalon and brainstem with consequential axonal stretching, twisting and tearing (3, 20). Clinically, DAI is characterized by a loss of consciousness beginning immediately at the time of the trauma which can evolve into a coma that is often irreversible (3, 13, 22). DAI is the most severe of all primary brain lesions, being ahead in degree of severity of that of cortical contusions, intraparenchymal haematomas and extraaxial haemorrhages. Pathoanatomically,

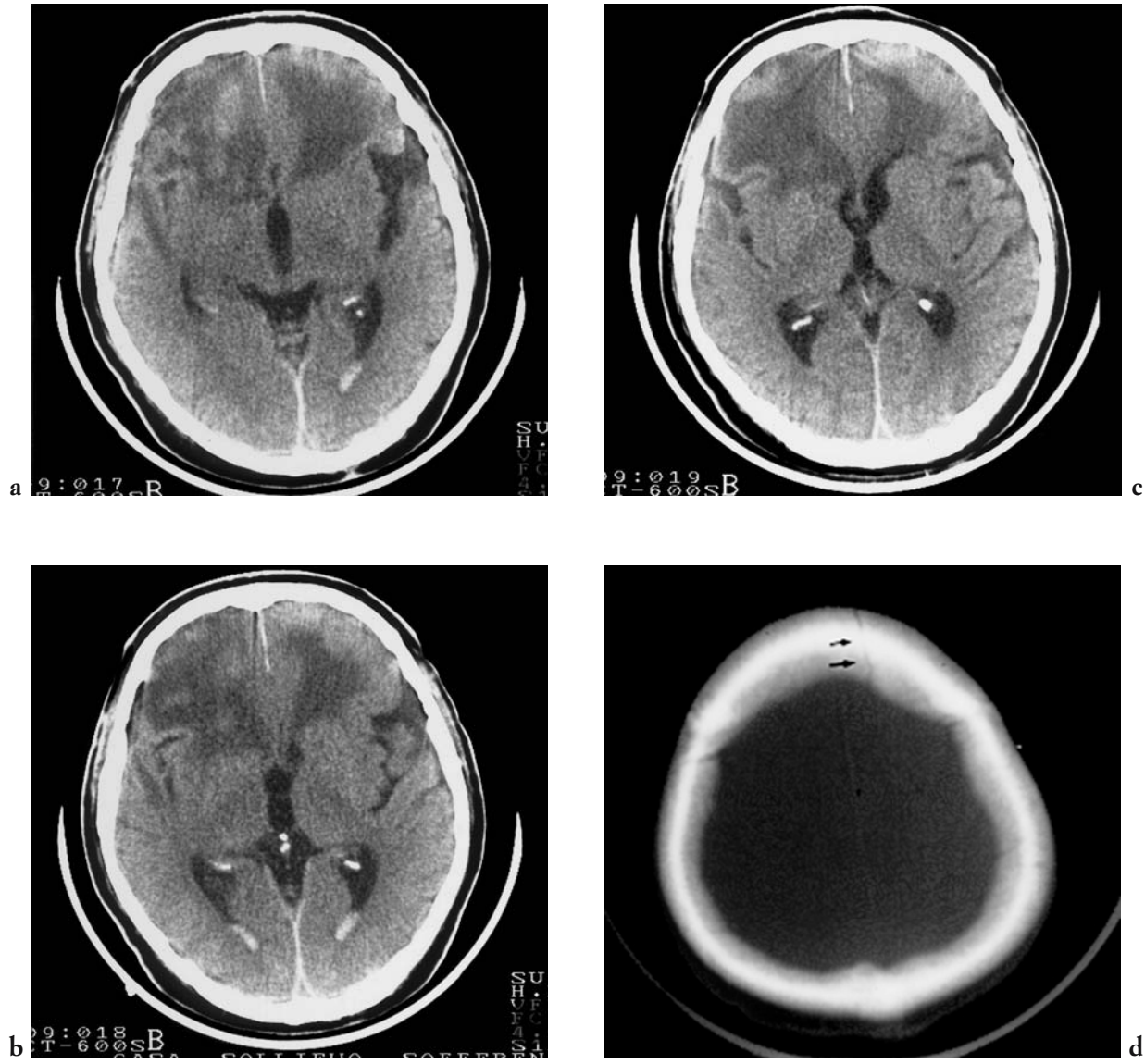


Fig. 2.1 - (**a-d**) Recent head injury. Irregular hypodense cortical-subcortical areas are observed bilaterally in the frontal lobes; hyperdense haemorrhagic material is noted in the occipital horns of the lateral ventricles (**a-b**). Also seen is a frontal bone fracture line (**c**).

ball-shaped deformations of the damaged axons can be observed; the lesions are not usually haemorrhagic, although in 20% of cases they can be associated with small haemorrhages resulting from the rupture of penetrating arterial vessels (3). DAI usually occurs in one of three typical positions: the cerebral hemispheric white matter, the corpus callosum and the dorsolateral aspect of the midbrain. More precisely, in approximately two thirds of cases the lesions are located at the subcortical grey-white matter junction in the frontotemporal region, 20% are located in the corpus callosum (especially the body and the splenium), and less frequently the alterations affect the dorsolateral side of the brainstem tegmen, the internal capsule, the thalamus and the caudate nucleus (20). During the early phases of a traumatic injury, the CT picture is often normal despite the fact that the clinical status is that of a seriously compromised

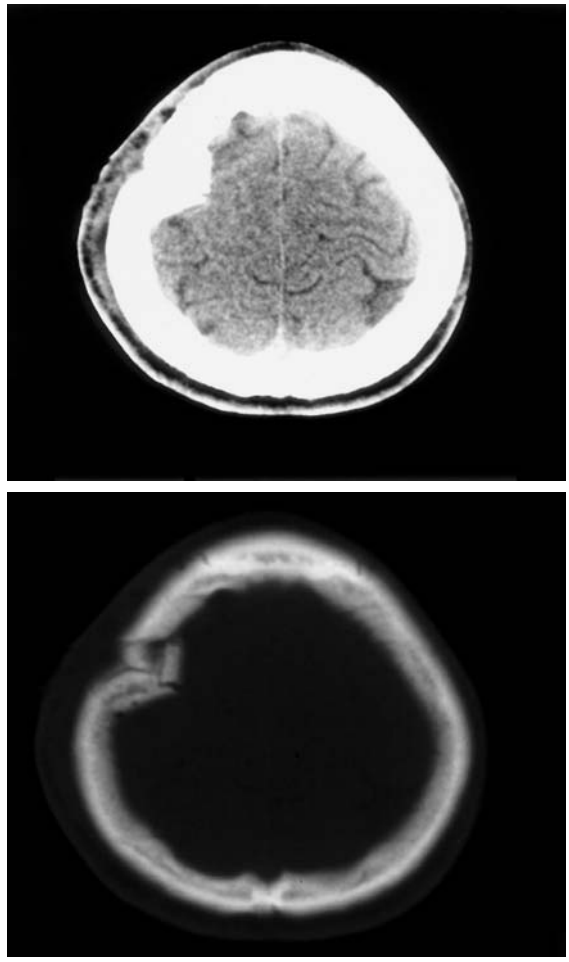


Fig. 2.2 - (**a, b**) Demonstrated is a depressed fracture of the right parietal bone.

patient; research indicates that alterations can only be documented by imaging in 20-50% of patients with DAI (3, 20, 24). The alterations that can be documented by CT consist of small haemorrhages and hypodense lesions that affect the corpus callosum and the grey-white matter junction of the cerebral hemispheres (Fig. 2.7). Small haemorrhages also occur in the internal capsule, in the periventricular grey matter and in the dorsolateral quadrants of the brainstem, the latter being a somewhat vulnerable area in patients subjected to angular accelerations-decelerations. Although MRI also underestimates the real extent of axonal damage in DAI relative to the pathoanatomical findings, it is currently the most sensitive imaging technique available (3, 20, 21).

Cortical contusion

Cortical contusion is the second most frequent form of parenchymal lesion following DAI and represents approximately 45% of all intraaxial primary traumatic injuries (3, 20). Unlike DAI, cortical contusion is less frequently associated with loss of consciousness and occurs following a violent traumatic shaking injury of the cerebral tissue (3, 13). It can occur secondary to linear accelerations of the cerebrum that produce the typical coup and countercoup parenchymal contusions, or alternatively after angular accelerations that produce cortical contusions resulting from collisions of the brain against the inner surface of the skull. As the grey matter is far more vascularized than the white matter, cortical contusions tend to be more frequently haemorrhagic than do those lesions associated with DAI (7, 29). Pathoanatomically, these contusions are characterized by isolated or multiple foci of focal or linear microhaemorrhages of the cortical gyri. The lesions may be associated with focal oedema resulting from alterations of the permeability of the regional capillaries and the walls of involved glial cells. Petechial lesions, which are often combined with overlying bony skull fractures, tend to expand into more widespread haemorrhagic foci that often manifest 24-48 hours after the trauma (3, 20, 29). Because the lesions are caused by the impact of the cerebral parenchyma against the surrounding bony structures, most contusions are located in characteristic locations. In approximately half of all cases, the lesions are situated within the temporal lobes (especially in the poles), over the lower surface of the frontal lobes and in the cortex surrounding the Sylvian fissure (Fig. 2.8).

The CT appearance of cortical contusions varies in part with the manner in which the lesions evolve over time. At the onset, the CT picture can be negative because the lesions are initially isodense, and therefore are impossible to distinguish from the surrounding healthy parenchyma. In other cases, early stage contusions appear as small hypodense areas often in-

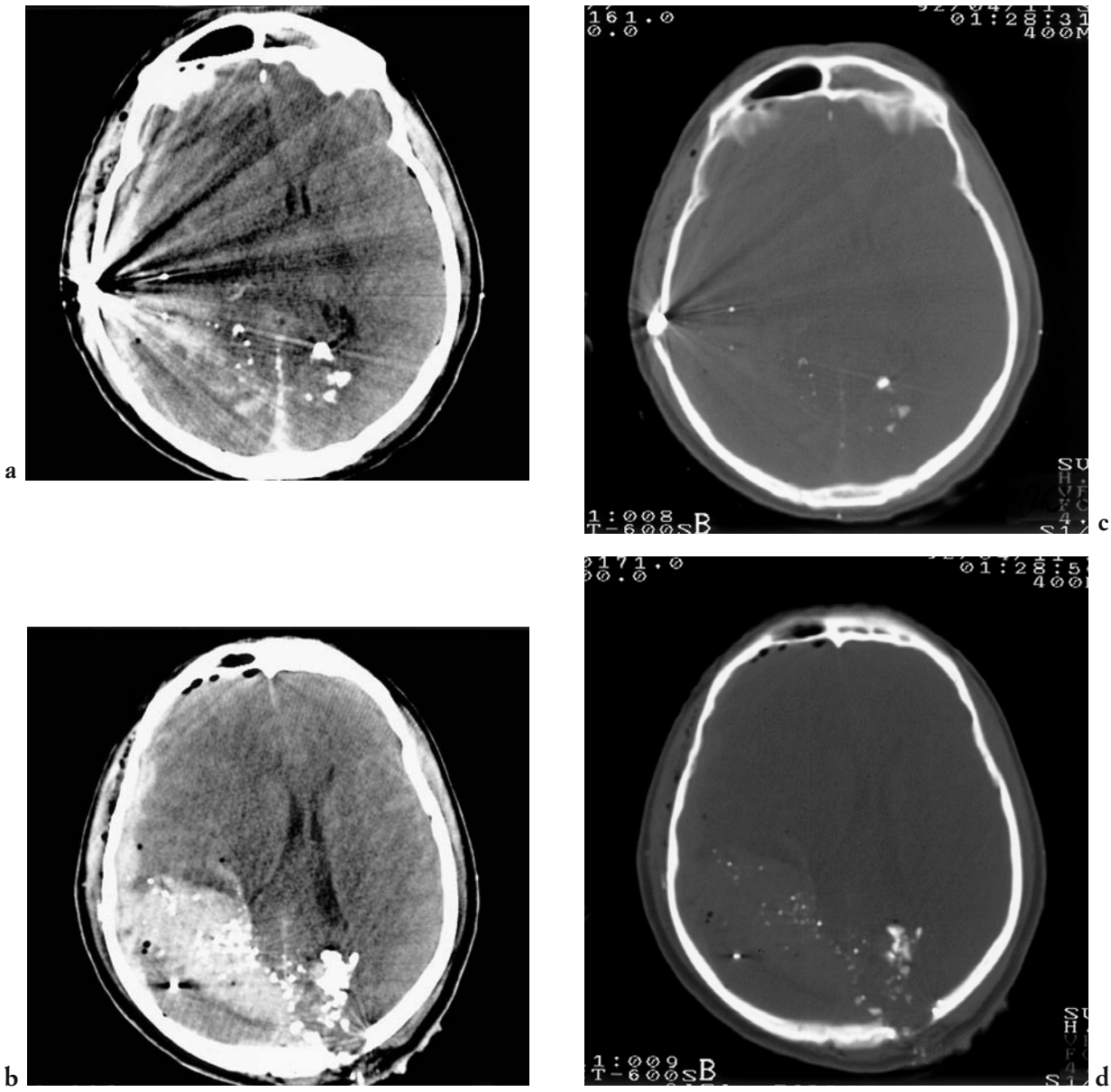


Fig. 2.3 - (**a-d**) Open head injury caused by firearm, examined using a soft tissue (**a**, **b**) and bone (**c**, **d**) algorithm. One can clearly observe the bullet entrance hole (**b**, **d**) in a left paramedian parieto-occipital location, the bullet's pathway that ends against the fractured parietal bone (**a-c**), various metal and bone fragments which appear hyperdense a right parietal subdural haematoma, midline shift to the left and obliteration of the cortical sulci (**a**, **b**).

termingled with small hyperdense haemorrhagic foci.

In serial CT scans performed 24-48 hours following the trauma, the lesions become more obvious due to the increase in oedema and the resulting enlarging mass effect. The trauma may also be responsible for frank damage to the intracerebral vessels with consequent progressive haemorrhagic extravasation (7, 29). Therefore, it is important that repeat CT scans be performed within the first 24 hours of the initial trauma to reassess the condition of the brain for possible intervention.

In such cases the CT shows areas of mixed density due to a combination of hyperdense haemorrhagic foci often in combination with

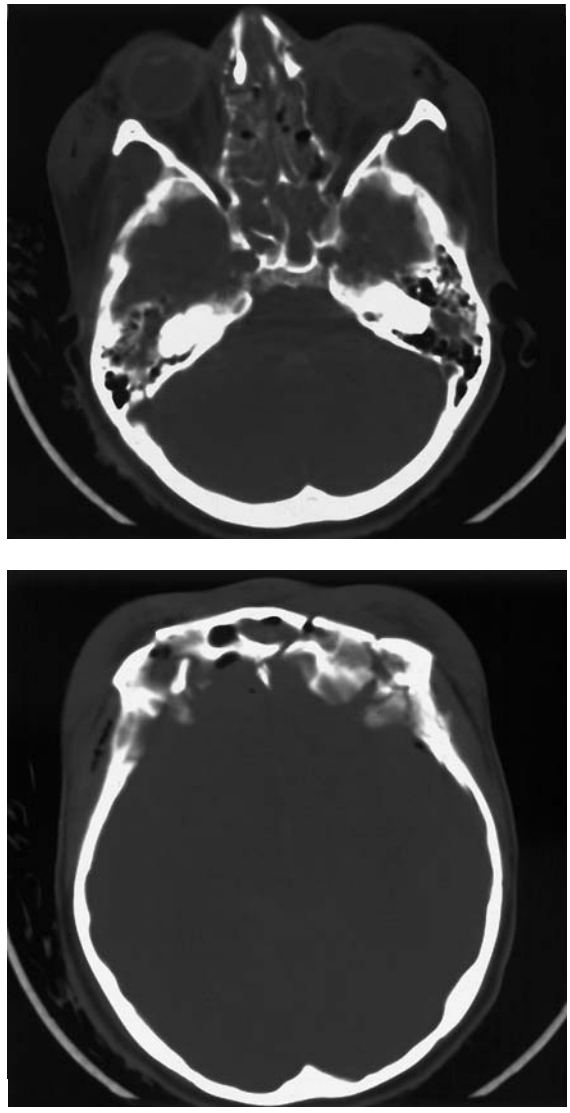


Fig. 2.4 - (**a**, **b**) Open head-facial injury caused by road traffic accident. Multiple fractures of the left orbital, ethmoidal and frontal bones are noted, with fluid within the ethmoid air cells and frontal sinus (**a**, **b**).

confluent hypodense non-haemorrhagic areas (Fig. 2.8 a, b). The haemorrhagic component gradually resolves and disappears completely within 2-4 weeks (Fig. 2.8 c, d), whereas the oedematous areas may persist for longer periods before complete recovery; alternatively, irregular areas of hypodensity may remain indicating residual encephalomalacia (Fig. 2.8 e, f), and in some cases parenchymal cavitation occurs (3, 20).

Traumatic intraparenchymal haematoma

Intraparenchymal haematomas are usually caused by combined torsion and compression forces applied to the intraparenchymal vessels, causing them to rupture; less frequently they are a consequence of direct vascular damage in-

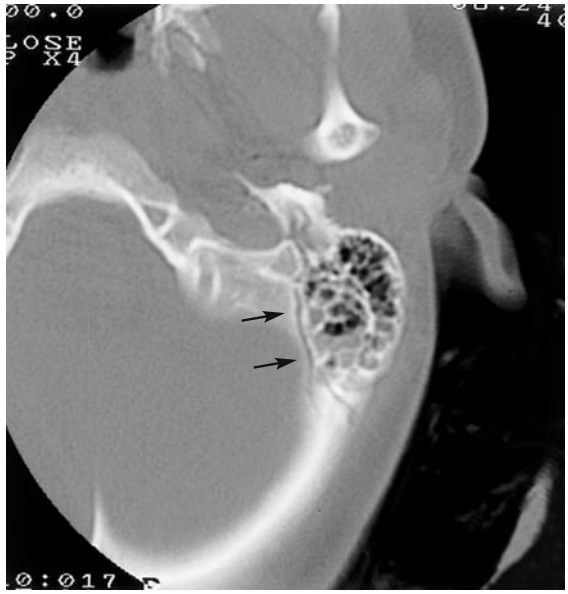


Fig. 2.5 - Fracture of the left petrous bone, with fluid within the mastoid air cells (arrows).

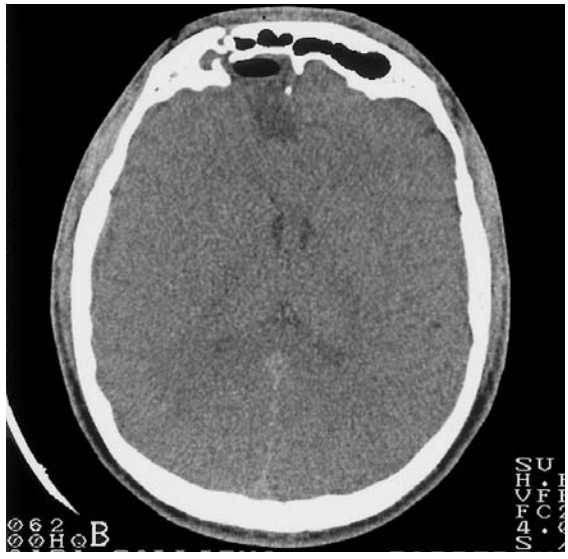
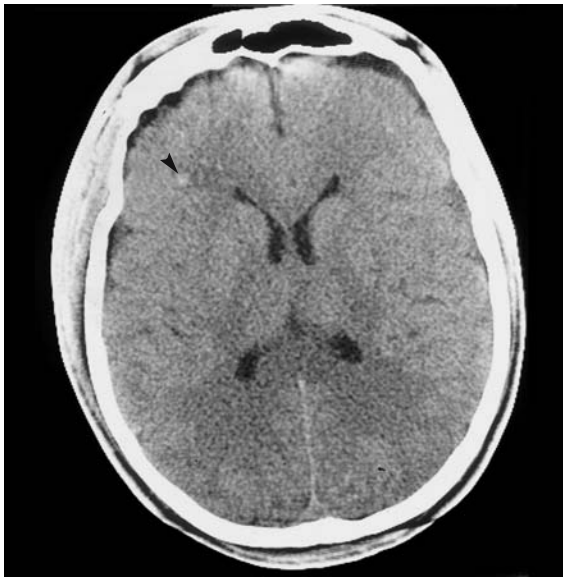


Fig. 2.6 - Frontal head injury caused by road traffic accident with fractures of the walls of the right frontal sinus and the formation of an air-fluid level.



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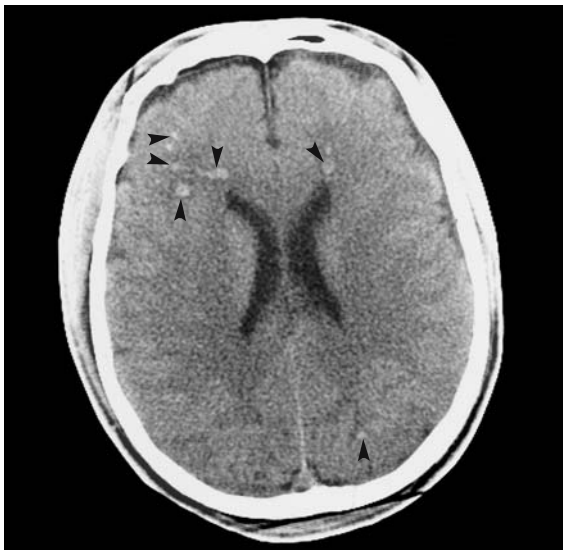

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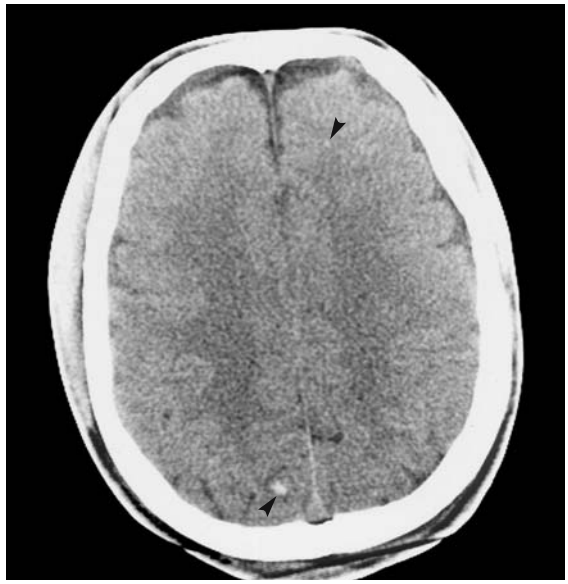


c

Fig. 2.7 - (**a-c**) Diffuse axonal injury (damage). Multiple hyperdense foci are observed with haemorrhage and compression of the white-grey matter junction bilaterally (arrow heads).

curred from penetrating injuries. These haemorrhages vary in dimension from a few mm to several cm and occur in 2-16% of all head injury patients (3). Haematomas can occur acutely, typically due to primary vessel rupture, or may appear sometime after the trauma, as a result of the confluence of multiple small laceration-contusion foci; CT scans performed in such cases immediately after the traumatic incident may be completely negative. Unlike patients with DAI or cortical contusion, trauma patients with single intraparenchymal haematomas may not lose consciousness; in 30-50% of cases these patients remain lucid for the entire duration of the clinical outcome (3, 15). Signs and symptoms in parenchymal haematomas and their clinical evolution are similar to those observed in extraaxial haematoma patients, with the exception of large or temporal lobe haemorrhages that have an unpredictable outcome; even when small, these haematomas can cause brainstem damage due to associated transtentorial herniation (3, 7, 29).

Intraparenchymal haematomas usually occur in the same or nearly the same position in which the primary trauma was applied to the cranium as a typically well-circumscribed hyperdensity with or without a rim of perilesional oedema, depending upon the time of the CT acquisition relative to the traumatic incident (acute: no perilesional oedema; subacute: perilesional oedema; Fig. 2.9). These haematomas are frequently observed within the frontal and temporal lobes. Up to 60% of cases are associated with extradural haematomas, either epi- or subdural in location. If the haematoma develops in a periventricular position, it can rupture into the cerebral ventricles. Larger haemorrhages result in considerable mass effect and have the added risk of downward transtentorial internal herniation of cerebral and brain-

stem tissue through the tentorial incisura, or even the possibility of herniation of the cerebellar tonsils through the foramen magnum later in the process of haemorrhagic expansion. Clinically it is not always easy to distinguish intraparenchymal haemorrhage from DAI or parenchymal contusion; this differential diagnostic difficulty probably accounts for the marked variability in the clinical evolution statistics as reported in the literature (3). The prognosis for isolated intraparenchymal haematomas is fairly good, but worsens considerably with increasing degrees of mass effect or when it is associated with DAI or haemorrhages into the basal ganglia.

rhages are occasionally seen in combination with choroid plexus haematomas, haematomas of the deep grey or white matter and subarachnoid haemorrhage (Fig. 2.22c).

Traumatic lesions of the deep grey structures and the brainstem

This type of lesion is caused by stretching and torsional forces that cause the rupture of small perforating vessels, or by the direct impact of the dorsolateral surface of the brainstem against the tentorial incisura. These lesions are less frequently observed than those mentioned in the preceding sections and represent approximately 5-10% of primary traumatic pathology (3, 20, 22). They are associated with severe clinical states, and usually have unfavourable prognoses. Depending upon the clinical severity, CT can be entirely negative or may show relatively minor findings, including small haemorrhages in the areas of the brainstem surrounding the cerebral aqueduct and in the basal grey nuclei. As compared to CT, on MRI these types of lesions appear relatively more clearly.

Tab. 2.4 - Comparison between subdural and epidural haematomas

	EPIDURAL HAEMATOMA	SUBDURAL HAEMATOMA
Incidence	1-4% of trauma cases; 10% of fatal trauma cases	10-20% of all trauma cases; 30% of fatal trauma cases
Aetiology	Associated fractures in 85-95% of cases; Laceration of middle meningeal artery/dural venous sinus in 70-80% of cases.	Tearing of the cortical veins of the pons
Site	Between skull and dura mater; Crosses the dura mater but not the cranial sutures; 95% are supratentorial 5% are subtentorial; 5% bilateral	Between dura mater and arachnoid mater; Crosses the cranial sutures but not the dura mater; 95% are supratentorial; 5% are bilateral
CT findings	Biconvex (lens) shape; Shifts the white-grey matter interface; 66% are hyperdense; 33% are mixed (hyper-/hypodense) Crescent shape;	*Acute*: 60% hyperdense; 40% mixed (hyper-/hypodense) *Subacute*: isodense; *Chronic*: hypodense

Intraventricular haemorrhage

Traumatic intraventricular haemorrhage is somewhat uncommon and is only present in 1-5% of closed head injuries; it is usually a consequence of particularly severe trauma and tends to be associated with DAI and traumatic lesions of the deep grey matter and brainstem. The clinical prognosis is generally poor (3, 13, 20, 22). CT shows hyperdense intraventricular collections, which may or may not have associated fluid-fluid levels. Intraventricular haemor-

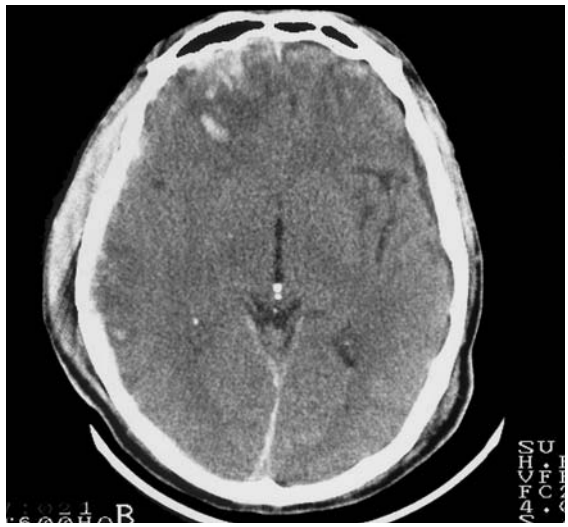
Extraaxial traumatic lesions

Epidural haematomas

Overall, epidural haematomas are present in 1-4% of head injury cases, and in 10% of fatal cranial head injuries. These injuries are quite rare in patients over 60 years of age, because of the increased adherence of the parietal dura mater to the overlying inner table of the skull (Tab. 2.4).

Epidural haematoma patients may present with few or minor signs and symptoms. In almost 50% of all cases there is a typical interval of mental lucidity between the traumatic event and the onset of severe neurological deterioration or coma (3, 13). In 10-30% of cases, the imaging findings may first appear and even progress over the first 24-48 hours following the traumatic incident (20, 22).

Epidural haematomas are situated between the dura mater and the internal bony table of the skull, and they are almost always associated with skull fractures (Fig. 2.10). On occasion, parenchymal countercoup lesions may be observed on the side of the cranium opposite to the primary traumatic blow (3, 20, 22). These haematomas are often secondary to lacerations of the middle meningeal artery (when in the temporoparietal region), or more rarely, tears of the cranial veins such as the diploic and meningeal veins, and the dural venous sinuses. In this latter case, the epidural haematoma straddles the midline when the sagittal venous sinus is involved (direct coronal acquisitions or scans reconstructed in the coronal plane may be required for precise demonstration); alternatively, the epidural haemorrhage may extend between the supra- and infratentorial compartments, if the lateral dural venous sinuses are involved. Arterially fed epidural haematomas are typically observed in the acute phase because they grow under arterial blood pressure, thus compressing the brain and resulting in early downward internal cerebral herniation coupled with rapid clinical deterioration and eventual coma (7, 29). Venous epidural haematomas are usually small by comparison as the accumulation of blood takes place under relatively low venous pressure. Larger venous epidural haematomas are typically seen when resulting from the rupture of one of the larger dural venous sinuses. The CT density of the extraaxial blood collection depends in part upon the phase in which the haematoma is imaged. In the acute phase, epidural haematomas appear as homogeneously hyperdense lesions, with a biconvex or lens shape; the varying mass effect can be observed in the contralaterally displaced ventricular and extraventricular CSF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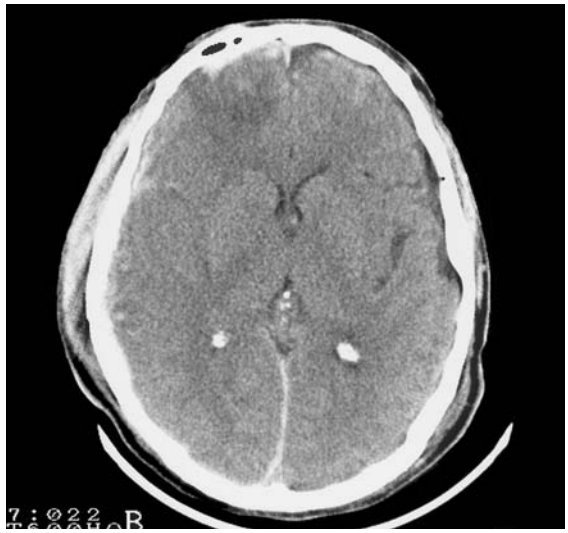
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Fig. 2.8 - (**a-f**) Temporal evolution of contusive haemorrhagic focus. (**a-b**) CT examination a few hours following trauma: irregular haemorrhagic foci are seen in both frontal lobes and in right temporal-parietal region with the obliteration of the ipsilateral Sylvian fissure cistern.

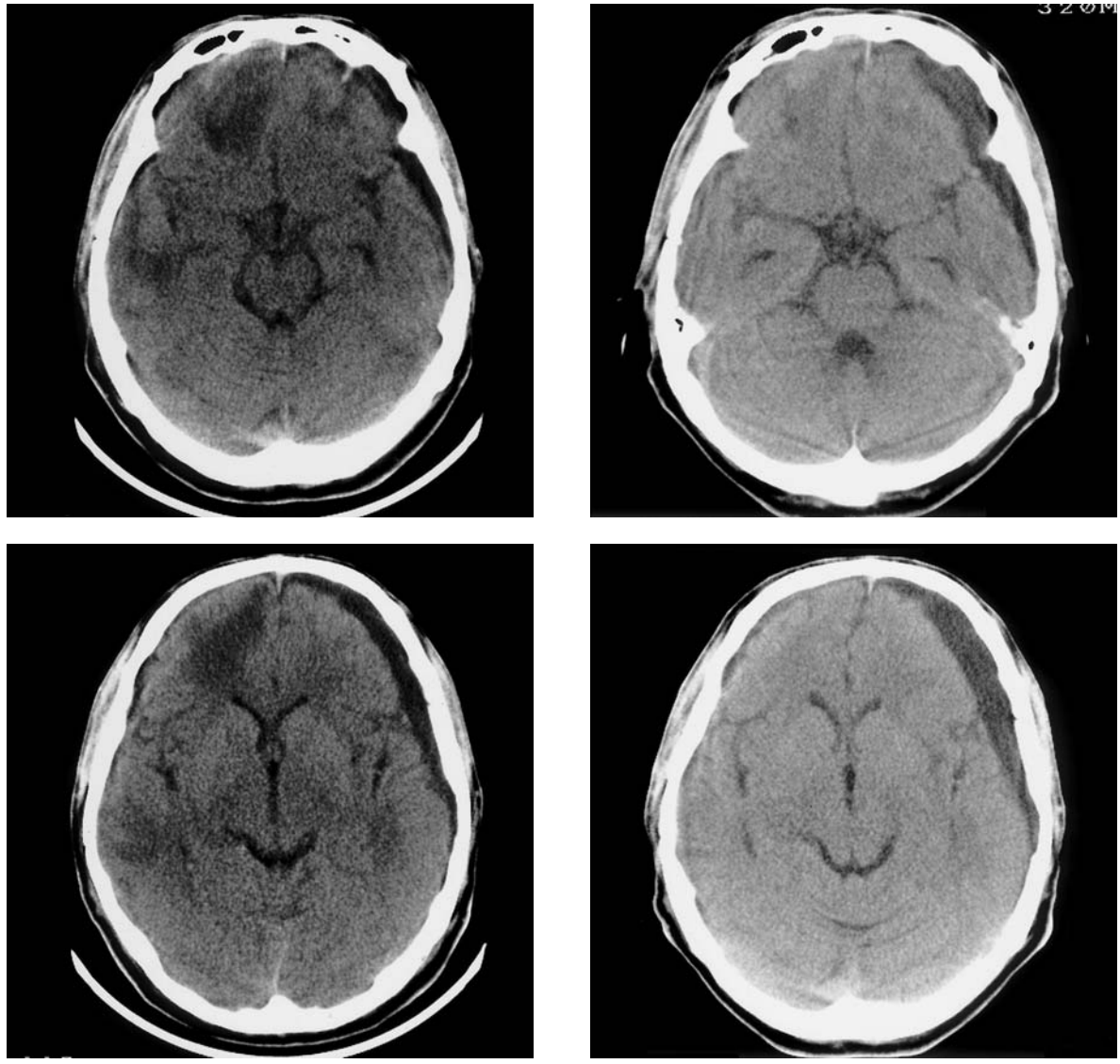


Fig. 2.8 (*cont.*) - (**c-d**) Follow up after 3 weeks: The hyperdense haemorrhage has been almost completely resolved, whereas the hypodense component is still clearly visible; a subdural hygroma has accumulated on the left side. (**e-f**) Follow up after 6 weeks: There is partial resolution of the hypodense component, a residual of which persists in a right frontal region; the hygroma on the left side remains.

spaces (Figs. 2.10 and 2.11). Rarely epidural haematomas can have an isodense appearance immediately subsequent to the traumatic incident, if imaging is undertaken before the fresh blood clots (another cause may be a very anaemic patient).

In certain cases of venous epidural haematoma, CT examinations conducted immediately after the trauma may reveal little, whereas the haematoma appears only 2-3 days later. This delay in the appearance of the haemorrhage is due to the fact that in venous epidural haematomas the injured veins bleed relatively slowly; alternatively, a delay in epidural haemorrhage from a traumatized arterial source may occur because blood can only flow into the epidural space after resolution of the trauma-induced arterial spasm. The haematoma can also be dampened by the mass effect of overlying brain contusion. In the case of persistent bleeding within an extraaxial blood collection, the haematoma can appear of mixed den-

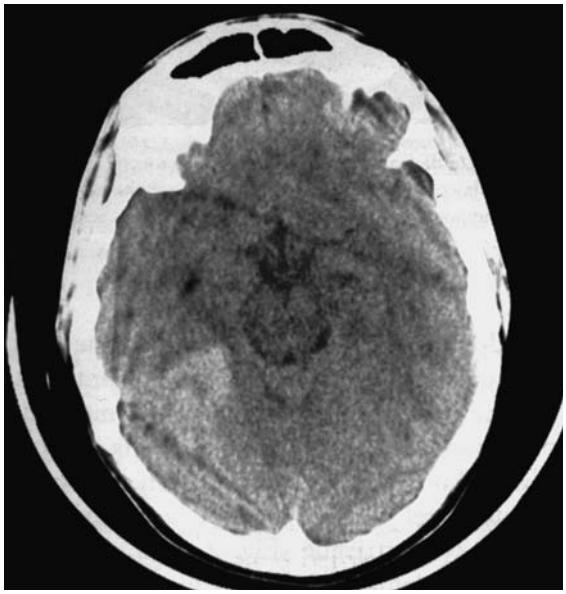


Fig. 2.9 - An acute post-traumatic intraparenchymal haematoma in observed in the right temporal-occipital area.

sity due to the presence of non-clotted new blood alternating with older clotted haemorrhage (7, 29). In subacute forms, as in the case of bleeding caused by the rupture of the dural venous sinuses, the density of the clotted haemorrhage varies in part according to the resorption of the solid blood constituents (Fig. 2.12).

Acute epidural haematomas represent the most urgent of all cases of cranial trauma. They require swift treatment before irreversible parenchymal damage occurs, caused by the compression of brain structures, especially the brainstem. Although rare (5% of extraaxial haematomas), extradural haematomas in the posterior fossa are potentially the most worrisome and tend to demonstrate the most rapid compromise of patient vital functions. As an additional factor, the compression of the aqueduct of Sylvius may cause acute obstructive hydrocephalus resulting in progressive supratentorial mass effect and accelerated downward transtentorial internal cerebral herniation.

Subdural haematoma

Subdural haematomas (SDH's) are one of the most life threatening events that can occur

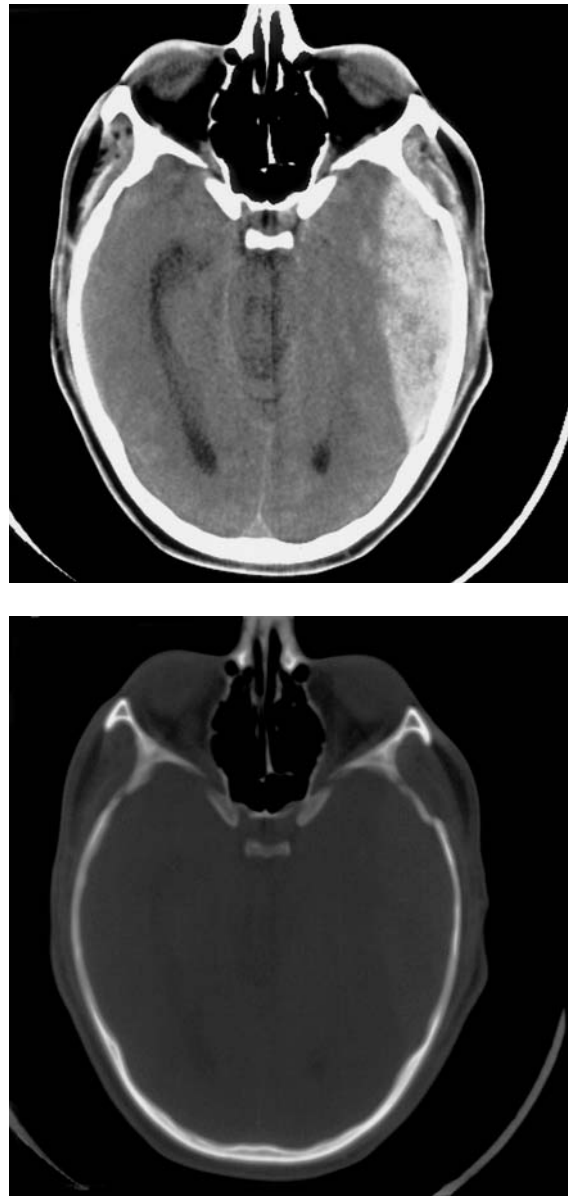


Fig. 2.10 - (**a-b**) A large epidural haematoma is seen in left temporal-parietal region (**a**) associated with an overlying skull fracture (**b**).

Tab. 2.5 - Post-traumatic sequelae of head injuries

1) - Cortical atrophy
2) - Encephalomalacia
3) - Pneumocephalus
4) - Leptomeningeal cyst formation
5) - Cranial nerve lesions
6) - Diabetes insipidus (pituitary injury)
7) - Hydrocephalus (communicating or obstructive)

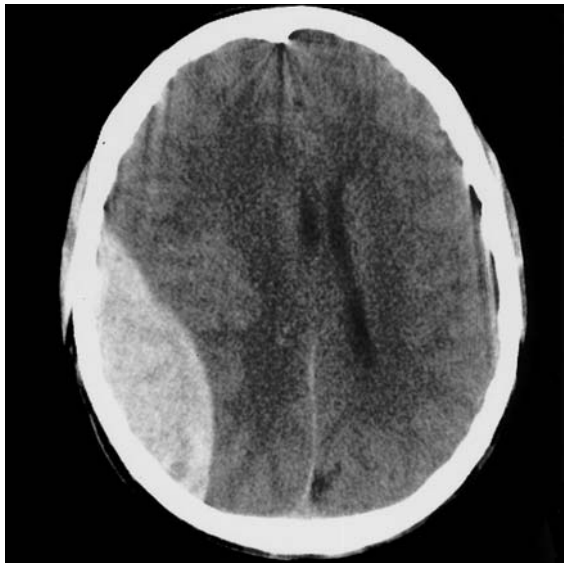


Fig. 2.11 - Axial CT shows an epidural haematoma with biconvex lens shape, in right parietal region with compression of the cerebral parenchyma and midline shift.

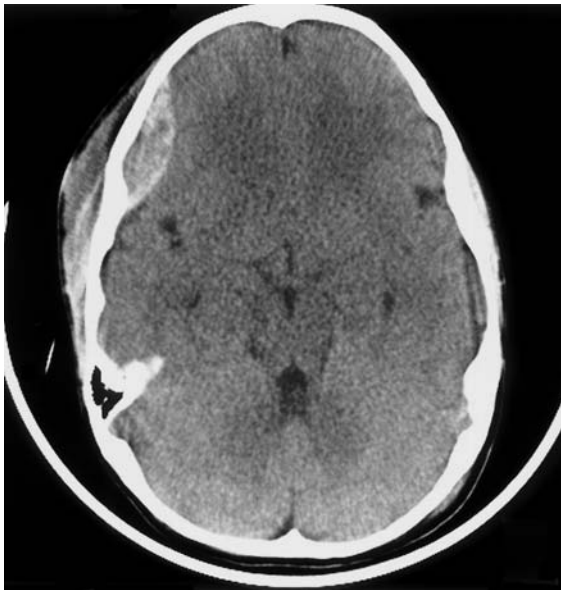


Fig. 2.12 - Axial CT reveals a small right frontal subacute epidural haematoma with mixed hypodense and hyperdense component, concomitant extracranial haematoma.

following head injuries, having a 50-85% mortality rate (20) (Tab. 2.5). They occur in 10-20% of all cases of cranial trauma and are frequent in the elderly with underlying brain atrophy and large intracranial subarachnoid spaces, and in physically abused children who have been subjected to strong shaking (i.e., "shaken baby syndrome") (3, 6, 20, 23). In elderly patients they may arise in the absence of a traumatic episode (18, 19). If the haematoma is isolated and small, symptomatology may be absent or minor (e.g., headache). However, the SDH is often associated with DAI and/or severe mass effect, and patients therefore tend to be clinically compromised, with low Glasgow Coma Scores; in up to 50% of cases, patients appear flaccid or even decerebrate (13).

SDH is a collection of blood in the subdural space between the external surface of the leptomeninges and the dura mater secondary to the laceration of veins (in particular the veins involved are those that join the cerebral cortical veins and veins of the pons with the dural sinuses), or in the more severe forms, secondary to or associated with underlying cortical laceration. SDH's can occur at any age, but are most frequently observed in patients aged 60-80 years because of the greater mobility of the brain within the skull secondary to senile atrophy and therefore the greater ease with which veins rupture upon acute traumatic stretching of the attached cortical venous structures (18, 19). SDH's are more frequent than are epidural haematomas and are relatively more often associated with contusion-type injuries rather than skull fractures. This relationship is useful in predicting patient prognosis and in part explains why the overall intracranial mass effect is larger than are the actual dimensions of the SDH. Distinctions are made between the acute (i.e., within 3 days from the trauma), subacute (i.e., within 3 months) and chronic (i.e., more than 3 months) forms of SDH. In the acute phase, subdural haematoma appears as a hyperdense, sickle-shaped extraaxial lesion (Fig. 2.13); in some cases there may be a medial beak at the level of the pterion, where it penetrates the anterior and lateral aspects of the Sylvian fissure between the opercula of the frontal and the temporal lobes. Atypical configurations may be encountered in very large SDH's (Fig. 2.14a) or in the presence of fibrous bands traversing the subdural space resulting from previous trauma or inflammation; these cases of SDH can take the form of a biconvex lens or a multilocular appearance.

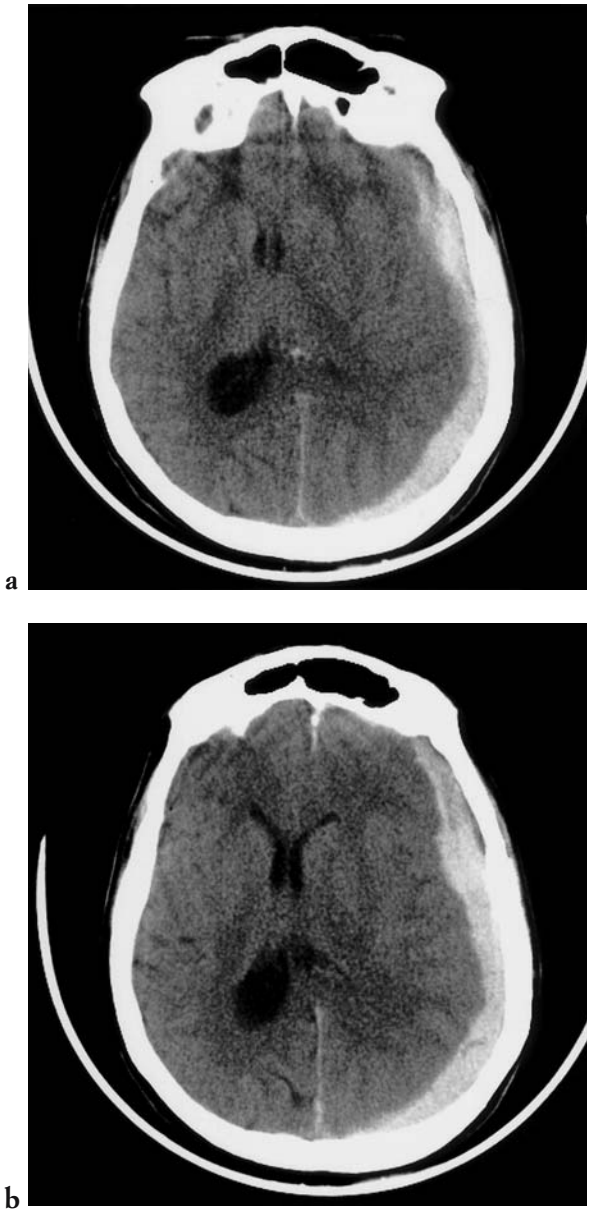


Fig. 2.13 - Axial CT demonstrates an acute subdural haematoma along the left hemisphere convexity with right shift of the midline structures and compression of the left lateral ventricle. [**a**), **b**) axial CT].

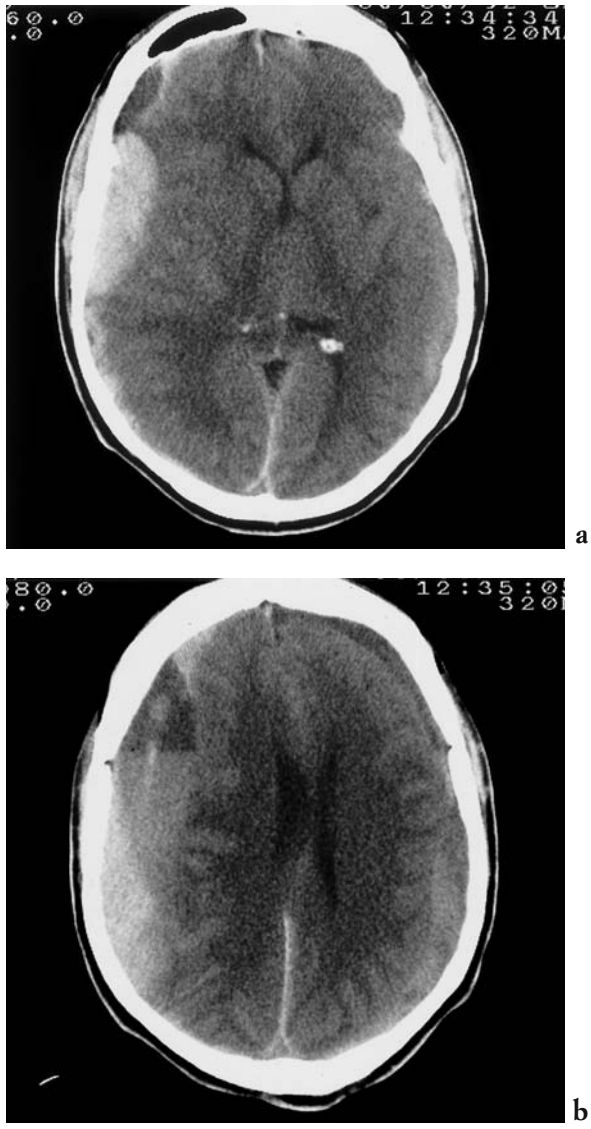


Fig. 2.14 - Bilateral subdural haematomas. There is a biloculated acute-subacute subdural haematoma on the right side (**a**), a heterogeneous appearance of the two haematomas with fluid-fluid levels (**b**), and presence of haemorrhagic components of different ages. The left sided subacute subdural haematoma is almost isodense as compared to the underlying brain and is therefore somewhat difficult to visualise. [**a**), **b**) axial CT].

Subsequently, due to the effect of the metabolism of the protein content of haemoglobin, SDH's initially become isodense (e.g., between the 7th and 21st day following the traumatic incident) (Fig. 2.15), and then evolve into a generally hypodense lesion (Figs. 2.16, 2.17) relative to the density of the underlying brain tissue. In practice, an SDH can also be isodense or hypodense outside of this predicted phase density pattern if the patient is anaemic, or if the systemic blood is diluted with iatrogenically administered fluid; conversely, chronic SDH's can show progressive hyperdensity due to interim haemorrhage(s) which can be asymptomatic and unprovoked by recurrent trauma (i.e., spontaneous) (Fig. 2.16). In cer-

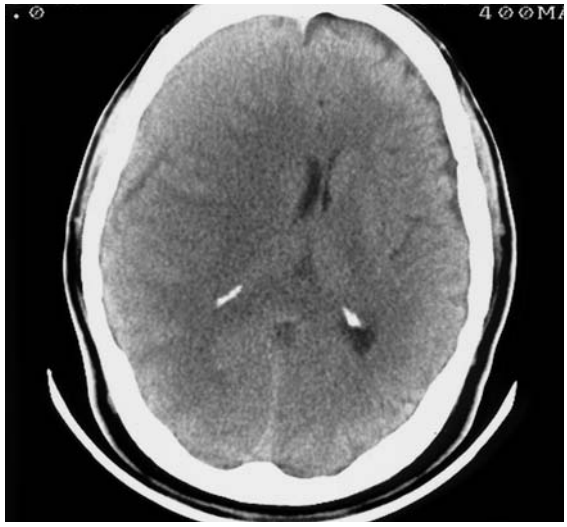
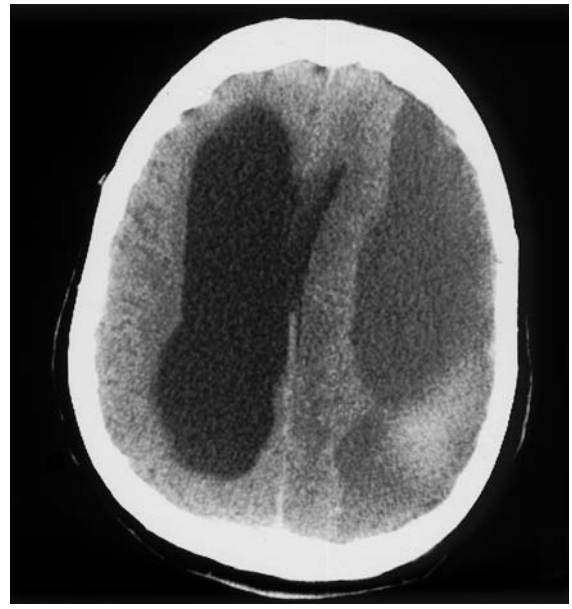


Fig. 2.15 - Subdural haematoma. Axial CT shows a subacute right frontal subdural haematoma is noted that is isodense and associated with obliteration of the cortical sulci, displacement of the corticomedullary junction away from the overlying skull, leftward shift of the midline structures and compression of the adjacent lateral ventricle.

tain cases in the subacute or chronic phases, a blood-fluid level can be observed following blood clot liquefaction, perhaps contributed to by haemorrhagic sedimentation (hypodensity superiorly and iso- or hyperdensity inferiorly) (Fig. 2.14b). In some cases, haemorrhagic events are followed by blood clot organization, which gives the crescent image a layered curvilinear appearance, with the older, hypodense collections on the medial and lateral margins and the more recent haematomas showing a relatively denser core; complex, crescent-shaped layers of varying density, representing haematomas and membranes of different ages, may subsequently occur.

In rare cases SDH's can reabsorb spontaneously, although some increase in volume and become chronic in duration. The eventual evolution depends in part upon the hyperosmolarity of liquefied blood products, as well as on whether or not new bleeds have occurred over the interim. The sites of SDH's are usually supratentorial and especially over the convexity; due to the lack of barrier to progression at this level, the extravasated blood easily extends along the entire hemicranium. If the SDH is situated at the vertex, it can go unnoticed on axial slices, and therefore coronal scans are required in appropriate cases (Fig. 2.18). If unilateral, SDH's are accompanied by a contralateral shift of the midline structures and the lateral cerebral ventricles (Fig. 2.19); if bilateral, these extraaxial haemorrhages cause compression of the ventricles that assume a



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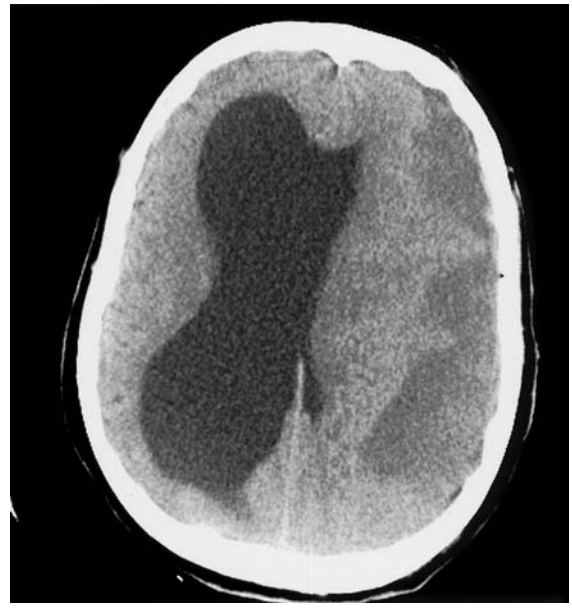


b

Fig. 2.16 - Subdural haematoma. Axial CT reveals and unusual appearing left frontoparietal subdural haematoma with a hyperdense acute haemorrhagic component layering posteriorly. The left lateral ventricle is compressed by the haematoma and the contralateral lateral ventricle is dilated. [**a**), **b**) axial CT].

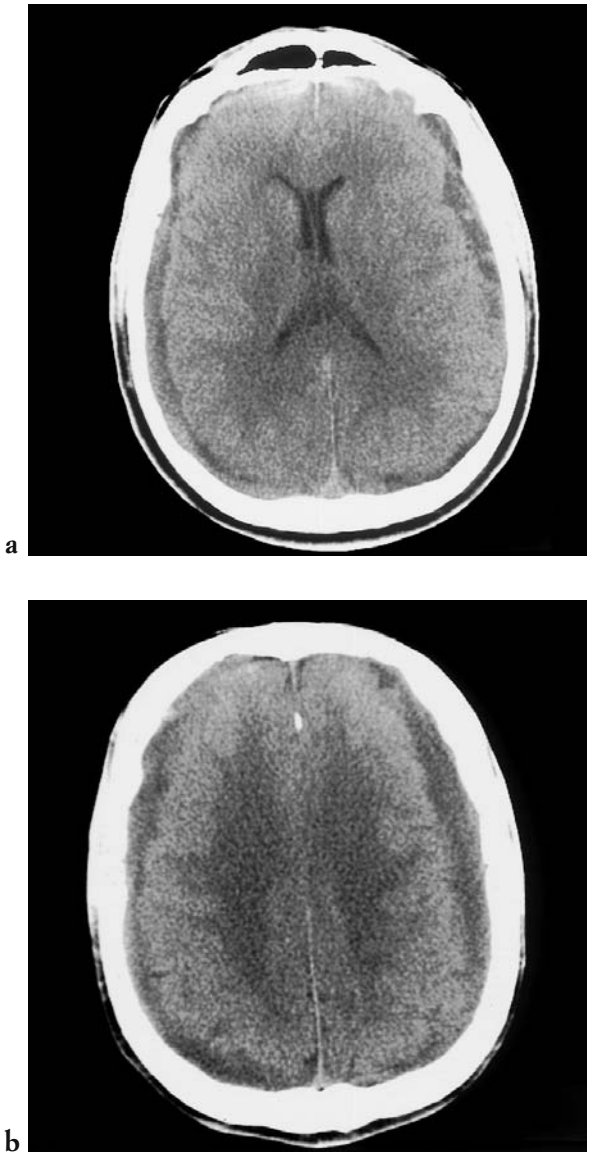


Fig. 2.17 - Bilateral subdural haematomas. Axial CT shows bilateral hypodense subdural haematomas. [**a**), **b**) axial CT].

thinner, elongated and parallel appearance (Fig. 2.20). In both instances, the characteristic finding is the disappearance of the underlying cortical sulci and the displacement of the superficial brain parenchyma away from the bony inner table of the skull. For the demonstration of these SDH's, especially when the haemorrhage is isodense, it is important to identify the corresponding cortico-medullary junction and the digitations of the white matter that penetrate into the gyral folds. The centrum semiovale usually has a convex lateral margin; however, in the presence of an SDH this margin becomes flattened, concave or irregularly distorted. If the cortico-medullary junction is not visible, a bolus injection of contrast medium administered during the scan may be useful in identifying the cortical surface and delineating the border of an isodense SDH. This image enhancement method can document

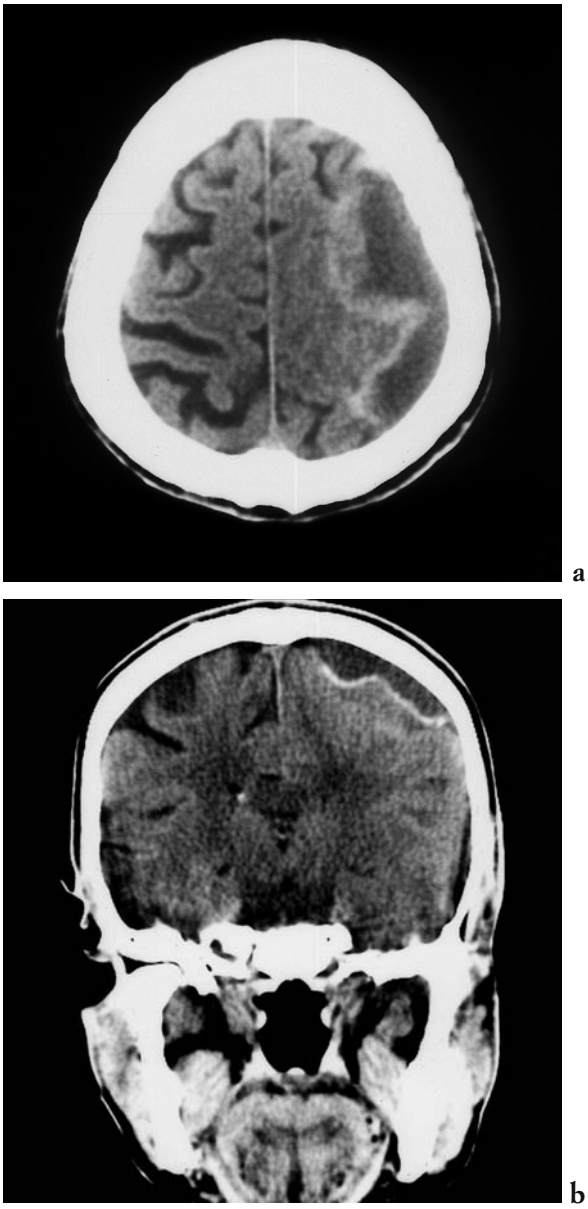


Fig. 2.18 - Subdural haematomas. Axial and coronal CT shows small, chronic subdural haematoma over the convexity of the brain is observed which is better seen on coronal scans. [**a**) axial CT; **b**) coronal CT].

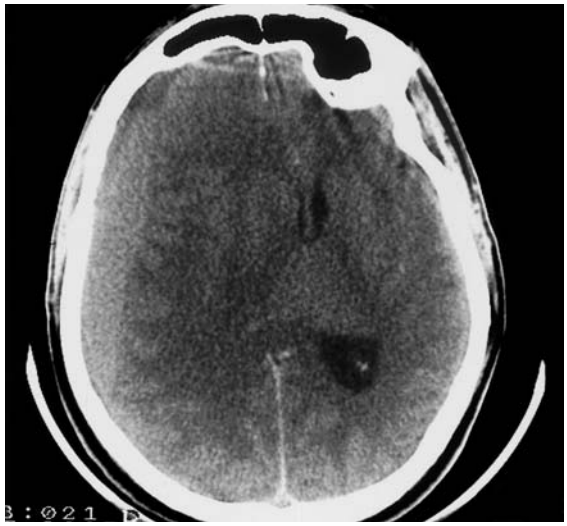


Fig. 2.19 - Subdural haematoma. The axial CT shows a right frontoparietal subdural haematoma with associated mass effect upon the adjacent cerebral parenchyma, which causes a subfalcian herniation of the cingulate gyrus and the compressed right lateral ventricle. The left lateral ventricle is dilated due to a CSF obstruction at the level of the left foramen of Monro.

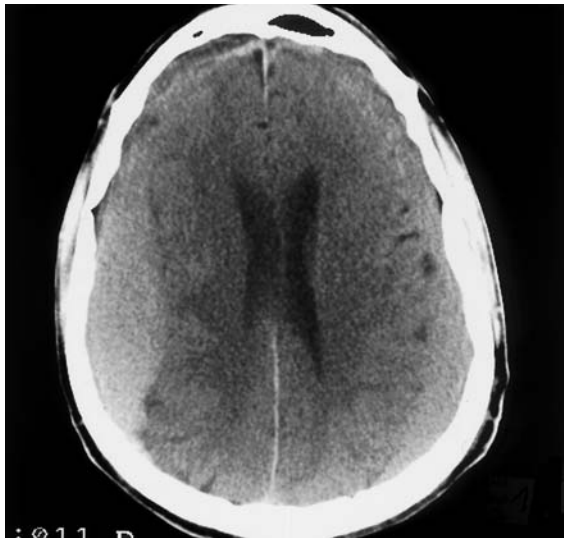


Fig. 2.20 - Bilateral subdural haematomas. The CT study shows lateral ventricles that are thinned, elongated and parallel to one another. The left sided subdural haematoma is almost isodense as compared to the underlying brain.

a thin line of enhancement at the brain surface (perhaps due to compression ischaemia), a focal traumatic rupture of the blood-brain barrier, visualization of a membrane related to the prior haemorrhage or prominent enhancement of dilated cortical veins.

SDH's do not cross the midline because the subdural spaces on the two sides of the cranium do not communicate. These haematomas are occasionally observed beneath the temporal lobe within the middle cranial fossa and under the occipital lobes extending along the tentorium cerebelli, and are often difficult to see with axial slices alone. In these instances, supplemental coronal slices are helpful for visualization.

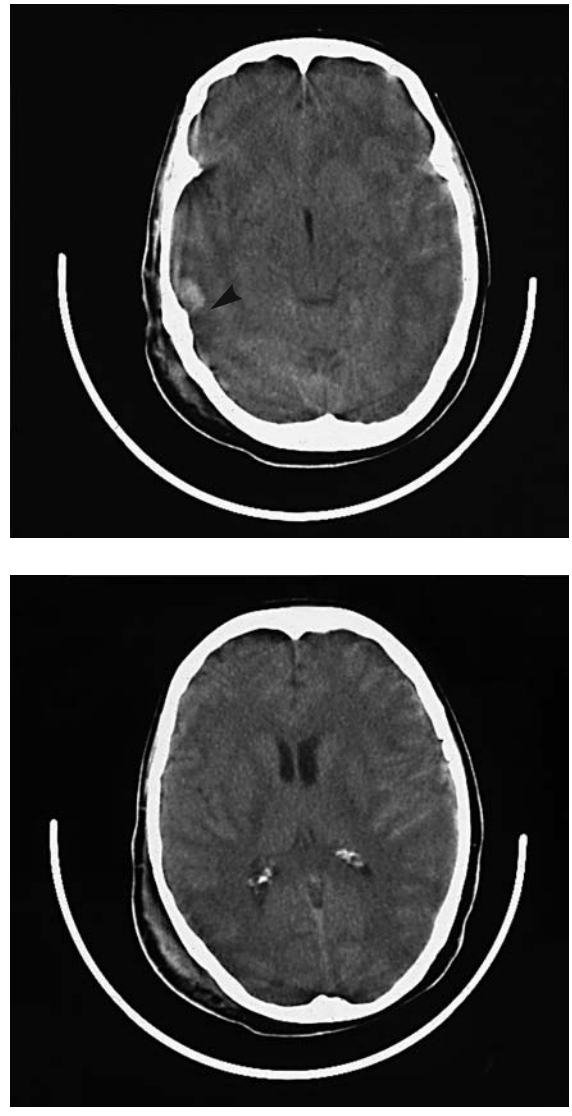


Fig. 2.21 - Subarachnoid haemorrhage. Hyperdensity is noted within the cortical sulci over the left cerebral hemisphere. Small haemorrhagic cortical contusions are also seen, the largest of which can be observed in the right temporal region (arrowhead). [**a**), **b**) axial CT].

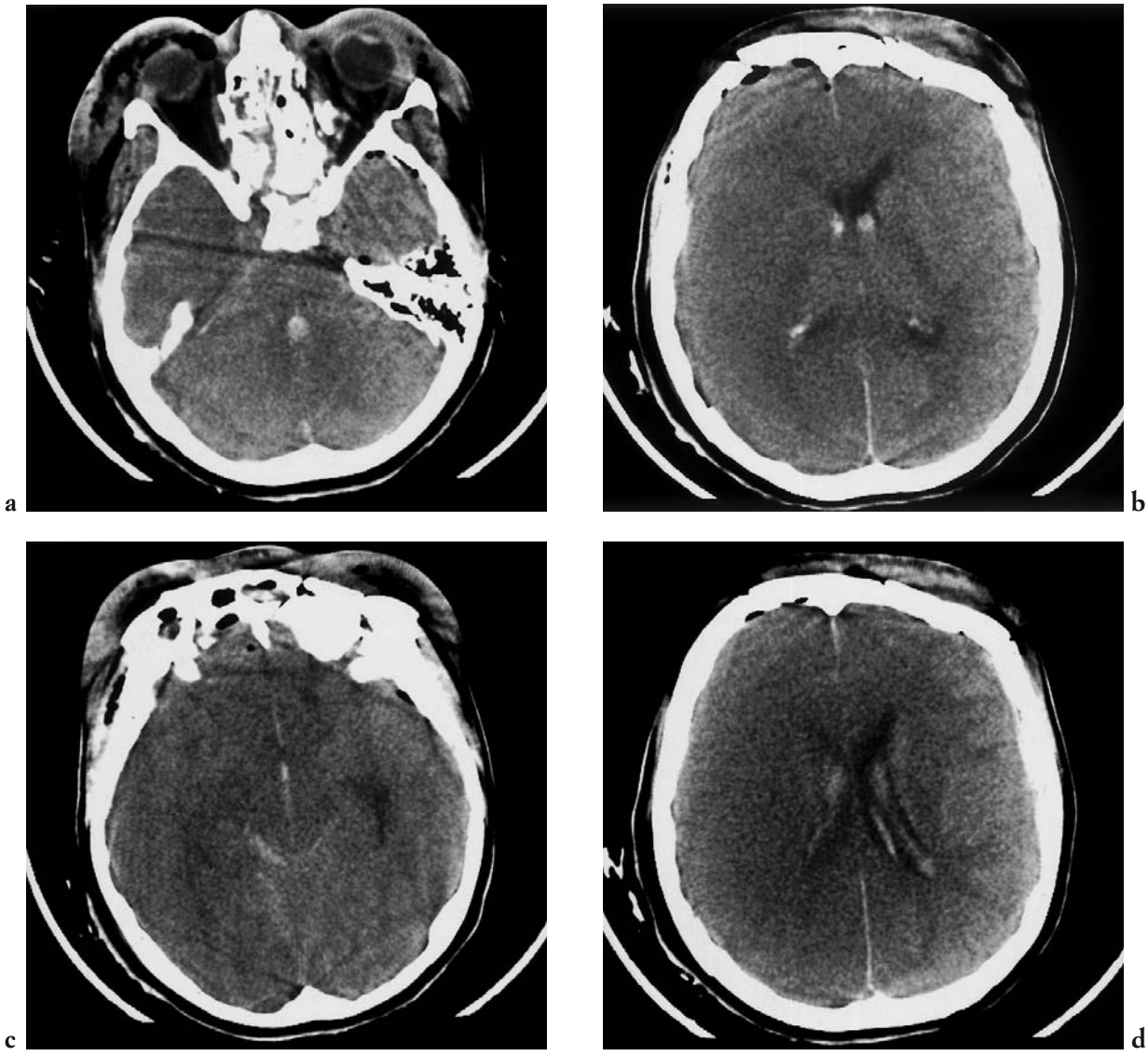


Fig. 2.22 - Posttraumatic intraventricular haemorrhage, pneumocephalus, and cerebral oedema. Bilateral eyelid haematomas are seen associated with subcutaneous emphysema (**a**). Intracranially are observed subarachnoid (**b**) and intraventricular haemorrhage (**a**), air bubbles in the CSF spaces (**c**) and the widespread oedema within the right cerebral hemisphere (**d**). [**a**)-**d**) axial CT].

Subarachnoid haemorrhage (SAH)

SAH's typically occur after severe cranial trauma and are usually associated with haemorrhagic parenchymal contusions (Figs. 2.21 and 2.22) (3, 29). Traumatic SAH's are usually somewhat insignificant from a clinical point of view, being linked pathologically to the rupture of small cortical vessels that traverse the subarachnoid space. These haemorrhages are seen as hyperdense collections of blood in the sulci, fissures and cisternal spaces, especially around the Sylvian fissure and the interpeduncular cistern (Figs. 2.21 and 2.22) (20, 22).

The site of the SAH is often somewhat distant from that of the trauma because the blood tends to diffuse within the subarachnoid spaces. In some cases the blood can reach the ventricular system, due to retrograde flow through the foramina of Luschka and Ma-

gendie. The ability of CT to detect SAH is directly related to the quantity of extravasated blood as well as to the time from the traumatic incident; the SAH can be negative if the CT scan is performed some days after the event. SAH's can sometimes be falsely simulated in certain particularly severe cases of diffuse cerebral oedema, in which the brain appears relatively hypodense in comparison to the underlying dura mater and neural tissue (20).

Subdural hygroma

A subdural hygroma is an extraaxial collection of CSF caused by the extravasation of this fluid from the subarachnoid space through a traumatic tear in the arachnoid mater (Fig. 2.23). The acute form is particularly frequent in children and less so in adults (6, 23). Subacute

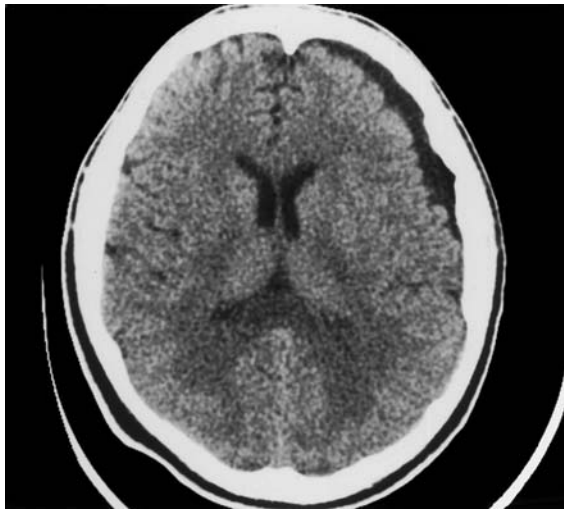


Fig. 2.23 - Posttraumatic subdural hygroma. The axial CT shows a left sided frontal posttraumatic subdural hygroma.

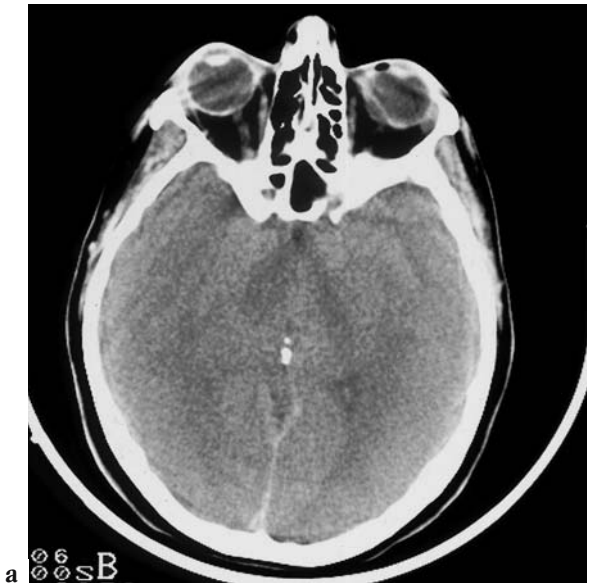
and chronic forms can be seen following surgery performed to treat severe head injuries. In such cases, the hypodense subdural collection is either located in the region of the operation or alternatively on the opposite side due to an *ex vacuo* mechanism following the evacuation of a contralateral haematoma. The differential diagnosis includes chronic, hypodense SDH.

Traumatic vascular lesions

Traumatic cerebrovascular lesions are somewhat rare, but are probably less infrequent than reported in the medical literature (3, 9, 20). In certain cases these lesions can be asymptomatic or have a clinical onset sometime after the initial traumatic incident and can therefore be overlooked on routine imaging studies in patients having undergone trauma. In other cases the presence of other craniocerebral lesions related to trauma can conceal the presence of related underlying vascular lesions, as both clinical symptoms and imaging findings may be attributed to other dominant traumatic parenchymal pathology such as DAI, intraparenchymal haematomas or extraaxial haematomas. CT is a most useful technique for identifying patients with an increased risk of vascular lesions such as those with fractures at the base of the skull extending into the bony internal carotid canal, the sphenoid bone, and the petrous pyramid of the temporal bone and the basiocciput. Of course, it should be pointed out that the presence of fractures in such sites does not necessarily indicate the presence of associated vascular lesions (7, 29).

The internal carotid artery, which is the most frequently affected vessel in cranial trauma, can undergo dissection due to the forced extension and torsion of the neck or due to the direct laceration of the arterial wall by a skull base fracture; this is especially true with trauma to the region directly adjacent to the anterior clinoid processes and the bony internal carotid canal (9). In certain cases the adventitia of the carotid artery wall can remain intact and thereby develop a pseudo-aneurysm (3, 9, 20). Other possible cerebrovascular lesions related to the trauma include dissections, lacerations and frank vessel occlusions, which may or may not be associated with perivascular haematomas.

Another pathological entity connected with brain trauma is the formation of arteriovenous fistulae. The most typical site is a fistula between the internal carotid artery siphon and the cavernous venous sinus, usually a consequence of the fracture of the central skull base. In such



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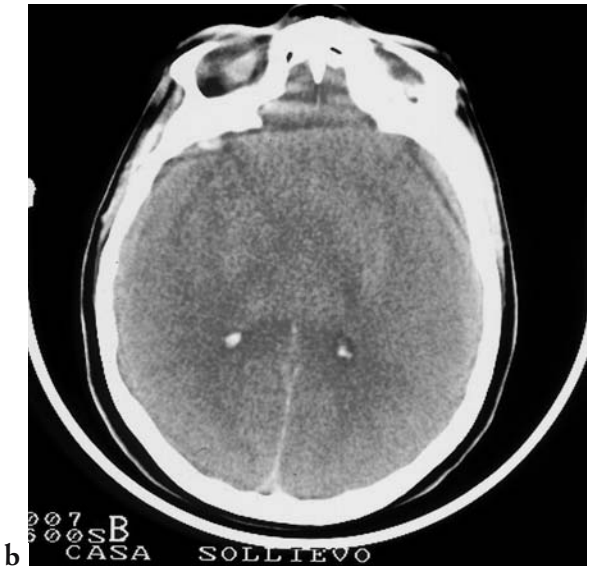

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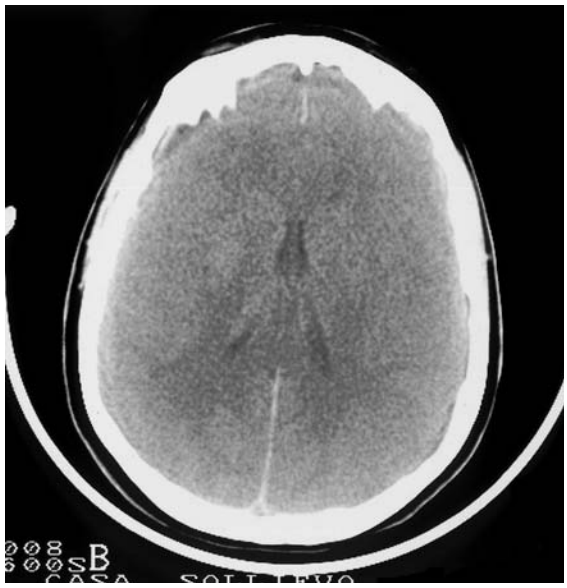

c

Fig. 2.24 - Posttraumatic cerebral swelling. The CT demonstrated diffuse posttraumatic cerebral swelling is seen with obliteration of the basal subarachnoid cisterns and superficial cortical sulci, compression of the 3rd ventricle and reduction in size of the lateral ventricles. The midline structures are not shifted, and there are no focal haemorrhages are present. [**a**), **b**), **c**) axial CT].

cases, one suggestive indirect sign on imaging is the CT finding of an enlarged, arterialized superior ophthalmic vein.

CT is very accurate in demonstrating fractures of the base of the skull, whereas greater diagnostic sensitivity in demonstrating traumatic vascular lesions can be obtained with MR or MR angiography examinations (3, 20). In selected cases where such traumatic cerebrovascular lesions are suspected on the basis of non-invasive imaging studies and clinical information, selective cerebral angiography is required to clearly define the diagnosis and to suggest optimal treatment options.

Secondary lesions

Internal cerebral herniation

Bony and dural structures grossly subdivide the cranial cavity into functional supra- and infratentorial compartments. Internal cerebral herniations are a mechanical shift of the cerebral parenchyma, cerebrospinal fluid and the attached blood vessels from one compartment to another. These alterations are the most common secondary effects of expanding intracranial processes. Based on the site and direction of the shift, they can be divided into subfalcian, transtentorial (ascending and descending), cerebellar tonsillar and transphenoid (ascending and descending) in type. Patients with internal cerebral herniations are usually in compromised clinical states that only permit the use of axial CT acquisitions, which are not the most suitable for viewing craniocaudal shifts of the cerebral parenchyma. Therefore, due attention

must be paid to gleaning the indirect signs of internal cerebral herniation (3, 20).

Subfalcian and descending transtentorial herniations are the most common subtypes. A subfalcian herniation is defined by a shift of the cingulate gyrus across the midline, traversing below the free margin of the falx cerebri. As the shift progresses, the compressed ipsilateral cerebral ventricle becomes thinner, while the contralateral ventricle dilates as a consequence of CSF obstruction at the level of the foramen of Monro (Fig. 2.19). In addition, distal branches of the anterior cerebral artery are also shifted towards or across the midline, and, in the most severe cases these vessels can be compressed against the free edge of the falx cerebri; this in turn may result in secondary ischaemia or infarction due to pressure-occlusion of the pericallosal or callosomarginal arteries.

Descending transtentorial herniations consist of a medial and caudal shift of the uncus and the parahippocampal gyrus of the temporal lobe beyond the free margin of the tentorium cerebelli. This results in an asymmetric appearance of the peripontine cisterns and the cerebellopontine angle, which are widened on the side of the mass lesion due to a contralateral shift of the brainstem; the contralateral cisterns are consonantly narrowed by both the lateral shift as well as the downward herniation of cerebral tissue. The anterior choroid, posterior communicating and posterior cerebral arteries are also displaced medially and downward and can be compressed against the free edge of the tentorium cerebelli with resulting ischaemia or infarction of the occipital lobe if severe. In rare cases, compression of the perforating vessels emerging from the arterial circle of Willis can cause ischaemia and infarction in the basal cerebral nuclei. Other possible complications of transtentorial herniations include periaqueductal brainstem necrosis, brainstem haemorrhage (i.e., Duret haemorrhages) and direct contusion of the cerebral peduncle(s) due to traumatic impact against the free edge of the tentorium cerebelli (i.e., Kernohan's notch) (20).

Ascending transtentorial herniations are more rare, and are defined by the cranial shift of the cerebellar vermis and parts of the superior-medial aspects of the cerebellar hemispheres through the tentorium incisura. This in turn results in compression of the superior cerebellar and superior vermian cistern and the upper fourth ventricle. If severe, hydrocephalus may develop due to the compression of the aqueduct of Sylvius.

Tonsillar herniations are usually caused by an increase in mass effect within the posterior fossa, which causes a downward displacement of the cerebellar tonsils through the foramen magnum. It is estimated that up to half of all descending transtentorial herniations and approximately two-thirds of ascending transtentorial herniations are associated with tonsillar herniations at some point in the evolution of the herniative process.

Descending transphenoid herniations are produced by a posterior and downward (caudal) shift of the frontal lobe beyond the margin of the greater wing of the ipsilateral sphenoid bone, with backward displacement and compression of the Sylvian fissure, the middle cerebral artery and the temporal lobe on the same side. Conversely, in ascending transphenoid herniations, the frontal lobe is pushed upwards and anteriorly, to extend above the margin of the greater wing of the sphenoid.

Posttraumatic diffuse cerebral oedema

Diffuse cerebral oedema with generalized swelling of the brain occurs in up to 10-20% of all severe head injuries; this is encountered more commonly in children and can be either unilateral or bilateral (Figs. 2.24, 2.25). Unilateral diffuse cerebral oedema is associated with ipsilateral subdural haematoma formation in 85% of cases and with epidural haematoma in 9% of cases; it is an isolated finding in only 4-5% of cranial trauma patients (3, 20). Despite the fact that it can develop in just a few hours in the most serious cases, it usually evolves over a period of 24-48 hours. Posttraumatic diffuse cerebral oedema is caused by an increase in the water content of the brain and/or an increase in intravascular blood volume, both of which can

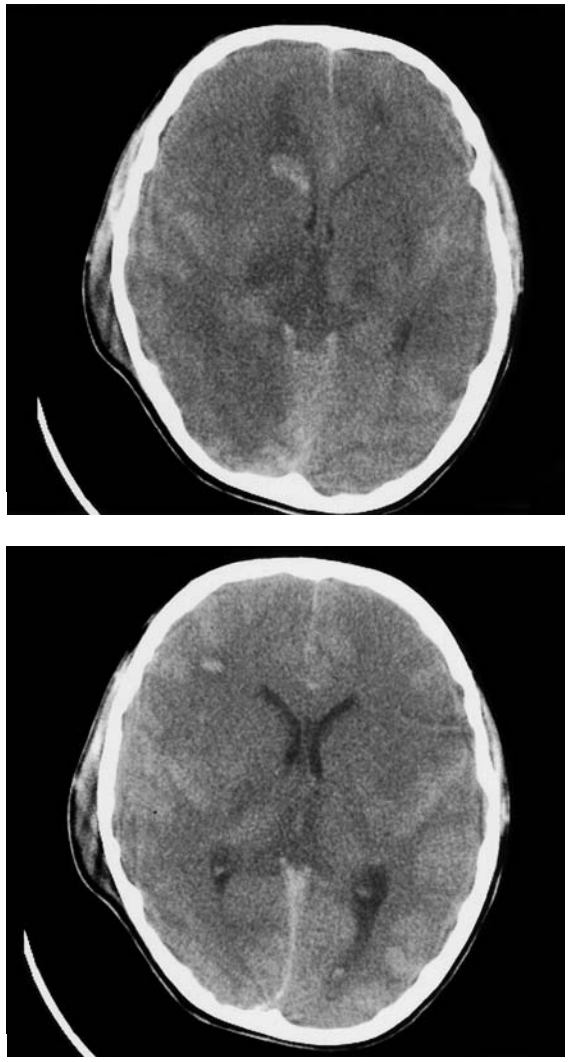

Fig. 2.25 - Posttraumatic cerebral oedema. The CT reveals posttraumatic cerebral oedema is observed associated with diffuse hypodensity of the white matter and obliteration of the Sylvian and perimesencephalic cisterns, a reduction in the size of the ventricular system and small intraparenchymal right haemorrhages in the right frontal region.

be precipitated by a number of factors. This is a severe clinical condition and is fatal in approximately 50% of cases (20). The CT picture is characterized by a generalized obliteration of the cerebral cortical sulci and the intracranial subarachnoid spaces of the suprasellar and perimesencephalic cisterns (e.g., ambiens and quadrigeminal), and the cerebral ventricles appear thinned and compressed.

The brain appears diffusely hypodense, with a loss of the distinction of the grey-white matter interface. The cerebellum is generally spared and can appear relatively hyperdense as compared to the cerebral parenchyma which is isodense. In the later stages of evolution in severe cases, diffuse cerebral oedema is often accompanied by transtentorial internal cerebral herniation (3, 20, 22).

Posttraumatic cerebral ischaemia and infarction

The herniation of brain parenchyma across the falx cerebri and through the tentorium cerebelli is the most common cause of posttraumatic infarction. The occipital lobe is the territory most frequently affected by ischaemic events, which are usually associated with herniation of the temporal lobe through the tentorial incisura, with consequent compression-occlusion of one or both of the posterior cerebral arteries (3, 20). The second most common area of infarction associated with cranial trauma is the vascular territory of the anterior cerebral branches contralateral to the traumatic mass lesion, to include the pericallosal and callosomarginal arteries, consequent to subfalcian herniation of the cingulate gyrus. More rarely, infarcts may occur in the basal ganglia due to the compression of the choroid, lenticulostriate and thalamoperforating arteries against the structures at the base of the skull (7, 29).

Another category of important secondary manifestations of brain injuries is that of posttraumatic haemorrhages related to direct injury of larger arteries and veins. The caudal shift of the upper portion of the stem in transtentorial herniations can cause a compression of the perforating vessels in the interpeduncular cistern. This in turn causes small haemorrhagic foci, which can be confluent, in the tegmen (i.e., Duret haemorrhage), which must not be confused with the rarer primary haemorrhagic contusion lesions in the dorsolateral portion(s) of the midbrain resulting from collision of the brainstem with the free edge of the tentorium cerebelli. As an additional factor in descending transtentorial herniations, the impact of the cerebral peduncle contralateral to the traumatic mass lesion(s) against the free edge of the

tentorium cerebelli may cause oedema, ischaemia and/or haemorrhagic necrosis of this structure which results in focal atrophy that may take the gross form of a notch (i.e., Kernohan's notch). Clinically this may result in hemiparesis ipsilateral to the side of the primary traumatic effects; this is known by the term "false localizing sign" because it occurs in the peduncle contralateral to the supratentorial traumatic mass effect (20).

POSTTRAUMATIC SEQUELAE

The most common sequelae of severe cranial trauma include cortical atrophy, encephalomalacia, pneumocephalus, CSF leaks (i.e., fistulae), leptomeningeal cysts, cranial nerve lesions and diabetes insipidus (Tab. 2.8).

Encephalomalacia is characterized by foci of cerebral parenchyma loss in the area of the contusion and by diffuse cortical atrophy (3, 20). Encephalomalacic foci appear on CT as hypodense areas, often associated with varying degrees of dilation of the adjacent cerebral ventricles and overlying cortical sulci.

Skull base fractures with interruption of the dura resulting in direct communication of the cranium with the paranasal sinuses, can lead to intracranial air collection(s) (i.e., pneumocephalus). This is easily detected using CT because of the extremely low attenuation coefficient of air (Figs. 2.3, 2.22) (8). Air limited to the epidural space tends to remain localized and does not vary in position with the placement of the head; conversely, air localized to the subdural space tends to move its principal focus with head movements (20). Subarachnoid air is typically multifocal, non-confluent, has a "bubble-like" appearance and is often localized within the cerebral sulci. Posttraumatic intraventricular air occurs in association with fractures at the base of the skull with lacerations of the dura mater. Intravascular air is only rarely observed and is usually detected only in cases of fatal trauma.

CSF leaks are a consequence of fractures of the base of the skull in 80% of cases (20). Typically they are frontally positioned with CSF draining via a fistula into the ethmoid or the sphenoid paranasal sinus, and in 20% of cases they are complicated by meningitis, which if untreated can in turn lead to the formation of cerebral abscess or extraaxial empyema (20). Clinical onset of a posttraumatic CSF leak usually occurs within a week of the initial trauma, but can develop as late as several years after the event. High resolution coronal acquisition CT is the examination of choice to identify the associated skull base fracture, although the visualization of the fistula is often difficult or impossible to achieve. Positive contrast CT or MR cisternography may be required to prove the presence and pinpoint the location of the fistula preoperatively.

Cranial fractures can later cause leptomeningeal cysts. These cysts are typically limited to children, occurring months to years after the cranial trauma. They are associated with underlying meningeal lacerations and theoretically result from an interposition of meningeal tissue within the space of the fracture line of the overlying calvaria at the time of the traumatic event. Sometimes known as an "expanding posttraumatic fracture", CSF pulsations have been hypothesized to be the mechanism of cyst accumulation as well as fracture expansion.

Diabetes insipidus is an infrequent sequela of cranial trauma, most commonly seen in infants as a result of birth trauma. Diabetes insipidus can be a direct result of either descending transtentorial herniation causing hypothalamic infarction or pituitary stalk transection occurring at the time of the traumatic event.

Posttraumatic paralysis of one or more of the cranial nerves, especially the second, third, fourth and sixth nerves and the second division of the fifth cranial nerve, are typically due to cranial base fractures that involve the cavernous venous sinus and the apex of the orbit. The third cranial nerve can also be affected individually by transtentorial herniation of the temporal lobe, whereas the fourth cranial nerve can be injured by compression against the free margin of the tentorium cerebelli during violent shaking movements of the head.

One final sequela to cranial trauma is hydrocephalus, usually secondary to intraventricular

haemorrhage or traumatic adherence of the meninges over the cerebral convexity, the basal cisterns or the aqueduct of Sylvius. This is caused by an inflammatory meningeal reaction to the effects of the trauma and the presence of blood products with consequent defective CSF resorption.

CONCLUSIONS

The advent of CT and its progressive technological improvement have revolutionized the diagnosis and clinical management of acute cranial trauma patients, resulting in early accurate analysis and swift evidence-based treatment of potentially fatal head injuries. Unenhanced CT is the examination technique of choice in these cases as it is quickly accomplished, readily available and does not require ancillary studies using other imaging technologies in most cases.

The spiral (helical) CT technique is principally useful in those cases in which the examination must be performed within an extremely limited time frame and in cases in which three-dimensional acquisition is necessary for multiplanar reconstruction of fractures of the orbit and the facial skeleton or CT angiographic studies of the intracranial vessels. Otherwise, standard CT acquisitions are preferred for their accuracy and absence of artefacts.

The use of IV contrast media is restricted to those rare cases in which a CT angiographic examination is needed when posttraumatic vascular pathology is suspected. Intrathecal positive contrast cisternography coupled with either CT or MRI may be required to analyse the presence and focus of a CSF fistula preoperatively.

One important limitation of the use of CT is the difficulty in detecting small parenchymal lesions located in the posterior fossa or at base of the skull, principally because of beam hardening artefacts typically present. However, this problem is of little practical significance clinically in the acute stage of trauma, as such pathology seldom requires surgical intervention.

It should be noted that MRI is more sensitive than is CT in detecting small cortical contusion lesions at the grey-white matter interface, DAI, extracerebral haematomas (especially when hypodense) and primary and secondary brainstem lesions. However, these changes are relatively minor and do not usually demand operative therapy in the emergency time frame. On the other hand, MRI can be a useful complement in the acute phase of cranial trauma in patients with significant clinical findings but no or few CT observations. And finally, MRI is the technique of choice in the evaluation of the subacute and chronic phases of symptomatic head injury.

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