ports, a cerebellar syndrome was the most common (12 patients). The pancerebellar syndrome following heatstroke is attributed to a direct thermal effect on the cerebellum resulting in degeneration of Purkinje cells (Yaqub 1987). Computed tomography and magnetic resonance imaging (MRI) studies have also revealed cerebellar atrophy and white matter involvement (Mehta and Baker 1970). However, in various cases, the cerebellar atrophy was noted on MRI after 10 weeks, being progressive for 1 year (Albukrek et al. 1997), or appeared 1 year later and became more marked at 2 years (Biary et al. 1995).

12.1.3 Trauma by Fire and Burns

12.1.3.1 Incidence

Death from fire that results directly or indirectly from the effects of flames and/or heat on the brain is extremely rare. In most cases, acute death is primarily caused by shock or inhalation of poisonous fumes, especially of carbon monoxide, before the brain can be affected by flames or heat. A delayed death, on the other hand, is caused by protein loss, edema, renal failure, and/or secondary infection.

The mortality rate from burns depends in large part on three factors: the age of the victim, the severity and extent of the burns, and the intensity and composition of the fumes. Mortality can be depicted by scatter diagrams showing survivors and fatalities plotted against age and the percentage of full-thickness burn. Elderly victims may die of chronic organic diseases in combination with burns, e.g., from stroke.

If the victim suffers only burning of the skin of the head, which comprises 9% of the body surface, the aforementioned correlation between age and percentage of full thickness burn would indicate that such an injury is compatible with survival almost regardless of age, provided other aggravating factors are excluded.

12.1.3.2 Clinical Features

As mentioned above, injury and acute death result primarily from *asphyxia* or *inhalation of poisonous fumes* rather than from the effect of flames on the brain. The relationship between the duration of exposure to noxious gases or oxygen depletion and their effects was summed up by Davies (1991, Table 14.2). The fumes, especially carbon monoxide (CO-intoxication, see pp. 347 ff), induce coma. In most cases, the intoxication itself proves fatal or the coma-induced immobility leads to death from the flames and heat. If more than 40% of hemoglobin (carboxyhemoglobin level) is bound to CO, the victim may fall into a coma and if more than 60% is bound, the victim will die.

In survivors, the heat as well as inhaled poisons can cause *inhalation injury* of the upper respiratory tract and lungs, with loss of surfactant-associated proteins and injury of type II pneumocytes. A *shock phase* develops if the victim survives the primary injury of heat, fire, fumes, and inhalation injury. The severity of shock symptoms depends on the extent of skin burned, not the depth of burning. The factors underlying the grave circulatory and metabolic disorders as a result of burn injury are gradually being identified. Distant effects of thermal injury of the skin include intravascular hemolysis, vascular permeability, acute lung injury, renal failure, and impaired liver function. Both primary fume-induced CNS intoxication and indirect sequelae such as hypoxia due to inhalation injury or shock can lead to changes in the brain.

12.1.3.3 Neuropathology

In cases of acute death as a consequence of CO intoxication, signs of CO poisoning predominate, with macroscopically evident bright red coloration in formalin-fixed brain sections (Fig. 17.3). Postmortem alterations, such as coagulation of the brain surface or of the entire brain, are also visible (Fig. 12.1c, d), especially if the cranium has lost all of its soft tissue or has burst from the heat (Fig. 12.1a, b). The brain may be shrunken, although the structures of the gray and white matter can still be differentiated. The brain is partially or wholly dehydrated (Dotzauer and Jacob 1952; Klein 1975) and coagulated. These changes, like the accumulation of blood in the epidural space (burn hematoma – Fig. 12.1e, f), occur postmortem (see below). The almost invariably demonstrable high carboxyhemoglobin levels of >50% indicate that the victim had already died of CO poisoning before heat affected the brain (Gerling et al. 2000; Oehmichen 2000a).

The morphological differentiation between intravital and postmortem injuries may be difficult as demonstrated in Fig. 12.2. Similarly, proof of epidural and subdural accumulation of blood as well as the demonstration of herniation, of cortical hemorrhages in the frontal base of the brain and the pons give evidence of a vital mechanism, while burn hematoma and brain surface coagulation are postmortem alterations.

If burns are survived, secondary phenomena will result from the fire-induced burn wounds and/or the wound healing process. Survival depends on the severity of the burns, i.e., on the extent of skin surface

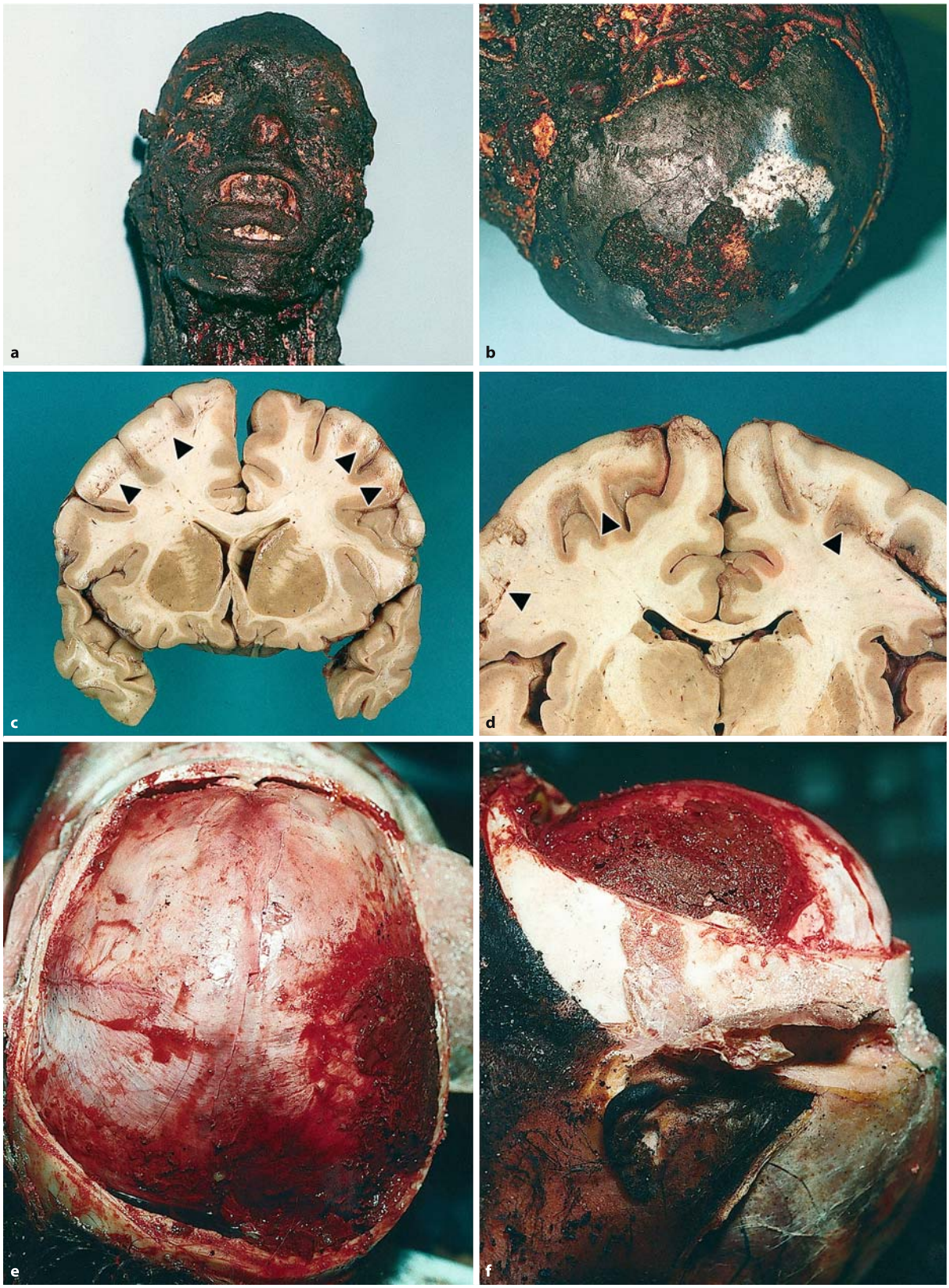

Fig. 12.1a–f. Postmortem alterations as a result of fire and burning on scalp, skull and brain. **a**, **b** Loss of hairs, cutis, and soft tissue, i.e., loss of total the scalp, as well as fracture and loss of parts of the external table of the skull; **c**, **d** coagulation of the brain surface (arrowheads) **e**, **f** accumulation of blood in the epidural space = burn hematoma

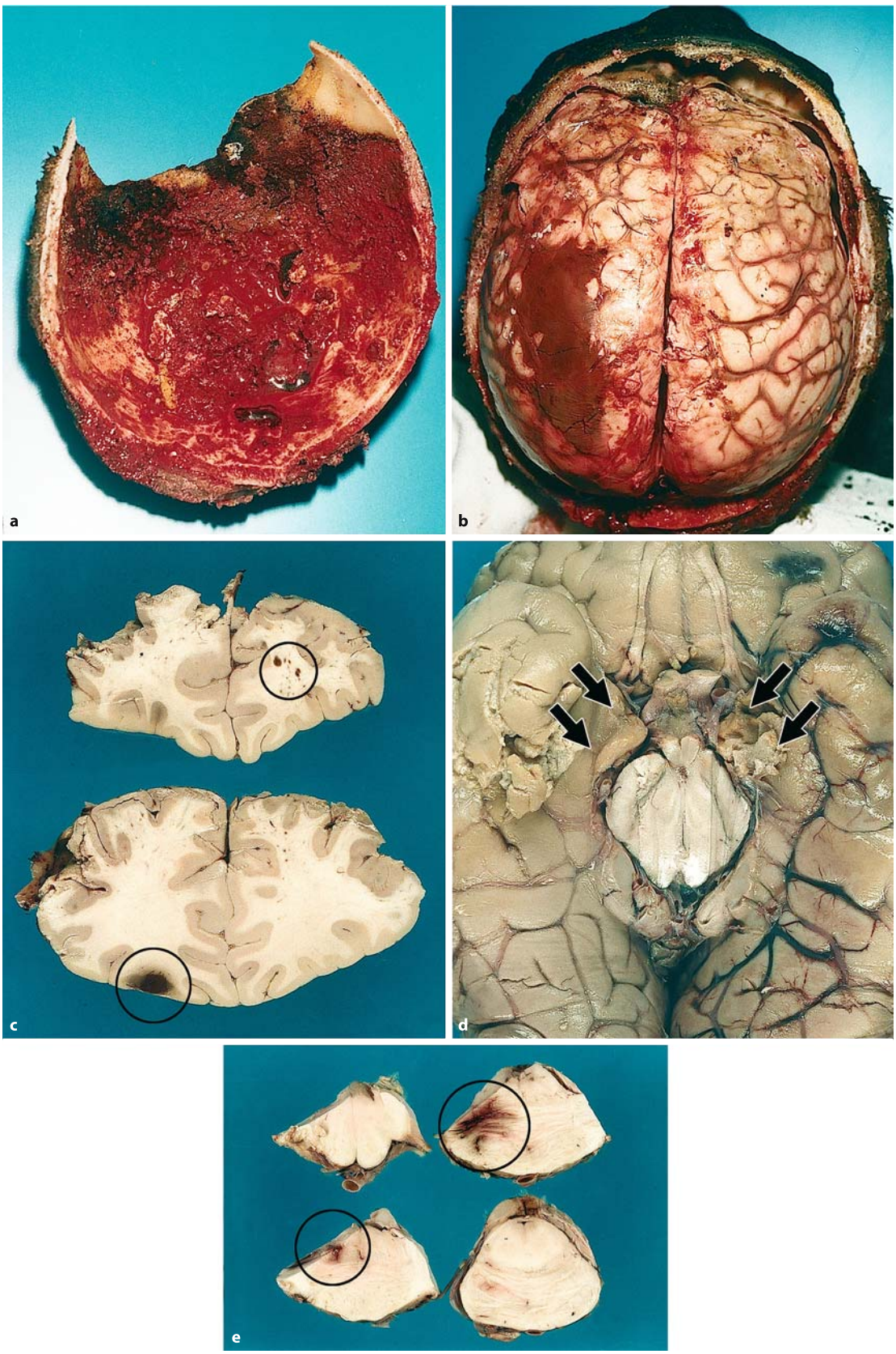

Fig. 12.2a–e. Differentiation between intravital and postmortal alterations. **a** Postmortem (extradural) burn hematoma; **b** intravital "subdural" hemorrhage; **c** intravital white matter and cortical hemorrhages (*circles*); **d** intravital herniation (*arrows*); **e** intravital pontine hemorrhage (*circles*)

and soft tissue destruction, and on the severity of the CO-induced brain damage.

Clinically, burn victims who survive only briefly (*early death*) develop a clouding of consciousness, often lapsing into coma. The morphological picture is characterized by hyperemia and edema secondary to the altered hemodynamics and toxic vessel damage (Hagedorn et al. 1975). The simultaneous increase in the permeability of the blood–brain barrier to plasma proteins causes inflammation with intravascular thromboses and sometimes also gives rise to intravascular fibrin bodies secondary to shock (Hagedorn et al. 1975). Perivascular spongiform disintegration of myelin, perivascular aggregation of lipid-containing and hemosiderin-containing macrophages, cerebral hemorrhages, nerve cell shrinkage, pallor and astroglial swelling and proliferation have all been described (Jacob 1955).

Delayed death from burn injuries is caused mainly by infections of burned skin areas and airways. Extravascular water retention increases throughout the body (including the brain) secondary to a pathologic increase in the permeability of vessel walls and enhanced loss of plasma proteins. The result is both generalized edema and brain edema, which may be potentiated by systemic hypoxia. Common delayed complications of burn injury include CNS infection with sepsis (15% of cases), secondary changes (septic arterial occlusion or DIC), which induce infarction (18% of cases), and intracerebral hemorrhage (Winkelmann and Galloway 1992). In cases with long survival times, the clinical picture is dominated by the ischemic injury. Internal hydrocephalus has the clinical features of an encephalopathy, which is seen mainly in children.

The Problem of Vitality. In cases of acute death, it must always be determined whether the victim was already dead or still alive at the time of the fire. This question can usually be answered at autopsy by demonstration of soot in the airways and elevated CO-Hb levels in the blood.

In some cases, signs of vital processes may be lacking, an indication that death did not result from the fire. The neuropathologist must then help to establish the exact cause of death and find signs of whether, for example, the fire was intentionally set to conceal a prior homicide (e.g., by strangulation, asphyxia, MBI, etc.). Two frequent morphological findings are of importance in this respect:

1. *Skull fractures* and/or skull destruction (Fig. 12.1a, b): biomechanical analysis of the type of fracture can help to establish whether the dome of the skull burst as the result of a heat-induced increase in intracranial gas pressure (bursting fractures) or has broken as a consequence of a blow (bending fractures).
2. *Epidural hematoma* (Fig. 12.1e, f): a so-called burn hematoma must be differentiated from primary mechanically caused hemorrhage. If there are no signs of external mechanical violence to the head, and no secondary (vital) effects such as shifting of the brain, brain edema, or cell reactivity, then primary mechanically induced hematoma can be ruled out. There are, however, difficulties in the interpretation of findings in cases in which blow and fire injure the head at the same time or at only very brief consecutive intervals. In cases of burn hematomas the head has usually already undergone skeletonization, with incineration of the overlying soft tissues as well as heat-induced deformation of the inner table of the skull.

Local thermal changes in the brain are caused almost exclusively by electrically induced burn injuries. The effects of heat can cause considerable destruction of the scalp and skull at the point of contact, which is typical of electrical burns. Heat-induced local tissue necrosis is also seen in the underlying dura mater and brain tissue, especially in high voltage industrial accidents or as induced by lightning.

12.2 Electrical Trauma

12.2.1 Incidence

Deaths from electric violence are usually accidental, although occasionally they are associated with suicide or homicide. In the USA, about 1,000 deaths from electrical shock are reported annually (Lee 1997), another 100,000 injuries from electrical shock are survived (Mellen et al. 1992). In 1967, global rates of fatal electrical accidents per 100,000 inhabitants ranged from 0.13 in Northern Ireland to 0.76 in Italy (Wyzga and Lindroos 1999).

12.2.2 Clinical Features

The symptoms are determined by the type and the quantity of electrical current, its path through the body as well as its density, frequency, and duration. Even relatively weak intensity can cause acute symptoms such as paresthesias, muscular spasms and muscular pain, numbness of the limbs, somnolence, convulsions, and loss of consciousness (Panse 1955; Posner 1973; IEC 1987, 1994). The latter symptoms in particular, as well as persistent headache, nausea, and