31 Sudden Loss of Consciousness

31.1 Syncope

In most cases of sudden loss of consciousness, no recent history is available. Also, remote history which might provide a diagnostic clue is usually rather meager, as persons close to the patient are either too excited or uninformed.

31.2 Generalized Epileptic Seizure

The most common cause is one of the variants of syncope. These are discussed in detail in Chap. 32 in the context of sudden loss of posture. Frequently, not only posture but also consciousness is lost, even though only for a period of seconds. Prolonged unconsciousness is rare.

31.3 Intracerebral Hemorrhage

A postictal state is an apparently simple situation, but the seizure may have gone unnoticed or unreported for various reasons. It is difficult to imagine to what extent the family may be ashamed of their epileptic relative. The typical signs like bitten tongue or lips may be absent. Enuresis may have various causes. A postparoxysmal hemiparesis may mislead the diagnostician if the patient is beyond what is euphemistically called the prime of life (see Chap. 5). Elevation of creatine kinase provides a useful clue.

31.4 Subarachnoid Hemorrhage (SAH)

Intracerebral hemorrhage usually occurs in chronically hypertensive patients. The cause is rupture of a small arteriosclerotic aneurysm; preferred localizations are the basal ganglia, pons, and cerebellum. The patient is found somnolent or unconscious. He is likely to have hemiplegia, which can be assessed in the unconscious patient by unilateral loss of muscle tone. Proprioceptive reflexes may be diminished on the paralyzed side, but Babinski's sign will be present. In hemispheric hemorrhage there is frequently conjugate deviation of the eyes toward
the side of the brain lesion. In pontine hemorrhage there will be tetra­plegia with bilateral extensor reflexes and various oculomotor signs. If there is conjugate deviation of the eyes it is directed away from a unilateral pontine lesion, in contrast to hemispheric damage where it is directed toward the lesion (the hemispheric oculomotor system “pushes” the eyeballs to the contralateral side). “Swimming” conju­gate or disconjugate ocular movements are frequent and of little value for localizing lesions within the brain stem. Spontaneous nystagmus is likely to beat horizontally in pontine and vertically in midbrain le­sions.

*Ocular bobbing* is most frequently seen in lower brain stem compres­sion owing to a space-occupying lesion of the cerebellum. It is fre­quently, but not absolutely certainly, a sign of irreversible brain stem dysfunction. Attenuation of the oculocephalic reflex parallels depth of coma.

*Pupillary alterations* are frequent. Bilateral miosis indicates damage at the pontine level, if the reaction to light is preserved, and this can sometimes be seen only with a magnifying glass. Unilateral mydriasis is seen when the third nerve nucleus or its autonomic efference in the midbrain tectum are affected. Bilateral mydriasis is an ominous sign. CSF is sanguinolent in most cases. Neuroimaging clearly shows the locus and extent of the hemorrhage and its possible space-occupying effect, which might necessitate neurosurgical intervention.

### 31.4 Subarachnoid Hemorrhage (SAH)

The diagnosis of SAH is discussed in Chap. 4. Here it is important that some patients are found unconscious after SAH. There is almost always neck stiffness, and lumbar tap will provide hemorrhagic CSF. Centrifugation is standard, because the most experienced doctor may tap a vein, thus causing an artificial admixture of blood to the CSF. Neuroimaging shows SAH, and may even suggest prognosis. Large blood clots give a warning that arterial spasms are to be expected during the next few days. Also, communicating hydrocephalus is recog­nized early by neuroimaging.

### 31.5 Basilar Artery Thrombosis

Thrombosis of the basilar artery rarely occurs without premonitory symptoms, which may have been present for several days. Patients are reported to have exhibited slurred speech, diplopia, and ataxia or paresthesia of the limbs. These warning symptoms usually fluctuate until there is sudden or rapidly evolving loss of consciousness. History taking is essential in these cases.