The concept of anaesthetic "depth" is ingrained in every anaesthetic trainee from the outset of training. Yet this concept is illusory, and the continuing search for some method to measure anaesthetic depth (Robson, 1969; Saunders, 1981) resembles that for the Philosopher's Stone. In this issue, Evans, Bithell and Vlachonikolis investigate the relationship between spontaneous and evoked oesophageal motility in man, and the end-expired concentration of halothane. Is this a new phenomenon, or is it MAC (minimum alveolar concentration) in disguise?

A series of correspondence in Anesthesia and Analgesia has yet again focused attention on our inability to define the phenomenon which we use daily to render patients insensitive to the trauma of surgery (Russell, 1985; Pinsker, 1986; Kissin and Gelman, 1987). The term anaesthesia, introduced by Oliver Wendell Holmes in 1846, was apposite: it was an attempt to describe a new phenomenon in a single word. While that word has precisely the same meaning to many of us today—that is, the state in which a patient is insensible to the trauma of surgery—it is used more liberally and also more loosely than before. Pinsker (1986) rightly questions our definition of anaesthesia, but compares the term with that of sickness which, like shock, is too non-specific to be of value in present day medical practice. Perhaps the reason why the term anaesthesia is so difficult to define is that successive generations of practitioners of the art have centred their concepts around the effects of the drugs available to them, rather than the responses of the patient to the trauma of surgery, and the suppression of those responses by the wide variety of drugs now available. In the mid-nineteenth century only nitrous oxide, di-ethyl ether and chloroform were available to induce the state of general anaesthesia.

In order to clarify our definitive understanding of anaesthesia, I offer first some definitions of essential terms, as a prelude to an alternative concept that surgery, as an example of noxious stimulation, induces a variety of reflex responses, each of which may be modified independently to the benefit of the patient.

Let us start with an essential premise, that pain is the conscious perception of a noxious stimulus. In this context, the state of anaesthesia can be defined as that in which, as a result of drug-induced unconsciousness, the patient neither perceives nor recalls noxious stimulation. Such a definition is entirely consistent with Holmes's original concept of anaesthesia, and is also consistent with most of our present use of the word. However, in that loss of consciousness is a threshold event, it follows that anaesthesia is an all-or-none phenomenon. There cannot be degrees of anaesthesia, nor for that matter can there be variable depths of anaesthesia. All other attributes of anaesthetics, the drugs which produce the state of anaesthesia, can be classed as alternative pharmacological properties of the drugs, and not as components of the state of anaesthesia. In many respects the term hypnosis is synonymous with anaesthesia as it implies a drug-induced sleep. Confusion has arisen unnecessarily because hypnosis (mental block) was proposed as a component of anaesthesia (Woodbridge, 1957), together with analgesia (sensory block), muscle relaxation (motor block), and suppression of reflexes. Pinsker questions our inability to distinguish between hypnosis and amnesia as components of anaesthesia, implying that recall indicates a failure to anaesthetize (Eich, Reeves and Katz, 1985). This is admittedly a philosophical problem, in that patients who were apparently in a state of anaesthesia have been made to recall under the influence of hypnotism (Levinson, 1965; Cherkin and Harroun, 1971).

Analgesia is normally defined as diminished or abolished perception of pain in an otherwise conscious patient. Thus we classify mild (aspirin) or powerful (opioid) analgesics according to their propensity to modify the perception of pain without significantly modifying the patient's consciousness. Given in sufficient dosage, opioids can produce a state indistinguishable clinically from that induced by i.v. or inhalation anaesthetics.
Unlike the latter, opioid-induced anaesthesia can be achieved only by doses which cause total suppression of ventilatory drive. There is no need to confuse this pharmacological effect of opioids with their specific analgesic or ventilatory depressant properties mediated at specific opioid receptors. There is little evidence at present to link the "anaesthetic" state induced by large doses of opioids to receptor-mediated activity (Dodson and Miller, 1985).

**Muscle relaxation.** Before the advent of drugs which induce neuromuscular blockade, adequate muscle relaxation for surgical access to the abdominal cavity could be achieved by increasing the inhaled concentration of volatile anaesthetics. It was, therefore, not unreasonable at that time to regard this dose-dependent pharmacological effect of the drugs which produced anaesthesia as a component of the state of anaesthesia. However, it is illogical and confusing to include muscle relaxation induced by neuromuscular blocking drugs as a component of the state of anaesthesia (Woodbridge, 1957; Kissin and Gelman, 1987). Muscle relaxation, however achieved, is a desirable goal to satisfy the requirements of the anaesthetist for laryngoscopy and the surgeon for surgical access, but it is neither a component of anaesthesia, nor an alternative to inadequate anaesthesia. The misery caused to many patients who have been aware during supposed general anaesthesia could have been avoided if this confusion had never arisen.

**Responses to Noxious Stimulation**

An alternative approach to understanding the effects of drugs which produce anaesthesia, analgesia or muscle relaxation, is to consider the way in which animals (including humans) respond to noxious stimulation. A noxious stimulus is one which can cause potential or actual damage to cells, and may be produced by mechanical (surgical injury), chemical, thermal or radiation injury. Surgery sets up a continuous noxious stimulation, of varying degree and character, during and for a variable period after an operation. Whatever the nature of the noxious stimuli, they generate action potentials which are transmitted by Aδ and C fibres in spinal or cranial sensory nerves. These action potentials may be relayed centrally or may set up reflex responses at a spinal level, particularly when the stimulus arises from visceral rather than somatic origins (Cervero, 1985). In addition to evoking the perception of pain (in a conscious subject), noxious stimulation also evokes a number of somatic and autonomic reflexes which Sherrington termed "pseudaffetive" reflexes. These reflexes occur simultaneously with the perception of unpleasant sensory experiences (Jänig, 1985; Schmidt, 1985), and are particularly prominent when evoked by stimulation of abdominal or thoracic viscera. The continuation of noxious stimulation into the post-operative period also evokes a metabolic and endocrine response (Kehlet, 1982).

Figure 1 shows a scheme of such reflex responses to noxious stimulation, ranked from left to right in the order in which they are suppressed by general anaesthetics.

**Sensory**

Impulses of somatic origin are relayed through thalamic nuclei and projected to the sensory cortex where there is spatial representation of the anatomical distribution of the stimulus. Good resolution of spatial localization is a feature of somatic sensory input, in contrast to the poor

![Fig. 1. Suppression of responses to noxious stimuli.](https://academic.oup.com/bja/article-abstract/59/11/1341/287530/11341287530)
localization characteristic of visceral stimulation (Cervero, 1985) which does not have spatial representation in the cortex. Perception of pain, either of somatic or visceral origin, is dependent on the state of consciousness, and may be of the "fast" or "slow" modality according to the transmission by Aδ or C fibres, respectively.

Suppression of both perception and recall of pain can be achieved with blood concentrations of either i.v. or inhalation anaesthetics which are too low to suppress the motor responses.

**Motor**

The motor response to somatic noxious stimulation is characterized by reflex withdrawal of the stimulated part. Suppression of this simple reflex has been used as the basis of the main quantitative indices of anaesthetic potency, the minimum alveolar concentration (MAC) for inhalation agents (Merkel and Eger, 1963; Eger, Saidman and Brandstater, 1965), or the minimum infusion rate (MIR) for i.v. anaesthetics (Prys-Roberts and Sear, 1984). In the normal way that these indices are determined in man, approximately 50% of the patients will move in response to the initial surgical incision; yet none of the patients who moved, in a series of the author's studies of i.v. anaesthetics, recalled any part of the period of anaesthesia and the associated surgery. It is therefore implicit, that the blood concentration of anaesthetic required to suppress the somatic motor response is higher than that required to induced unconsciousness, and by implication, perception of pain.

**Breathing**

Under conditions of clinically acceptable anaesthesia with inhalation or i.v. agents, increased frequency and volume of ventilation, breath-holding or laryngospasm can occur, even though there is no somatic motor response to the surgical stimulus. Greater concentrations of anaesthetics are required to suppress breathing responses to somatic noxious stimulation, than to suppress motor responses or to produce unconsciousness.

**Autonomic responses**

Noxious stimuli cause reflex activation of the sympathetic nervous system, which can be considered a part of the "Abwehrverhalten" (General Defence Reaction) as observed in animals (Hess and Brugger, 1943; Hess, 1949; Hilton, 1982). During clinical anaesthesia the manifestations of these responses may be modified by the use of drugs which specifically block central or peripheral transmission of sympathetic nervous action potentials (e.g. clonidine, or β-adrenoceptor antagonists). The autonomic responses can be classified simply into sudomotor, haemodynamic and hormonal.

**Sudomotor** responses (sweating) are commonly observed during nitrous oxide in oxygen–muscle relaxant–opioid anaesthetic combinations, mainly in response to visceral (intra-abdominal or intrathoracic) manipulations. They are readily suppressed by low concentrations of volatile or i.v. anaesthetic supplements (Jänig and Räth, 1980).

**Haemodynamic** responses represent the effects of increased sympatho-adrenal activity on the cardiovascular system. The most obvious effects are increased arterial pressure and heart rate, the two variables most commonly monitored in routine anaesthesia. These responses occur commonly in response to both anaesthetic (laryngoscopy) and surgical stimuli, even when anaesthetic concentrations are high enough to prevent sensory, motor and breathing responses. Roizen, Horrigan and Frazer (1981) introduced the concept of MAC-BAR as a correlate of MAC, being the alveolar concentration of an anaesthetic which would suppress haemodynamic and adrenergic responses in 50% of patients. They found that the ratio of MAC-BAR to MAC was 1.45 for halothane and 1.60 for enflurane. Ausems and colleagues (1986) determined the plasma alfentanil concentration (C₄₅₀) at which the haemodynamic, autonomic and somatic responses to surgery were suppressed in 50% of patients. Both these studies are consistent with the concept of a ranked order of reflex responses to noxious stimulation, in which the sensory class of response was abolished at the lowest concentration of anaesthetic or analgesic drug.

**Hormonal** responses to the injury of surgery have been long recognized because they can be measured well into the postoperative period. They are difficult to suppress by volatile anaesthetics, but can be partially suppressed by high doses of opioids (Hall et al., 1978) and by regional blockade (Kehlet, 1982). They are only partially suppressed by beta-adrenoceptor blockade (Cooper et al., 1980).

**A Restatement of Concepts**

Analgesia, muscle relaxation and suppression of autonomic activity, are all discrete pharmaco-
logical effects which can be achieved independently by specific drugs. While a number of inhalation and i.v. anaesthetics, and opioid analgesics, may exert some or all of these effects to a greater or lesser degree, the suppression of sensory perception is the only feature common to all general anaesthetic agents. If one accepts the premise that the ranked order of responses to noxious stimulation is a valid representation of the events associated with surgery, then it is logical to consider anaesthesia as that state which ensures the suppression of the somatic and visceral sensory components, and thus the perception of pain.

Analgesia, muscle relaxation, and suppression of autonomic activity, are not components of anaesthesia. Rather they should be considered as desirable supplements to the state of anaesthesia as a means to enable surgery to be performed. The use of low concentrations of anaesthetics with muscle relaxants has resulted in an unacceptably large number of patients being aware during general anaesthesia. Any reliable indicator that the level of anaesthesia is adequate to ensure lack of awareness in the presence of muscle relaxants, is therefore highly desirable.

Returning to the question posed earlier about the phenomenon of oesophageal motility and its correlation with anaesthetic "depth", one must conclude that this is a visceral (autonomic) motor function, in that spontaneous and evoked oesophageal contractions are more akin to visceral peristalsis than to somatic muscular activity. However, the graded suppression of oesophageal movements and their correlation with end-tidal halothane concentrations is hardly surprising; it can be considered to be a visceral MAC. A similar relationship has been observed during infusions of propofol (I. Norley and C. Prys-Roberts, unpublished observations).

Other methods have been described to achieve the same end-point, although they have involved different modalities such as the EEG and its derived parameters (Rampil et al., 1980; Schwinden, Schüttler and Stoeckel, 1985) and brain stem auditory evoked responses (Robson, 1969; Thornton et al., 1984). These clearly demonstrate a dose-effect relationship for many anaesthetic agents, but do not answer that fundamental question: is it feasible to find some measure which will ensure that the patient will be unaware of, and will not recall, events and sensations during surgery? The findings of Evans and his colleagues show that oesophageal motility, as a non-invasive monitor of visceral motor suppression, can be useful. While the presence of oesophageal activity, either spontaneous or evoked, may imply anaesthesia inadequate to prevent awareness, we cannot, in the present state of knowledge, be certain that absence of oesophageal motility guarantees that the patient is adequately anaesthetized!

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REFERENCES


