After having discussed that partial volume effects due to asymmetric projections (i.e., 55b in Fig. 54.2b) may be misinterpreted as acute hematomas, this chapter will point out the characteristics of the different types of intracranial hemorrhage.

**Bleeding Caused by a Contusion**

As a direct consequence of skull trauma, cerebral contusion bleeding may occur (Fig. 54.1a). An acute hemorrhage (8) appears as a hyperdense mass which may be accompanied by surrounding edema (180) and displacement of adjacent brain tissue. In anemic patients the hematoma is less dense and may therefore appear isodense to normal brain.

If the vascular wall is damaged only secondarily by hypoperfusion mediated by edema, hemorrhage may not occur until hours or, more rarely, days after skull trauma. A CCT obtained immediately after skull trauma which does not show any pathologic changes is therefore not a good predictor since delayed cerebral bleeding cannot be ruled out. A follow-up scan should be obtained if the patient’s condition deteriorates. After complete resorption of a hematoma (Fig. 54.2a), a well-defined defect isodense with CSF remains (132).

Contusion frequently leads to an epidural, subdural, or subarachnoid hemorrhage and may leak into the ventricles (Fig. 55.1a). Possible complications of such leakage or of a subarachnoid hemorrhage are disturbed CSF circulation caused by obstruction of the pacchionian granulations, the foramen of Monro, or of the 4th ventricle. An hydrocephalus with increased intracranial pressure and transtentorial herniation of the brain may result.

Epidural and subdural hematomas can also lead to major displacement of brain tissue and to midline shifts. Quite frequently this in turn causes obstruction of the contralateral foramen of Monro resulting in unilateral dilation of the lateral ventricle on the side opposite the bleeding (Fig. 56.3). The characteristics useful in differential diagnosis of the various types of intracranial bleeding are listed in Table 54.1.

<table>
<thead>
<tr>
<th>Type of bleeding</th>
<th>Characteristics</th>
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<tbody>
<tr>
<td>Subarachnoid bleeding</td>
<td>Hyperdense blood in the subarachnoid space or the basal cisterna instead of hypodense CSF</td>
</tr>
<tr>
<td>Subdural bleeding</td>
<td>Fresh hematoma: crescent-shaped, hyperdense bleeding close to the calvaria with ipsilateral edema; hematoma is concave toward hemisphere; may extend beyond cranial sutures</td>
</tr>
<tr>
<td>Epidural bleeding</td>
<td>Biconvex, smooth ellipsoidal in shape; close to calvaria; does not exceed cranial sutures; usually hyperdense, rarely sedimented</td>
</tr>
</tbody>
</table>

**Table 54.1**
If there is intraventricular extension of intracranial hemorrhage (Fig. 55.1a), physiologic calcification of the choroid plexus (123), in the lateral (133) and 3rd ventricles (134), as well as those of the habenulae and the pineal (148), must be distinguished from fresh, hyperdense blood clots (8). Please note the edema (180) surrounding the hemorrhage (Fig. 55.1a).

If the patient has been lying supine, a horizontal fluid–fluid level caused by blood sedimenting in the posterior horns of the lateral ventricles may be seen (Fig. 55.2a). The patient is in danger of transtentorial herniation if the ambient cistern is effaced (Fig. 55.2b). In this case the 3rd ventricle is completely filled with clotted blood (🩹 in Fig. 55.2a, b), and both lateral ventricles are markedly dilated. CSF has leaked into the paraventricular white matter (🩹).

In addition, a lower section of this patient shows subarachnoid hemorrhage into the SAS (🩹, 🩹 in Fig. 55.2b).

Subarachnoid Hemorrhage
An obstructive hydrocephalus, as caused by subarachnoid hemorrhage (8 in Fig. 55.3a, b), may easily be identified because the temporal horns (133) of the lateral ventricles appear distended. In such cases it is important to have a closer look at the width of the SAS over the cerebral surface: blunted cerebral gyri usually indicate a diffuse cerebral edema. In the present case though, the width of the Sylvian fissure (127) and the surface SAS are normal. Acute edema is therefore not present (yet).
Since the surface SASs are very narrow in younger patients, it is possible to miss a subarachnoid hemorrhage in children. The only identifiable sign may be a small hyperdense area adjacent to the falx (130). In adults a small subarachnoid hemorrhage also causes only a minor, circumscribed area of hyperdensity (8 in Fig. 56.1a). At the time of this CT scan the bleeding was so slight that it had not yet caused any displacement of brain tissue.

**Subdural Hematoma**

Bleeding into the subdural space results from cerebral contusions, damaged vessels in the pia mater, or from torn emissary veins. The hematoma initially appears as a long, hyperdense margin close to the skull (8 in Fig. 56.2a). In contrast to an epidural hematoma, it is usually somewhat irregular in shape and slightly concave toward the adjacent hemisphere. This kind of bleeding is not confined by cranial sutures and may spread along the entire convexity of the hemisphere.

Subdural hematomas can also cause marked displacement of brain tissue (Fig. 56.3a) and lead to disturbances in CSF circulation and to incarceration of the brain stem in the tentorial notch. It is therefore not as important, for treatment purposes, to distinguish between a subhematoma or an epidural hematoma as it is to ascertain the extent of the hemorrhage. Hematomas with the propensity to expand, especially if edema is a threat, should therefore be drained or treated surgically.

Chronic subdural hematomas (8 in Fig. 56.3a) may appear homogeneously hypodense or show inhomogeneous density with sedimentation of blood. The danger involved in a small, venous bleed is the symptom-free interval and the slow onset of somnolence up to the development of a coma. Therefore, a patient with suspected bleeding after cranial trauma should always be kept under observation in order to detect any clinical deterioration.
Extradural Hematomas

Bleedings into the extradural spaces are usually caused by damage to the middle meningeal artery, and rarely by venous bleeding from the sinuses or the pachyionian bodies. Predisposed areas are temporoparietal regions or sometimes the posterior cranial fossa, in which case there is severe danger of tonsillar herniation. Arterial hemorrhage lifts the dura from the inner surface of the cranium (55) and then appears as a biconvex, hyperdense area with a smooth border to the adjacent hemisphere. The hematoma does not extend beyond the sutures between the frontal (55a), temporal (55b), parietal (55c), or occipital (55d) bones. In small extradural hematomas (8) the biconvex shape is not distinct (Fig. 57.1a), making it difficult to differentiate the finding from a subdural hematoma.

It is important to distinguish between a closed skull fracture with an intact dura, and a compound skull fracture with the danger of secondary infection. An unequivocal sign of a compound skull fracture (Fig. 57.2a) is the evidence of intracranial air bubbles (4), which prove that there is a connection between intracranial spaces and the paranasal sinuses or the outside. It is difficult to determine whether the bilateral, hyperdense hematomas (8) in Figure 57.2 are extradural or subdural. In this case the distortion of the midline was caused by the right-sided, perilesional edema (left side of Fig. 57.2a) since it was shifted toward the left (the side of the hematoma).

Test Yourself! Exercise 8:

When looking at the image of another patient (Fig. 57.3), you will note several pathologic changes. Use the free space below the picture to note how many different types of bleeding (if any) you can distinguish and what other pathology/complications you suspect. You will find the answers at the end of the book, but remember: be a good sport and don’t cheat, think first!
Apart from cardiovascular and malignant diseases, cerebral infarctions are among the most frequent causes of death. A thrombus occludes a cerebral artery, which leads to irreversible necrosis in the area of blood supply. Vascular occlusion develops in association with atherosclerotic changes of cerebral arteries or, less frequently, as a result of arteritis. A further cause are blood clots from the left heart or thrombotic plaques from the carotid bifurcation which embolize into a cerebral vessel.

In case of embolization, diffusely situated, small, hypodense zones of infarction in both basal ganglia and hemispheres are typical. Old emboli result in small, well-defined areas (180) which eventually appear isodense to the CSF (132). Such areas are called lacunar infarcts (Fig. 58.1a). A diffuse pattern of defects calls for color flow Doppler imaging or carotid angiography and an echocardiogram to exclude atrial thrombus.

Please remember that in a suspected stroke it might take up to 30 hours to distinguish clearly the accompanying edema as a hypodense lesion from unaffected brain tissue. A CT scan should be repeated if the initial scan does not show any pathologic changes even though the patient is symptomatic and if symptoms do not resolve (resolution of symptoms points to a transient ischemic attack, TIA). In case of a TIA: no abnormalities are visible in the CT scan.

In contrast to the TIA, the prolonged reversible ischemic neurologic deficit (PRIND) is often associated with hypodense zones of edema in the CT scan.

If the area of infarction corresponds to the distribution of a cerebral artery, one should consider an occlusion of the corresponding blood vessel. In classical infarctions of branches of the middle cerebral artery, ischemia will cause a hypodense area of edema ( in Fig. 58.2a).

Depending on the size, the infarction may have severe mass effect and cause midline shift. Smaller areas of infarction do not usually show any significant midline shift. If the arterial walls are damaged, bleeding may occur and appear as hyperdense areas coating the neighboring gyri.

The unenhanced follow-up CT scan in Figure 58.2b shows an additional bleed into the head of the right caudate nucleus ( ) and right putamen ( ). In this case the infarction is 2 weeks old and necrotic tissue has been mostly resorbed and replaced by CSF.
Whereas differential diagnosis (DD) of intracranial hemorrhage and infarction may be obtained without the use of CM, detection of cranial metastases (7) is definitely improved by the administration of i.v. CM. Even small areas in which the blood-brain barrier is disturbed become visible (Fig. 59.1a). Large metastases sometimes cause surrounding edema (180) which could be misinterpreted as infarct-related edema on unenhanced images if the metastasis appears isodense to the adjacent tissue. After i.v. CM the lesion in the left hemisphere (7) is clearly demarcated (Fig. 59.2a). Did you also spot the second, smaller metastasis within the right frontal lobe, which also shows some surrounding edema (180)?

The differential diagnosis of brain tumors is made much easier by the injection of i.v. CM. In the unenhanced image (Fig. 59.3a), the temporoparietal glioblastoma on the left (7) which has a central necrosis (181) could have been mistaken for cerebral infarction. The post-CM image, however, reveals the typical appearance of a glioblastoma with an irregular rim enhancement of its margin (Fig. 59.3c).
Another example of the advantages of i.v. CM is the demonstration of inflammatory processes, since the accompanying defect in the blood-brain barrier will not show on an unenhanced image. Figure 60.1a shows hypodense edema \((\text{n})\) in an unenhanced section of a patient suffering from aortic valve endocarditis. Contrast medium (Fig. 60.1b) confirmed the finding by enhancing the inflammatory process \((\text{k})\). Bacteria from the aortic valve caused this septic embolism in the left occipital lobe.

Inflammation of the paranasal sinuses and of the middle ear can already be diagnosed in native images as effusions (8), for example in the normally air-filled mastoid cells (62). Swelling of the mucous membranes of the external auditory canal (63b) is visible without the need for CM. Figure 60.2a shows bilateral otitis externa and media, which is more severe on the right side where it involves the antrum and the mastoid cells. With progressing abscess formation, an image on bone windows should be obtained in order to detect possible bone erosion.

A retention cyst, which often appears in one of the paranasal sinuses, should be considered in the differential diagnosis of advanced inflammations. They typically have a broad base on the wall of a paranasal sinus, extend into its lumen, and have a roundish convex shape (\(\text{p}, \text{j}\) in Fig. 60.3).

Such cysts are only of significance if they obstruct the infundibulum (1) of the maxillary sinus or the semilunar canal (2), causing an accumulation of secretions. In patients with chronic sinusitis, it is therefore important to check for an unobstructed lumen of the semilunar canal (2) or for variations which may restrict mucociliary transport of secretory products.

Haller’s cells (\(\text{l}\)), a pneumatized middle concha (166), and a pneumatized uncinate process (3) are among the most frequent variations. All of these variations can obstruct the semilunar canal and cause chronic, relapsing sinusitis.
You have already seen pathologic changes in the lacrimal gland (pp. 39/40) and the CT morphology of an eye prosthesis (p.51). Every mass within the orbit should, of course, be diagnosed early and treated effectively because of the possibly severe consequences to vision. In order not to miss tumor invasion into the walls of the orbit, bone windows should also be obtained. In Figure 61.1a there is a hemangioma (7) within the retrobulbar fat (2), which is not necessarily an indication for operation because of its benign character. In this case it causes a minor proptosis.

Endocrine Ophthalmopathy
Minimal discrete changes can be missed during the reporting of a CT scan: endocrine ophthalmopathy often appears as part of Graves’ disease and can, in its early stage, only be diagnosed on the basis of a thickening of the external ocular muscles, e.g. the inferior rectus muscle (47b in Figs. 61.2a, 61.3a).

Myositis should be considered in the differential diagnosis. If this early sign is not detected, the disease of the orbital tissue, which is most probably an autoimmune disease, may progress in the absence of therapeutic intervention. Therefore, you should always examine the symmetry of the external ocular muscles (47) when looking at an orbital CT scan.

There will often be a typical temporal pattern of involvement. The first finding is an increase in the volume of the inferior rectus muscle (47b). The disease will continue and affect the medial rectus muscle (47c), the superior rectus muscle (47a), and finally all the other external ocular muscles.
In contrast to benign retention cysts (p. 60), malignant tumors of the paranasal sinuses often lead to destruction of the facial bones and may invade the orbit, the nasal cavity (77), or even the cranial fossa. It is therefore useful to examine both the soft tissue and bone windows. For planning a resection, different CT planes might be necessary. The following example shows a tumor of the paranasal sinuses (7) in an axial (Fig. 62.1a) and a coronal view (Fig. 62.2a). Originating from the mucous membranes of the right maxillary sinus (75), the tumor has infiltrated the nasal cavity (77) and the ethmoid cells.
The most common reason for doing a coronal CT scan is, apart from determining the extent of chronic sinusitis, the diagnosis of fractures: in fractures of the orbital floor (Figs. 63.1a, 63.2a) any accompanying herniation of retrobulbar fat (2) or the inferior rectus muscle (47b) into the fracture site (●) or even into the subjacent maxillary sinus (75) should be determined preoperatively. Diagnosis of the fracture in Figure 63.2a is easier because there are dislocated bone fragments. In addition, it is important to detect indirect signs of fracture, such as very fine, step-like contours of the bones and secondary bleeding (8) into the nasal cavity (77) or the frontal (76) and maxillary sinuses (75). Another important question is whether or not the head of the mandible (58a in Fig. 63.3a) is fractured or the maxillary bone (57) has been fractured and displaced (●) from the sphenoid (60) bone (Fig. 63.4a). In this case, severe bleeding (8) required intubation (182) and a nasogastric tube (182).

Fractures of the facial skull (Le Fort [33])

Type I: Straight across the maxillary bones and the maxillary sinuses (Guérin’s fracture)

Type II: Across the zygomatic process of the maxilla, into the orbit, and through the frontal process of the maxilla to the contralateral side; maxillary sinus not involved

Type III: Involving the lateral wall of the orbit and the frontal process of the maxilla to the contralateral side; ethmoid cells and zygomatic arch usually involved, sometimes also affecting the base of the skull.