THE HERING–BREUER REFLEX IN MAN?

Sir,—In a recent Postgraduate Educational Issue of the journal, Dr Nunn stated that the Hering–Breuer reflex is virtually non-existent in man [1]. On the contrary: this reflex has been demonstrated in humans anaesthetized with halothane [2] or Althesin [3], in addition to its existence in anaesthetized animals. It is regrettable that as eminent a physician as Dr Nunn failed to mention this, even if the exact role of this reflex is unclear.

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1. Nunn JF. Effects of anaesthesia on respiration. British Journal of Anaesthesia 1990; 65: 54-62.

Sir,—I am indebted to Dr Gaudy for drawing my attention to his studies in anaesthetized man. In one of these, end-inspiratory occlusion resulted in a 10 % prolongation of the following expiration in eight of nine subjects [1]. After end-inspiratory occlusion, lung inflation of 250 ml caused a 40 % slowing of breathing (which did not appear significant). Larger inflations caused significant slowing of breathing up to 15-fold at 1 litre (equivalent to an inflation pressure of about 20 cm H₂O). It is not in doubt that the Hering–Breuer reflex can be detected in man, but the question at issue is whether or not it is of any practical relevance to the control of breathing in man, as it is in most laboratory animals.

A letter does not allow adequate space to debate the validity of my statement that the Hering–Breuer reflex is virtually non-existent in man. However, perhaps I may be permitted to draw attention to the five most important observations on which my opinion is based.

(1) From the comparative study of Widdicombe [2], I would cite figure 1, reproduced here, which speaks for itself. Widdicombe concluded that "... caution must be exercised before ascribing any important role to the Hering–Breuer reflexes in modifying the pattern of breathing in healthy man."

(2) Bilateral vagal block in man causes no detectable changes in the pattern of breathing (in contrast to the slowing which occurs in a range of laboratory animals) [3].

(3) Human heart–lung transplant (with loss of baroreceptor output from the lungs) does not change ventilatory frequency or its components [4].

(4) Every anaesthetist will have observed that, in gross bradypnoea (e.g. after opioids), a brief lung inflation often triggers inspiration.

(5) End-expiratory obstruction (effectively, lung inflation) augments the inspiratory force developed by the diaphragm [5]. This augmentation resulted in a further increase in lung volume in all patients up to 6 cm H₂O, in 75 % of patients in the range 7-9 cm H₂O, and in 8 % of patients in the range 10-16 cm H₂O.

The recorded observations in the last study do not exclude the possibility that expiration was prolonged slightly by expiratory occlusion, but I quote from the first paper cited by Dr Gaudy [6]: "La trachée est occlue en fin d'inspiration ou d'expiration spontanée (Head, 1889) sans aucune réponse du type Hering-Breuer." The feature of practical importance is that inspiratory effort is stimulated by moderate inflation of the lung in anaesthetized man, which is quite contrary to what might be expected from an uncritical extrapolation of Breuer

![Fig. 1. The Hering-Breuer reflex in different species: ratio of apnoea during inflation to the duration of the previous ventilatory cycles (abscissa) as a function of the increase in transpulmonary pressure causing inflation of the lungs (ordinate). (Reproduced from Widdicombe [2] with permission of the author, and the editor and publishers of Clinical Science.)](https://academic.oup.com/bja/article-abstract/66/5/627/394218)
and Hering's observations in animals. Many new graduates do not appreciate that the Hering–Breuer reflex is not a major factor in control of ventilatory movements in man, as it is in so many laboratory animals.

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OXYGEN CYLINDERS ON ANAESTHESIA MACHINES

Sir,—The case report by Jawan and Lee [1] illustrates that there is always a possibility of administering the incorrect gas to a patient instead of oxygen, and they make two recommendations which should be mandatory to prevent future mishaps: first, increased vigilance is required on the part of anaesthetists and theatre staff in checking cylinders; second, there is a need to monitor the inspired oxygen concentration with an oxygen analyser, and oxygenation of the patient with a pulse oximeter.

However, they omitted a third recommendation concerning the number of oxygen cylinders which the anaesthetic machine should carry. It appears obvious from the case report that the anaesthetic machine used by the staff had only one oxygen cylinder, either because the machine had one cylinder only, without a spare oxygen cylinder on the second oxygen port, or because they may have used a newer anaesthetic machine with provision for only one oxygen cylinder (Ohmeda Excel 210), unlike other models which carry two oxygen cylinders in addition to the oxygen pipeline supply (Boyle Major). The recommendation in the former case is to have a second full oxygen cylinder available on the machine at all times. In the latter instance, a strong recommendation must be made to manufacturers that anaesthetic machines should have provision for two oxygen cylinders in addition to the oxygen pipeline supply.

In the absence of a pipeline supply (as in the authors' case) or during a prolonged failure in the oxygen pipeline supply during anaesthesia, the hurried replacement of an empty cylinder by a full cylinder could be difficult and dangerous. As demonstrated by the case report, the change of cylinders under the circumstances described may have contributed to the delay in recognizing the markings on the cylinder. For some reason, this safety feature of older anaesthetic machines has been discontinued on newer versions. In our opinion, this is unquestionably a retrograde step for the safety of modern anaesthesia. The provision of two oxygen cylinders in addition to the oxygen pipeline supply on anaesthetic machines must be included in the minimum standards of requirements for those machines, and in the minimum standards of monitoring during anaesthesia and recovery [2] in order to prevent future mishaps.

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EXTRADURAL OPIOIDS

Sir,—I read with interest the recent correspondence from Nagle and McQuay [1] on extradural pethidine. They suggested that its rapid onset resulted from systemic uptake rather than a spinal effect following dural transfer. In support of this, they made several assumptions on the potency of pethidine and its ability to penetrate the dura.

Several studies have shown a rapid onset of analgesia after extradural pethidine, and a high CSF: plasma concentration ratio exceeding 70 at 5 min [2] supports a predominantly spinal action. Systemically absorbed pethidine may contribute to the effect, but plasma concentrations of pethidine following extradural administration have been demonstrated to decrease to less than minimal analgesic blood concentrations before analgesia ceases [3].

With respect to fentanyl, Nagle and McQuay claimed that doses of 1–5 mg were necessary for spinal action; this represents a dose of 5 to 50 times that used commonly in clinical practice. The use of such large doses would invariably result in respiratory depression. Loper's recent study, which