SURGICAL STIMULATION DOES NOT ENHANCE VENTILATORY CHEMOREFLEXES DURING ENFLURANE ANAESTHESIA IN MAN

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ABSTRACT

To assess the impact of surgical stimulation on regulation of ventilation in anaesthetized man, we measured ventilation and the ventilatory responses to either hyperoxic hypercapnia or to isocapnic hypoxaemia in fifteen subjects anaesthetized with enflurane 1.1 MAC, just prior to and then during a surgical procedure. Anaesthesia alone reduced ventilation, increased Pa_{CO_2}, decreased the response to carbon dioxide and virtually abolished the response to hypoxaemia. The addition of operation at the same level of anaesthesia augmented ventilation and reduced Pa_{CO_2}, but did not improve the anaesthesia-induced impairment of the responses to hypercarbia and hypoxaemia. Over the range of P_{CO_2} and P_{O_2} values studied, the effects of surgery were constant and independent of chemical drive.

IN UNSTIMULATED SUBJECTS, halogenated anaesthetic agents are ventilatory depressants; they reduce minute ventilation, increase Pa_{CO_2} and impair ventilatory responses to added carbon dioxide, hypoxaemia and metabolic acidaemia, all in a dose-related fashion.[1-5] Surgery acts commonly as a ventilatory stimulant, augmenting minute ventilation and reducing Pa_{CO_2} from values observed during anaesthesia alone.[6,7] Whether surgical stimulation also affects chemoreflex activities such as the ventilatory responses to hypercarbia and to hypoxaemia has not been explored in man. Augmentation of resting ventilation during surgery does not necessarily imply a parallel improvement in ventilatory chemoreflexes.

METHODS

The protocol for this study was reviewed and approved by an Ethics Committee of the University of Western Ontario. Each subject gave written consent to participate, after being informed of the nature of the proposed study and the risks involved.

There were 15 healthy subjects, all of whom were about to undergo dental or peripheral orthopaedic operations. Their mean age was 22 ± 3 years, weight 66.8 ± 12.7 kg, and height 170 ± 11 cm (mean ± S.D.). All subjects were studied at enflurane 1.1 MAC, first without and then with operative stimulation. To reduce the duration of individual studies to a minimum, we arbitrarily divided subjects into two groups and measured only one chemoreflex response in the members of each, in group I (n = 8), the response to hypercapnia and in group II (n = 7), the response to hypoxaemia.

All subjects were unpremedicated and were supine. Anaesthesia was induced with enflurane in oxygen, employing a non-rebreathing system. Some inductions were supplemented with a small intravenous dose of thiopentone (1–2 $mg \cdot kg^{-1}$). With the aid of intravenous succinylcholine 1 $mg \cdot kg^{-1}$ the glottis and upper trachea were sprayed with a topical lidocaine solution and the trachea was intubated with an 8 or 8.5 mm cuffed tube. Administration of enflurane in oxygen continued for at least 45 minutes, with inhaled concentrations adjusted to achieve end-tidal values equivalent to 1.1 MAC (i.e. 1.5–1.9 per cent)[8]. Before commencing tests, we ensured that values of end-tidal enflurane, $P_{ET_{CO_2}}$ and minute ventilation were all steady for at least 10 minutes.

We first recorded a two-minute period of ventilation and withdrew a sample of arterialized blood from a free-flowing vein of the dorsum of the hand for determination of carbon dioxide tension.[9] Next, in subjects of group I, we induced hyperoxic hypercapnia by Read's rebreathing technique.[10] A circle rebreathing circuit of 10-litre capacity was flushed and filled with carbon dioxide 7–9 per cent in oxygen, along with

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This work was supported by the Medical Research Council of Canada, Development Grant 150. .

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enflurane in a concentration appropriate to maintaining end-tidal enflurane constant. The subject's airway was connected to this circuit and his lungs were inflated with four large volumes. Following the resumption of spontaneous breathing, the recording of exhaled carbon dioxide concentration was examined for the appearance of a steady mixed venous "plateau", indicative of an equilibrium of carbon dioxide between mixed venous blood, arterial blood and gas in the lung and rebreathing circuit.[11] With a plateau present, rebreathing was allowed to continue until the circuit carbon dioxide concentration had increased 1.5–2.0 per cent (approximately 1.5–2.0 kPa). This was usually achieved in four or five minutes. Subsequently, the subject was returned to the non-rebreathing circuit to re-establish control values of ventilation and $P_{ET_{CO_2}}$.

With ventilatory and end-tidal enflurane variables steady, the surgical procedure began. To maintain end-tidal enflurane at the 1.1 MAC level during operation it was frequently necessary to reduce inspired enflurane concentrations slightly. After at least 15 minutes of surgery and when values of $P_{ET_{CO_2}}$ became reasonably steady (±0.2 kPa) at a new level, we repeated the measurements made during anaesthesia alone.

In subjects of group II, we measured the ventilatory response to isocapnic hypoxaemia instead of the response to hypercapnia. Hypoxaemia was induced by a progressive and non-rebreathing technique, similar to that employed by Weil *et al.*[12] Inhaled oxygen was replaced with air and then with a mixture of air and nitrogen so that end-tidal oxygen concentration decreased to approximately 6 per cent (i.e. 6 kPa, 45 torr) over a 6 to 10-minute period. The lowest level of oxygen tension was maintained constant for about 20 seconds. End-tidal carbon dioxide was monitored continuously, with carbon dioxide available for inhalation if needed to maintain end-tidal isocapnia.

Exhaled gases were sampled continuously during all tests and were analyzed for carbon dioxide, oxygen and enflurane concentrations by a Perkin-Elmer #1100 mass spectrometer. The instrument was calibrated each testing day with oxygen and carbon dioxide standards from Canadian Liquid Air Company and an enflurane standard from Scott Speciality Gases. Output signals were fed to a multi-channel time-based recorder, from which end-tidal plateau concentrations could be read. Values of dried end-tidal gas concentrations were converted to tensions, employing the measured barometric pressure of the day of testing and assuming water vapour saturation of end-tidal gas.

Blood samples were capped and placed on ice immediately. They were analyzed within an hour for their carbon dioxide and oxygen tensions, using a Radiometer Copenhagen BMS III system. Arterialization of blood samples was considered adequate if P_{O_2} exceeded 26.6 kPa (200 torr).

Inspired volume was measured by pneumotachography, with a pneumotachograph head incorporated in the inspiratory limb of each circuit. The integrated signal (volume), was regularly calibrated with an air calibration syringe, and correction factors were applied for the density and viscosity of the various gas mixtures inhaled. Values of resting ventilation were the averages of one-minute recordings of ventilation; values of instantaneous ventilation during chemoreflex testing were the averages of at least three consecutive breaths. All volume and ventilation values were converted to B.T.P.S..

From each chemoreflex test, we collected 12 or more pairs of observations ($\dot{V}_I$, $P_{ET_{CO_2}}$ or $\dot{V}_I$, $P_{ET_{O_2}}$ points). To represent the response to carbon dioxide, we assumed a linear relationship between ventilation and $P_{ET_{CO_2}}$ and found the slope of its least-squares regression. To depict responses to hypoxaemia, we assumed that values of ventilation related to $P_{ET_{O_2}}$ in a hyperbolic fashion, according to the following regression equation:

$$\dot{V}_I = \dot{V}_{I_O} + A/P_{ET_{O_2}} - 30^*$$

We computed the parameter "A", which is the slope of the least-squares hyperbolic regression. The value of "A", with the units l·kPa/min represents the shape of the $\dot{V}_I$:$P_{ET_{O_2}}$ curve and therefore the magnitude of ventilatory response to hypoxaemia.[12] In addition, we determined the "$\Delta\dot{V}_{I_{45}}$"; that is, the increment in ventilation between $P_{ET_{O_2}}$ of 50 kPa and 6 kPa (approximately 400 torr and 45 torr).

To detect possible differences between data collected before and during surgical stimulation, we employed a Student's t-test for paired data. P values of 0.05 or less were regarded as indicative of a significant difference. To compare our data to the awake state, we have included in our results the carbon dioxide and hypoxaemia responses of 15 awake subjects who were matched closely to

*The constant "30" represents the $P_{ET_{O_2}}$ at which the extrapolated slope of the $\dot{V}_I$:$P_{ET_{O_2}}$ curve approaches infinity and the $\dot{V}_{I_O}$ represents the $\dot{V}_I$ at high $P_{ET_{O_2}}$ where the extrapolated slope approaches zero.[12]

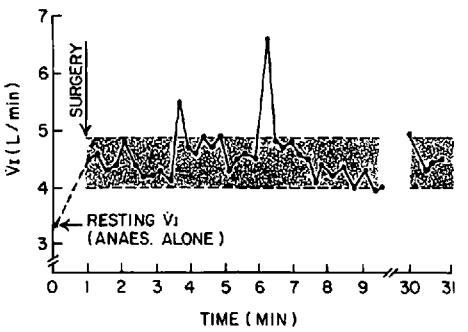

FIGURE 1 Pattern of ventilation of a typical subject anaesthetized with enflurane 1.1 MAC, just prior to and during surgical stimulation. Points represent the averaged values of ventilation over successive 15-second intervals. Anaesthesia alone reduced ventilation to a steady value below predicted awake. Surgery augmented ventilation variably from moment to moment. The two peak values of ventilation (points above the shaded area) were associated with drilling of bone.

our anaesthetized subjects for age, sex and body size.

RESULTS

All subjects recovered from anaesthesia and the surgical procedure without complications related to our study.

Compared to expected awake values, enflurane 1.1 MAC reduced minute ventilation, increased Pa_{CO_2} and altered the pattern of breathing to smaller volumes and increased frequencies (Table I).

Surgical stimulation always augmented ventilation, but the effect varied from moment to moment (Figure 1). Much of the variation could be attributed to particularly potent surgical stimuli, such as incisions of oral mucous membranes and drilling of bone. Typically, these stimuli produced a sudden and large increment of ventilation, followed by a gradual diminution over the minute or so after cessation of the stimulus (Figure 1). The basis for the remaining variability was not apparent.

As the abrupt increments of ventilation associated with potent stimuli made the determination of ventilatory responses to chemical variables difficult (see Discussion), we limited our measurements and tests to periods of surgery in which these stimuli were not present. Values of instantaneous ventilation then fell within ranges of less than one litre/min in all subjects (e.g. shaded area of Figure 1). The ventilation and ventilatory responses reported are in effect mean values from these ranges.

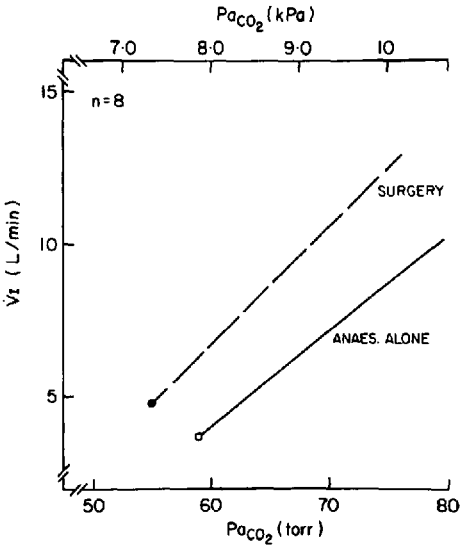

FIGURE 2 Effect of surgical stimulation on ventilatory response to carbon dioxide at enflurane 1.1 MAC. (Mean data of group I subjects.) Circles represent values of ventilation and Pa_{CO_2} observed at rest. Lines depict the calculated slopes of ventilation: P_{CO_2} relationships. While surgery augmented ventilation and reduced Pa_{CO_2}, it did not alter the slope of the ventilatory response to carbon dioxide.

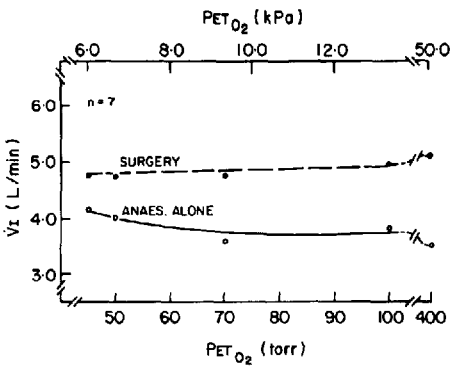

FIGURE 3 Effect of surgical stimulation on ventilatory response to hypoxaemia at enflurane 1.1 MAC. (Mean data of group II subjects.) Circles represent mean ventilation at $P_{ET_{O_2}}$ of 50, 13.3, 9.3, 6.7 and 6.0 kPa (approximately 400, 100, 70, 50 and 45 torr). Lines were hand drawn through these points. During anaesthesia, the ventilatory response to hypoxaemia was virtually absent. Surgery increased ventilation, but did not restore the hypoxaemic response.

Surgery increased minute ventilation and tidal volume, but did not alter ventilatory frequency. Pa_{CO_2} decreased during surgery, with individual reductions ranging from 0.1 to 1.3 kPa (Table I).

TABLE I
VALUES OF VENTILATION AND Pa_{CO_2}

	GROUP I		GROUP II	
	Anaes. alone	Anaes. & surgery	Anaes. alone	Anaes. & surgery
$\dot{V}_I$ (l/min)	3.55 ± 0.33	4.73 ± 0.34*	3.51 ± 0.39	5.11 ± 0.67*
V_T (litres)	.20 ± 0.02	.27 ± 0.02*	.23 ± 0.01	.31 ± 0.03*
f	18 ± 1	17 ± 1	15 ± 1	16 ± 1
Pa_{CO_2} (kPa)	7.5 ± 0.4	6.5 ± 1.0*	7.4 ± 0.4	6.9 ± 0.3*

All values are mean ± S.E.M.
*Significantly different from values during anaes. alone ($p < 0.005$).

TABLE II
VENTILATORY RESPONSES TO CO_2 AND HYPOXAEMIA

	Awake*	Anaes. alone	Anaes. & surgery
CO_2 response (Group I) Slope (L/min/kPa)	13.5 ± 1.5	2.3 ± 0.8	3.0 ± 0.8
Hypoxaemic response (Group II) "A" (l·kPa/min)	23 ± 2	2 ± 1	−0.3±1
$\Delta\dot{V}_{I_{45}}$ (l/min)	9.9 ± 0.8	0.5 ± 0.3	−0.4 ± 0.3

All values are mean ± S.E.M.
*Matched group of awake subjects.

Enflurane 1.1 MAC alone reduced the slope of the ventilatory response to carbon dioxide and virtually abolished the responses to hypoxaemia (Table II). With surgical stimulation, there was no statistically significant change in the reactivity to either chemical stimulus (Table II, Figure 2 and 3).

DISCUSSION

While there is considerable information on the depressive effects of anaesthesia on cardiorespiratory function in man, relatively little has been documented on the impact of an operative procedure. Anaesthetists know that surgical stimulation is a potent stimulus to breathing and can sometimes be useful in initiating and maintaining adequate spontaneous ventilation in their patients.[6,7] The aim of this study was to quantify this effect of surgery on both ventilation and ventilatory chemoreflexes in healthy subjects at enflurane 1.1 MAC.

To study and characterize regulation of breathing properly requires that the level of anaesthesia, the carbon dioxide tension and the volume of ventilation shall all be constant for at least ten minutes before making measurements. These "steady-state" end-points were achieved with relative ease during anaesthesia alone, but surgical stimulation presented a problem. Ventilation (and thus ventilatory control) varied from moment-to-moment in the course of surgery, especially during and immediately following intense surgical stimuli (Figure 1). Swings of instantaneous ventilation were a problem particularly when superimposed upon chemoreflex re-

sponses; their presence made discernment of the effects of progressive chemical stimuli (hypercarbia or hypoxaemia) nearly impossible.

To reduce the problem considerably, we elected to study periods of surgical stimulation which excluded acute potent stimuli. This reduced the variation of ventilation to much smaller and reasonably consistent ranges (such as the shaded area of Figure 1). Our methods of analysis in effect "damped" the remaining unsteadiness; we averaged values of resting ventilation over two-minute periods and computed mean parameters of chemical responsiveness from large numbers of data points collected over five to ten minutes of testing.

The ventilatory effects of enflurane 1.1 MAC (Table I) were similar to those previously reported.[5,13] When administered alone this agent is the most potent ventilatory depressant among inhaled halogenated agents. Surgical stimulation during enflurane anaesthesia increased mean ventilation 1.5 l/min and reduced mean Pa_{CO_2} by 0.7 kPa (5 torr); a change in Pa_{CO_2} approximating that observed during equi-potent anaesthetic doses of halothane, isoflurane, isoflurane-nitrous oxide or with a halothane or isoflurane-nitrous oxide-narcotic mixture.[1,6,7]

The reduction in Pa_{CO_2} in our study was not as great as would be expected on the basis of the ventilatory response alone; ventilation increased approximately 30 per cent, while the mean Pa_{CO_2} decreased less than 10 per cent. This difference suggested additional effects of operation on non-ventilatory determinants of Pa_{CO_2} such as carbon dioxide output and/or physiological dead-space. We measured these variables in five subjects* and found that surgery increased carbon dioxide output from 131 ± 16 to 172 ± 17 ml/min (mean ± S.E.M., S.T.P.D., $P < 0.05$), while having little effect on physiological dead-space to tidal volume ratio, from 0.48 ± 0.04 to 0.44 ± 0.04 (N.S.). This increment in carbon dioxide output during operation, which was similar in magnitude to values previously reported,[6] accounts for the reduced effect of the ventilatory response on Pa_{CO_2}.

The ventilatory responses to chemical stimuli, which are essential to the normal metabolic regulation of breathing, were of particular interest in this study. The two which were tested, the responses to hyperoxic hypercapnia and isocapnic hypoxaemia, represent the medullary chemoreceptor mediated response to $Pco_2/[H^+]$ and the peripheral chemoreceptor mediated response to Po_2, respectively. Details and limitations of our methods of testing have been outlined in other reports.[4,5]

In agreement with previous findings, enflurane 1.1 MAC alone reduced the ventilatory response to carbon dioxide and virtually abolished the response to hypoxaemia[5] (Table II). Although surgical stimulation increased resting ventilation, it did not alter chemoresponsiveness. During operation, the slope of the response to carbon dioxide was not detectably different from that during anaesthesia alone and there remained little or no ventilatory response to hypoxaemia. Stated another way, the stimulus of surgery augmented ventilation by a similar magnitude at all levels of Pco_2 and Po_2 that we tested. Thus, the impact of surgery was virtually independent of chemical drive (Table II, Figures 2 and 3).

Bourke, *et al.*[14] studied the effect of surgery on the ventilatory response to carbon dioxide in subjects lightly anaesthetized with nitrous oxide and enflurane. Although their method of testing differed from ours (steady states of hypercarbia at only two levels of Pa_{CO_2}), their essential finding was the same; surgical stimulation increased ventilation without altering the sensitivity of response to carbon dioxide. To our knowledge, the effect of surgery on the hypoxaemic response has not been previously studied.

What is the clinical importance of our results? We suspect that the quantitative findings, the magnitude of changes of ventilation and Pa_{CO_2} during surgery, may be closely dependent upon the conditions we studied (i.e. basal stimulation associated with particular operative procedures in healthy young subjects at a steady state of anaesthesia with enflurane 1.1 MAC) and may vary in other clinical circumstances. The observation of greatest interest is that a surgical stimulus which augments ventilation during anaesthesia does not necessarily improve the activity of chemical reflexes. Thus a lightly anaesthetized patient may have a ventilatory response to surgery which reduces carbon dioxide levels toward normal[6,7] and yet still retain the impairment of chemoreflex associated with anaesthesia alone. The implications of obtunded responses to hypercarbia and hypoxaemia are an inability to compensate normally for added carbon dioxide or rebreathing and for episodes of hypoxaemia.

The mechanism by which surgery stimulates

*Carbon dioxide output was determined from the carbon dioxide content of a five-minute collection of exhaled gas. Physiological dead-space to tidal volume ratio was found by Bohr's method.

ventilation in the presence of anaesthesia is uncertain. Nociceptive mechanisms in general are poorly understood. The rapidity of ventilatory changes during operation suggest that neurogenic mechanisms play a prominent role. It is known that peripheral nerve fibres carrying painful stimuli, such as small diameter myelinated (A, delta) and unmyelinated (C) fibres, have extensive connections with brain stem reticular neurons,[15] where respiratory control mechanisms are located. When these peripheral fibres are stimulated electrically in anaesthetized cats, there is an abrupt increase in ventilation similar to that observed with electrical stimulation of the reticular neurons themselves, which is thought to be mediated by "reverberation" of reticular activating circuits.[16–18] Thus, a plausible explanation for the ventilatory effects of surgery or nociceptive stimuli is simply augmented "neural traffic" in the brain stem reticular formation. Specifically where and how this additional "traffic" might act on mechanisms of ventilatory control is not clear. The present data suggests only that the action does not interact with chemical drive. Other factors associated with surgery which might contribute to its ventilatory effects include increases in catecholamine level, metabolic rate, carbon dioxide production and cardiac output, all of which have been reported to be ventilatory stimulants.[19–22]

Acknowledgements

The authors wish to thank the patient subjects who participated in this study, the surgeons at University Hospital, London, Ontario, who provided the stimulation, and Mrs. L. Nolan who helped prepare the manuscript.

Abbreviations

$\dot{V}_I$	inspired minute ventilation
V_T	inspired tidal volume
f	frequency of breathing (breaths/min)
Pa_{CO_2}	arterial carbon dioxide tension
$P_{ET_{CO_2}}$	end-tidal carbon dioxide tension
$P_{ET_{O_2}}$	end-tidal oxygen tension
$\Delta\dot{V}_{I_{45}}$	isocapnic change of inspired minute ventilation from hyperoxia to a $P_{ET_{O_2}}$ of 6.0 kPa (45 torr)
"A"	calculated variable of hypoxic responsiveness (see Methods)
B.T.P.S.	body temperature and ambient pressure, saturated with water vapour
S.T.P.D.	standard temperature and pressure, dry
S.D.	standard deviation
S.E.M.	standard error of the mean

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RÉSUMÉ

Dans le but d'évaluer l'influence de la stimulation chirurgicale sur le contrôle de la ventilation de l'humain sous anesthésie, on a mesuré la ventilation et la réponse ventilatoire à l'hypercapnie en hyperoxie et à l'hypoxémie en isocapnie chez 15 sujets anesthésiés à l'enflurane (1.1 MAC). Les mesures ont été effectuées juste avant la stimulation chirurgicale ainsi que durant la chirurgie. L'anesthésie sans stimulation chirurgicale réduisait la ventilation avec élévation de la Pa_{CO_2}, diminuait la réponse au CO_2 et abolissait virtuellement celle à l'hypoxémie. La chirurgie au même niveau d'anesthésie s'accompagnait d'une augmentation de la ventilation avec diminution de la Pa_{CO_2} mais ne modifiait pas les réponses ventilatoires à l'hypoxémie et à l'hypercarbie produites par l'anesthésie. L'influence de la chirurgie était constante aux valeurs de P_{CO_2} et de P_{O_2} étudiées.