II. Stages of Anesthesia

Narcotic, analgetic, and neuroleptic substances used in anesthesiology for general anesthesia cause a continuous reversible suppression of all or individual structures of the CNS (holencephalic and telencephalic narcotics). The changes in the level of consciousness and in the vegetative functions during the induction, maintenance, and termination of anesthesia are at all times related to the actual depth of anesthesia. The various stages of anesthesia can thus be clinically classified, as was first described by Gueidel in 1920 [5]. In the course of anesthesia with holencephalic drugs, these stages are basically passed through in a similar sequence – analgesia with complete alertness, loss of consciousness, coma due to narcotic overdose, and intoxication. The time leading up to the individual stages and their duration depends on the type of narcotic agent and its specific appearance and disappearance in the CNS. In modern anesthetic techniques with combinations of several anesthetic-narcotic relaxing substances, the individual stages of anesthesia are frequently varied in their clinical manifestations and thus can be less easily distinguished than with the monoanesthetic formerly used.

Cerebral function is reflected in the EEG. Based on this fact, Gibbs et al. found in 1937 [4] that all anesthetics cause comparable EEG changes (today this is true for most anesthetics [9]). Thus, the depth of anesthesia can be determined by the EEG. The distinction of individual stages by EEG changes is much more exact than by clinically detectable changes. Based on the EEG classification of stages of sleep by Loomis et al. [10] and Gueidel’s clinical stages of anesthesia [5], Schneider and Thomalske [15], and similarly Gibbs and Gibbs (1951), introduced an EEG classification of anesthesia, which subsequently was made more precise by Martin et al. [11], Kugler (1981), Kubicki [6, 8], and Kubicki et al. [7] (Tables 1, 2).

The individual stages of anesthesia are defined by EEG characteristics, determined by the type and degree of the cerebral depression (Tables 1, 2).

In the course of anesthesia, one defined stage glides smoothly into the next. Accordingly, the corresponding EEG pattern does not show changes abrupt. Sometimes, the frequency patterns, which are typical for different anesthetic stages, are found simultaneously. The various levels of consciousness during anesthesia and the corresponding EEG characteristics are shown in Table 2. The dominant frequency in the alert patient (mainly alpha activity, see Chap. AIV) during induction is first lowered then suppressed completely. Simultaneously, or immediately afterward, cerebral
functions are activated. This is characterized by the appearance of fast waves and is clearly manifest by clinical signs of motor activity and psychic restlessness, or even excitation. Suppression, characterized by isolated slow waves, may also occur.

During the stage of somnolence, the slow frequencies become more marked; they form the pattern of so-called surgical anesthesia, when the anesthesia is further deepened. The stage of anesthetic coma is characterized by further deceleration of the frequency. With continuous medication of the anesthetic agent, the suppression—and suppression also of the deeper cerebral functions—is evident in electrocerebral silence (ECS), initially interrupted by bursts ("outbreaks of frequency"). These bursts become less frequent in the stage of complete anesthetic coma and eventually cease. The level trace in the EEG demonstrates the complete breakdown in brain function. This corresponds to the failure of all vegetative functions. At the same time, the tolerance limit for the anesthetic has been reached or exceeded. Even in the stage of complete anesthetic coma, a reversibility of the EEG changes and of the clinical condition is basically possible, however, this is dependent on the patient’s general condition, the rapidity of the cerebral recovery from the substance, and the duration of the complete failure of brain function, with its negative effects on circulation and respiration. During recovery from anesthesia, the stages and the clinical and EEG signs appear in the reverse sequence to that seen during the application of anesthesia. During recovery—especially after the application of barbiturates—light stages of narcosis and fluctuations in the physiological stages of sleep are observed both clinically and encephalographically (KUGLER 1981). During the postnarcotic phase, an increase in the beta frequencies and/or a decrease in the amplitude of the initially dominant frequency can be demonstrated, even in the clinically alert patient (KUGLER 1981 [13, 14]). During anesthesia, the individual variations in the initial EEG disappear; in the deep stages of anesthesia, any local differences in the cortical activity become less noticeable [11].

The ability to determine exactly the anesthetic depth by the encephalographically recorded course of cerebral function described above are basic requirements for an EEG monitoring of anesthesia. This monitoring should be simplified, both from a theoretical and practical point of view, by decreasing the number of leads and measured values. The reduction of the number of recording leads from 18 to 2 still permits sufficient general changes in cerebral function to be detected, which is essential during anesthetic monitoring. The most advanced degree of reduction in the number of EEG leads has been achieved with the so-called integrated EEG—cerebral function monitor (CFM) [12]. Approximate values are derived for the frequency and the amplitude of a recorded and filtered one-lead EEG, which is traced as the integration graph of the total cerebral activity. SCHWILDEN and STOECKEL [16], on the basis of their EEG studies, regard