Achieving adequate depth of anaesthesia during surgical procedures is desirable. While too deep anaesthesia, resulting in cardiovascular depression (easy to detect) and prolonged awakening times (a rather harmless complication) is of minor clinical interest, the opposite - too light anaesthesia – is more difficult to detect and frightening from the patients point of view.

Usually, an incidence of 1 to a few in 1000 general anaesthesia cases is reported with three major risk factors: trauma, caesarean section and cardiovascular surgery. The hereby increased incidences of intraoperative awareness are easy to re-construct. Under these circumstances light anaesthesia is intentionally put at risk in order to avoid severe cardiovascular depression or fetal impairment respectively. However, there is a risk for the anaesthesiologist too, since some of these patients take legal action. A closed claims analysis of more than 4100 anaesthesia related claims in the U.S.A. has shown that despite all advantages in modern anaesthesia there is an increasing incidence of claims concerning intraoperative awareness, 1% in the 1980th, 2% in the 1990th and 3% in the last decade [ii]. Surprisingly, risk factors for claims were totally different from the above cited “classical” risk factors: age at around 40 years, ASA physical status I or II, routine surgery and female gender.

In the 1930th Guedel [iii] proposed four stages of depth of ether anaesthesia, 1st stage - analgesia, 2nd stage – delirium, 3rd stage – surgical anaesthesia, 4th stage respiratory paralysis. Clinical observation of physical signs, such as somatic muscle tone, respiratory patterns and ocular signs allowed assigning to the stage of anaesthesia, true at least for ether anaesthesia. As long as a sole anaesthetic agent (i.e. ether, chloroform, ethylene) was used, depth of anaesthesia was dose-dependent.

The idea of depth of anaesthesia as an unique construct has been abandoned with the increasing number of drugs from different pharmacological classes (volatile anaesthetics, nitrous oxide, opioids, barbiturates or non-barbiturate hypnotics, benzodiazepines, phenothiazides, muscle relaxants, alpha-2-receptoragonists et.c.) with different pharmacological properties (analgesia, anxiolysis, amnesia, sedation, hypnosis, unconsciousness and suppression of somatic motor, cardiovascular and hormonal responses to surgery) [iv]. Depth of anaesthesia becomes more and more a multidimensional phenomenon.

In the era after the 2nd world war determining depth of anaesthesia focussed on suppression of somatic and autonomic central nervous system responses to painful stimuli [v]. Somatic responses include sensory (perception of pain) and motor activity (movement), autonomic system responses include respiratory (increased tidal volume or frequency of breathing), haemodynamic (elevated blood pressure and heart rate), sudomotor (sweating) and hormonal stress responses. This idea of defining depth of anaesthesia by the degree of suppression of central nervous system responses to painful stimuli comprises both the MAC concept and the so called “clinical signs” of too light anaesthesia. The MAC concept (the minimal alveolar concentration that suppress motor response to skin incision in half of the patients), introduced by Eger and colleagues in the early 1960th became a “gold standard” to compare the relative potency of inhaled anaesthetics [vi]. The “clinical signs” of too light anaesthesia, increases in arterial blood pressure, heart rate, sweating and tearing in coherence with the surgical stimulation served (and still serves) as clues to titrating our anaesthetics to individual patient requirements. A PRST score (pressure, heart rate, sweating and tear production) has been proposed for detection of inadequate depth of anaesthesia [vii]. However, using the isolated forearm technique (application of a tourniquet to an extremity before the administration of muscle relaxants), Russell et al. demonstrated that movement following a verbal command, sometimes detected in anaesthetised patients, had a very low correlation with the PRST score [viii].

Today we know that the state of general anaesthesia is not an “all in one phenomenon”, various stages of deprivation of cognitive function have been described during general anaesthesia [ix]:
• Conscious awareness with complaint of pain perception
• Conscious awareness with explicit recall but without pain
• Conscious awareness or “wakefulness” (ability to respond to simple verbal commands [x]) without explicit recall and pain but possible implicit memory
• Subconscious awareness without explicit recall but evidence of implicit memory of intraoperative events
• No awareness

Especially, the implicit memory function has been made responsible for serious psychosomatic disorders, including insomnia, recurrent nightmares, emotional distress and posttraumatic stress disorders.

A number of authors investigated anaesthetic induced changes in electroencephalographic (EEG) patterns. Since the 1940th it has been known that anaesthetics slow down the frequencies of EEG patterns and increase the amplitudes. At higher anaesthetic concentrations the EEG shows burst suppression patterns (isoelectric lines interrupted by high frequency waves) and at very high anaesthetic concentrations isoelectric lines. To quantify the slowing of electroencephalographic patterns single EEG parameters, e.g. spectral edge frequency or median power frequency, were calculated from the electroencephalogram. Since the problems of EEG data collection in the operating theatre surrounding small amplitudes of the EEG signal (10-50 uV) and numerous artefacts has not been eliminated, EEG monitoring is seldom used in routine anaesthesia. Early clinical experience in using EEG signal analysis has not been very convincing. In order to optimise simple interpretation of EEG patterns processed EEG parameter indices have been generated. The most often used one, the Bispectral index™ (BIS, Aspect Medical Systems Inc., Newton, MA, U.S.A.), is calculated from the so called power spectrum and the bispectrum (including synchronisation of brain waves) [xii]. The BIS is a simple parameter ranging from 100 to zero. BIS values near 100 are observed in conscious subjects, whereas BIS values decrease during light (BIS values 70 – 80) and deep sedation (BIS values 70 – 60). Further decrease is associated with unconsciousness and ongoing BIS decrease is observed during burst suppression. A BIS value of 0 indicates isoelectric lines. BIS values of 40 – 60 are suggestive of adequate hypnosis during surgery [xiii].

As an alternative approach to analyse the spontaneous EEG patterns, evoked potentials are derived from the EEG in response to auditory, somatosensory, nociceptive and visual stimuli. Most research with respect to depth of anaesthesia has been done using auditory evoked potentials (AEP) as a response to acoustic stimuli (click). Because of the small size of the bioelectric signal it is necessary to average multiple individual signals over a period of 30 – 120 sec and thereby extract the evoked responses from the overlying spontaneous EEG patterns (background noise). The AEP comprises a series of positive and negative peaks representing the electrical signal processing from cochlea to cortex. The AEP peaks generated from the brainstem (< 10 msec.) are followed by the midlatency (or early cortical) potentials (10 – 100 msec.) and the late cortical response, generated from the frontal cortex and associated areas. The brainstem response waves (early acoustic response) are relatively insensitive to general anaesthetics, whereas midlatency auditory evoked potentials (MLAEPS) are depressed when the subject is anaesthetised. The amplitudes of the peaks decrease, and at the same time the latencies of the peaks are prolonged in a dose-dependent fashion. During anaesthesia persistent MLAEP may indicate insufficient blockade of auditory information processing with the risk of intraoperative awareness [xiii]. A recently introduced MLAEP monitor (AAFM Monitor, Danmeter A/S, DK) partially overcomes the problems of signal averaging with a rapid extraction procedure (called “autoregressive model with exogenous input, ARX”). To quantify the level of consciousness, an index (A-line ARX Index; AAI) in the range from 99 – 0 is calculated from the morphology of the signal (xiv). ARX values > 60 represent awake, 60 – 40 drowsy, 40 – 30 lightly anaesthetised and < 30 deeply anaesthetised subjects during surgery.

Despite these advantages of visualizing and measuring anaesthetic effects on the brain, defining “depth of anaesthesia” is still one of the most controversial, emotional and subjective aspects of our discipline [xv]. However, there is a “gleam of hope” that monitoring adequacy of anaesthesia becomes available in common anaesthesiologic practice.

So let’s try a restate of concepts from the point of view of monitoring “adequacy” of anaesthesia: Assuming anaesthesia should allow surgery without physical or psychical damage to the patient, there are five main targets of anaesthetic drug delivery:
• unconsciousness
• analgesia
• amnesia
• suppression of cardiovascular responses and
• (if required) muscle relaxation
The question is how to monitor these targets.

Monitoring muscle relaxation and suppression of cardiovascular reflexes is common clinical practice and requires no further comments within this review.

Today we do not have in view any specific monitoring for amnesia in anaesthetised patients. Afterwards, having regained consciousness patients may spontaneously recall or remember events that (may) have occurred during anaesthesia. More sophisticated tests are required to detect implicit memory [xvi]. There is some evidence that suppressing memory function with increasing anaesthetic doses may correlate with changes in electroencephalographic activity [xvii, xviii]. Thus, there is some hope that monitoring electroencephalographic activity may prevent from both, awareness and memory in anaesthetised patients.

Again, analgesia is impossible to monitor during general anaesthesia. Starting with the premise that acute pain is the conscious perception of noxious stimuli, patients must be conscious to perceive pain. Consequently, there is no test for detection of pain (nor analgesia, defined as absence of pain) in unconscious subjects. Before having clearly studied the requirement of analgesics during surgical procedures, i.e. with respect to regulation of intraoperative stress response or postoperative behaviour and pain, monitoring the effect of drugs with preferentially analgesic properties would be anyway abdicable.

Thus, the first of the above given targets for anaesthetic drug administration (unconsciousness) is crucial for the concept of monitoring adequacy of anaesthesia. Given that consciousness is a higher cortical function, it is not surprising that electroencephalographic indices predict awareness better than autonomic signs.

Nowadays a number of investigators have developed and presented commercially available EEG or AEP monitors. They all differ in the mathematical approach used for signal analysis. Until today it has not been clearly demonstrated which is the best way or which monitor is superior to ensure patients unconsciousness and suppression of memory function. However, neurophysiologic monitoring seems to be the most promising tool to reduce the incidence of intraoperative awareness. May be some day in the near future “awareness monitoring” becomes a matter of course like a speedometer while driving.

REFERENCES
5. Quasha AL, Eger EI, Tinker JH: Determination and applications of MAC. Anesthesiology 1980; 53: 315-34