COMPONENTS OF ANAESTHESIA

Sir,—We would like to comment on two important questions raised in the excellent editorial by Prys-Roberts [1]. Prys-Roberts doubts (and we agree) that the dose-effect relationship demonstrated for various pharmacological effects of general anaesthetics may be used to predict their basic effect: prevention of conscious perception of a noxious stimulus. However, pharmacological effects, regarded as the basic components of anaesthesia (unconsciousness, blockade of motor response and suppression of autonomic responses to noxious stimulation) may have different interrelationships, and therefore the index of one of the components may not be a reliable indicator of another component. Figure 1 demonstrates that the relationship and even the ranked order of the components of anaesthesia are such that prediction for one component of anaesthesia based on the results obtained with another component is different between agents. With a combination of several drugs, the relationship between the components becomes even more complicated [2].

The second question is related to the definition of anaesthesia. Prys-Roberts rejects the notion of components of anaesthesia. He defines anaesthesia as a drug-induced unconsciousness when the patient neither perceives nor recalls noxious stimulation. He states that “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 the definition of general anaesthesia suggested by Holmes in 1846, the complex of effects provided by a single inhalation anaesthetic was named after one most important effect: unconsciousness that prevents perception of pain. When it became obvious that some of the effects of inhalation anaesthetics can be induced more selectively with other drugs, the concept of components of anaesthesia emerged [3]. As a result, some of the components of anaesthesia could not strictly fit the initial definition. The best example is muscle relaxation: when induced by a general anaesthetic, it is regarded as a component of anaesthesia, but when it is induced by a peripherally acting muscle relaxant, we doubt if it may be termed a component of anaesthesia. Similarly, we may regard psychological detachment, complete analgesia, and amnesia achieved by a combination of specific drugs (neuroleptanalgesia) as a substitute for unconsciousness. This does not contradict Woodbridge’s concept of components of anaesthesia [3]. He indicated in his table of four components that mental block implies “ataxia, or amnesia, or sleep.”

Can the combination of complete analgesia and amnesia be regarded as anaesthesia consisting of two components? Probably yes. The requirements for different operations/patients can be different, dictating appropriate combinations of components of anaesthesia. Unconsciousness may not necessarily be “the most important” component. In our opinion, all separate effects used to protect the patient from the trauma of surgery should be termed components of anaesthesia, induced selectively by several drugs or unselectively by one drug. They may not fit the strict definition of anaesthesia, but they do reflect appropriate components of anaesthetic management. Our use of words is often not very precise and, at present, we do not see any need to reconcile the contradiction between the initial definition of anaesthesia with its new meaning by restricting this meaning: anaesthesia should include all of its components, even muscle relaxation.

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FIG. 1. Median effective doses of thiopentone, diazepam, isoflurane and morphine for different end-points of anaesthesia in rats. HE = ED$_{50}$ for hypnotic effect (loss of the righting reflex). PM = ED$_{50}$ for blockade of purposeful movement response to noxious stimulation (equivalent of MAC). CA = ED$_{50}$ for suppression of cardiac acceleration response to noxious stimulation. LE = ED$_{50}$ for lethal effect (LD$_{50}$). Horizontal lines = 95% confidence limits. Data from references [4–6].
REFERENCES


SUXAMETHONIUM MYALGIA

Sir,—We were very interested in the comments of Drs Mather and Carter [1] relating to suxamethonium myalgia. We would, however, challenge the validity of the deduction that pretreatment with suxamethonium is not effective in ameliorating myalgia, because of the time relationship of 45 s which was used in the study between pretreatment and administration of suxamethonium.

It is generally accepted that, to be effective, pretreatment with a non-depolarizing neuromuscular blocking agent must be given 2–3 min before suxamethonium [2], whereas pretreatment with suxamethonium must be “immediately prior to the full relaxant dose of suxamethonium” [3]. This discrepancy arises because of the different pharmacokinetics of the two groups of drugs. Our clinical experience convinces us that correctly timed pretreatment with suxamethonium, or its very slow administration (over a period of 10 s) significantly reduces the incidence and severity of suxamethonium myalgia.

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SIR,—We were interested in the comments of Drs Mather and Carter. We consider that there is little or no definitive evidence that the time interval between pretreatment with suxamethonium and administration of the subsequent dose is of critical importance. In his original description of the technique, Baraka [1] used a latent period of 45–60 s; a similar interval has been used by most subsequent authors, including ourselves [2–5]. We would accept that this technique does not appear to modify postoperative myalgia, but remain unconvinced that the shorter time interval used by Drs Mather and Carter (a difference of approximately 30 s) is of crucial importance. Although it has been suggested that the incidence of muscle pain may be reduced by the slow injection of suxamethonium [6], this has not been confirmed by other authors [7].

We are unable to understand the reference to the different pharmacokinetics of suxamethonium and non-depolarizing blockers. As far as we are aware, the pharmacokinetics of suxamethonium have not been studied, since its plasma concentration cannot be determined reliably.

Finally, although it is often stated that pretreatment with a non-depolarizing neuromuscular blocker must be given 2–3 min before suxamethonium in order to be effective, our data suggest that a shorter time interval may be adequate.

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ASSESSMENT OF THE DATEX RELAXOGRAPH IN ACCESSORY NERVE STIMULATION

Sir,—Since the report of Schumm and Stöhr [1] we have frequently observed that stimulation of the accessory nerve is useful, not only in the assessment of myasthenia gravis, but also in the assessment of neuromuscular transmission in anaesthetized patients. The method is sensitive and reliable, and we decided to use it with the Datex Relaxograph in the “train-of-four” mode of stimulation.

Stimulating electrodes were placed just behind the sternocleidomastoid muscle, the exploring electrode 4 cm dorsal to the edge of trapezius muscle, the indifferent electrode above the clavicle and the ground electrode medial to the indifferent electrode.

Since our purpose was to monitor the trend of neuromuscular transmission for prolonged periods (rather than to detect absolute values) we used only submaximal stimuli with intensities of current that are selected on the basis