THE EFFECT OF POSTURE ON THE VENTILATORY RESPONSE TO HYPOXIA

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ABSTRACT

The ventilatory response to carbon dioxide is known to be uninfluenced by changes in posture. To obtain similar data on the ventilatory response to hypoxia we studied seven subjects in each of two postures, lying and sitting, at a constant P_{CO_2} midway between end-tidal and mixed venous. Minute ventilation was higher in the seated position than supine ($p < 0.01$) at this isocapnic level, attributable mainly to an increase in tidal volume ($p < 0.05$). However, there were no statistically significant differences in the ventilatory response, the tidal volume response, or the frequency response to hypoxia between the two postures.

WHILE IT IS RECOGNIZED that minute ventilation, alveolar ventilation, P_{CO_2}, and functional residual capacity change with alteration of posture from the supine to the erect posture,[1] it has more recently been reported that indices of ventilatory drive, such as mouth occlusion pressure[2] and the carbon dioxide response,[3] remain the same whether subjects are supine or sitting. We had earlier established that measurements of carbon dioxide response in the supine posture are comparable with measurements made in the sitting posture,[3] and we wished to investigate whether similar conclusions could be drawn for the hypoxic response. As there are no data available concerning postural effects and the hypoxic response, we make this information available in this brief report. These observations become important when clinical studies of ventilatory control are contemplated, since testing the response to hypoxia is usually done in the sitting position; but quite often the results are applied to clinical situations in which the subjects are lying down, such as during sleep or anaesthesia. Our findings indicate that although under conditions of mild hypercapnia minute ventilation is higher in sitting subjects than when they are supine, the ventilatory response to hypoxia is comparable in the two postures.

METHODS

Ventilatory response to hypoxia was measured by a rebreathing method in which P_{CO_2} was held constant. Two measurements were made in each of the seated and supine positions, and the experiments were designed to minimize the effect of order.

Seven subjects (Table I), members of the medical and technical staff of the Toronto General Hospital Respiratory Care Unit, were studied. All were normal healthy adults, without cardiac or respiratory disease. None were aware of the results of any study until all experiments were completed. Written consent was obtained from all the subjects after the protocol for the experiments had been explained to them.

Hypoxia was induced by a rebreathing method, described in greater detail elsewhere[4,5]. The principle of the technique was to have the subject rebreathe from a 7-litre bag that was enclosed in a large bottle, connected to a spirometer. The bag initially had a gas mixture of 24 per cent oxygen, 7 per cent carbon dioxide and the balance nitrogen. The subject rebreathed from this bag, initially taking three large breaths, and the end-tidal P_{CO_2} was held constant at a value midway between the subject's end-tidal and mixed venous P_{CO_2} by drawing gas from the rebreathing bag through a soda-lime carbon dioxide absorbing bypass using a variable-speed pump. We were able to maintain this isocapnic level at ± 0.13 kPa (± 1 mm Hg) for more than 90 per cent of the run. Occasionally the P_{CO_2} exceeded this value but was within ± 0.27 kPa (± 2 mm Hg) for the entire study. The partial pressure of oxygen in the bag could be maintained at any level either by adding oxygen to the bag to increase the P_{O_2} or by letting the P_{O_2} in the bag fall secondary to the subject's oxygen consumption. Gases were analyzed continuously with a paramagnetic rapid oxygen analyzer (Taylor Servomex), 90 per cent response in less then seven seconds, accuracy ± 1 per cent of full scale of calibration range (0–21 per cent), and

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TABLE I

PHYSICAL CHARACTERISTICS OF THE SUBJECTS AND THE ISOCAPNIC LEVELS AT WHICH THE HYPOXIA CHALLENGES WERE APPLIED. THESE ISOCAPNIC LEVELS WERE IDENTICAL FOR THE SUPINE AND SITTING EXPERIMENTS

Subject	Age, year	Sex	Weight (kg)	Isocapnic P_{CO_2} level kP_2 (mm Hg)
1	28	M	84.1	6.12 (46)
2	32	F	62.7	5.85 (44)
3	29	F	59.1	5.72 (43)
4	27	M	65.9	5.99 (45)
5	35	M	75.0	5.99 (45)
6	26	M	73.6	5.85 (44)
7	34	F	56.8	5.99 (45)

infra-red carbon dioxide analyzer (Capnograph, Godart) accurate to ±0.1 per cent over the range 0–9 per cent. Arterial oxygen saturation (Sa_{O_2}) was monitored continuously with a fiberoptic ear oximeter (Hewlett Packard #47201A) that provided an estimate of arterial oxygen saturation accurate to within ±2 per cent over the range 70–100 per cent.

Subjects were studied at least two hours after the last meal and refrained from drinking any fluids, including tea and coffee, during this time. In the seated position, the subject was supported comfortably by a straight-backed chair. In the supine position, the head was rested flat on the mattress. Before each study, resting minute ventilation and end-tidal P_{CO_2} were recorded for ten minutes. Rebreathing was then initiated by turning a three-way tap close to the mouthpiece so that the subject was now breathing in and out of the bag. The P_{CO_2} in the bag was adjusted to the predetermined isocapnic level at which the hypoxic run was to be carried out and was maintained at this level throughout the rest of the experiment using the variable carbon dioxide absorbing bypass. During the next seven minutes oxygen was bled into the circuit at a rate approximately equal to the subject's oxygen consumption to maintain the P_{O_2} in the bag at 22–24 per cent oxygen. This added oxygen was then discontinued and the oxygen concentration in the bag started to fall as a consequence of the subject's oxygen consumption. Rebreathing was continued until the ear oximeter reading was 65–70 per cent. Each study took approximately 20–25 minutes, but the actual period of hypoxia lasted only about 5–7 minutes. The isocapnic level was identical for both the sitting and supine hypoxic challenges. Each subject was studied in a single experimental session of about 2.5 hours.

The data for the first 30 seconds of hypoxia were not used. The rest of the data were analyzed by calculating the tidal volume (V_T), frequency (f), P_{CO_2} and Sa_{O_2} for each breath. Least squares linear regressions were done using Sa_{O_2} as the independent variable, with each of the following as the dependent variable: (i) V_T or (ii) f or (iii) ventilation (= f × V_T). The slopes of these lines were the tidal volume sentitivity ($\Delta V_T/\Delta Sa_{O_2}$), frequency sentitivity ($\Delta f/\Delta Sa_{O_2}$) and ventilatory sensitivity ($\Delta \dot{V}/\Delta Sa_{O_2}$) respectively. Results for the lying and sitting experiments were compared using a t-test for paired variates. Results were considered statistically significant if the p values were less than 0.05.

RESULTS

Physical characteristics of the subjects as well as the isocapnic level at which each of the hypoxic runs was done are presented in Table I. There was no systematic variation in the end-tidal P_{CO_2} between the two postures during the hypoxic runs. As shown in Table II there was no significant difference in ventilatory response to hypoxia between the lying and sitting positions. Minute ventilation under normoxic conditions at the isocapnic level used during the studies was higher in the seated position than in the supine ($p < 0.01$), as was tidal volume ($p < 0.05$). The slight increase in respiratory rate was not statistically significant. The ventilatory response to hypoxia at the end-tidal P_{CO_2} while sitting was 1.2 l/min per one per cent fall in Sa_{O_2} (±S.D. 0.66). Neither this ventilatory response nor frequency response to hypoxia were significantly influenced by changes in posture.

DISCUSSION

These findings may be viewed as base-line data for future studies of ventilatory response to

TABLE II

SUMMARY OF DATA OBTAINED FOR THE SITTING AND LYING POSITIONS. ALL THESE VALUES WERE OBTAINED AT THE VALUES OF P_{CO_2} AT WHICH THE HYPOXIA CHALLENGES WERE APPLIED (SEE TABLE I)

	Saturation (Per cent)		Ventilation ($l \cdot min^{-1}$)		Frequency (breaths $\cdot$ min^{-1})		Tidal volume (litres)	
Subject	Sitting	Lying	Sitting	Lying	Sitting	Lying	Sitting	Lying
1	97.2	97.9	28.6	21.0	19.7	15.2	1.50	1.37
2	98.8	97.4	27.5	24.5	22.8	22.8	1.20	1.08
3	96.0	95.7	30.3	29.4	23.5	24.4	1.30	1.21
4	98.1	98.7	23.3	15.7	28.5	24.0	0.82	0.66
5	96.0	98.6	27.6	17.0	10.0	9.3	2.73	1.88
6	96.0	96.5	38.1	24.1	26.4	20.3	1.45	1.20
7	97.9	98.0	24.1	15.9	20.5	19.1	1.17	0.83
Mean	97.15	97.53	28.52	21.09	21.65	19.31	1.45	1.18
±S.D.	±1.17	±1.11	±4.89	±5.22	±5.22	±5.99	±0.606	±0.394
t-Stats	−0.84		4.46		2.29		2.75	
Statistical Significance	N.S.		$p < 0.01$		N.S.		$p < 0.05$	

TABLE III

SUMMARY OF HYPOXIC SENSITIVITY DATA. ALL DATA WERE OBTAINED AT THE ISOCAPNIC LEVELS SHOWN IN TABLE I. THERE WERE NO STATISTICALLY SIGNIFICANT DIFFERENCES IN ANY OF THE PARAMETERS BETWEEN THE SITTING AND LYING POSITIONS

	Ventilatory sensitivity ($-\Delta\dot{V}/\Delta Sa_{O_2}$) ($l \cdot min^{-1} \cdot \%Sa_{O_2}$)		Frequency sensitivity ($-\Delta f/\Delta Sa_{O_2}$) (breaths $\cdot$ $min^{-1} \cdot Sa_{O_2}$)		Tidal volume sensitivity ($-\Delta V_T/\Delta Sa_{O_2}$) ($l \cdot \%Sa_{O_2}^{-1}$)	
Subject	Sitting	Lying	Sitting	Lying	Sitting	Lying
1	2.38	1.78	0.35	0.17	0.069	0.083
2	1.46	1.40	0.44	0.31	0.029	0.038
3	0.55	0.63	0.05	0.05	0.020	0.023
4	0.85	0.78	0.17	0.27	0.022	0.020
5	0.62	0.73	0.13	0.15	0.024	0.035
6	1.64	1.49	0.10	0.06	0.072	0.067
7	0.99	0.66	0.30	0.16	0.023	0.022
Mean	1.212	1.069	0.222	0.168	0.037	0.041
±S.D.	±0.656	±0.474	±0.144	±0.099	±0.023	±0.025
T-Stats	1.53		1.40		−1.50	
Statistical Significance	N.S.		N.S.		N.S.	

hypoxia in subjects whose clinical condition mitigates against adopting a sitting position and in patients before anaesthesia or with diseases such as sleep apnoea syndrome, in which the subjects' posture during the hypoxic challenge may be different from their posture during the clinically relevant period.

Our findings are neither surprising nor controversial. They are in accord with our earlier study of the effect of posture on the carbon dioxide response[3] and with that of Burki[2] who, with a similar experimental design, measured mouth occlusion pressure in the two postures. It is important to point out that our studies were carried out at isocapnic levels that were greater than the subject's normal end-tidal P_{CO_2}. We were careful to ensure that this level of carbon dioxide was identical between the two postures and that there was no systematic difference in the P_{CO_2}'s throughout the measurement period in each posture. We chose this higher level of P_{CO_2} because we thought that this would amplify any differences in response since, from the previous studies mentioned, we expected that the ventila-

tory response to hypoxia might be the same in both postures. It might be argued that the results reflect not only the response to hypoxia but to the hypoxia-carbon dioxide interaction. Although this is true, it is also true for any level of P_{CO_2} chosen, even normal end-tidal P_{CO_2}, since it is difficult to entirely remove carbon dioxide as an interacting variable. In addition, it is not entirely certain which is the ideal P_{CO_2} at which to do hypoxia testing[5] and it has been suggested that to characterize the hypoxia response entirely it is necessary to do hypoxic testing at two or more levels of carbon dioxide.[6] We have previously shown that the hypoxic response ($\Delta\dot{V}/\Delta Sa_{O_2}$) is a linear function of P_{CO_2}[7] and thus it seems likely that our results are valid for the entire range of P_{CO_2} at which hypoxic testing is usually done. If this were not correct, then the hypoxic response at normal levels of end-tidal carbon dioxide must change in the opposite direction to the hypoxic response at the mixed-venous carbon dioxide level, since the linear plot of $\Delta\dot{V}/Sa_{O_2}$ vs. P_{CO_2} passes through the same point at the isocapnic level studied in both postures.

The supine position has been suggested as more suitable for measuring the response of mouth occlusion pressure because of changes in functional residual capacity during carbon dioxide rebreathing in the sitting position.[8] As hypoxic ventilatory drive is proving a valuable clinical tool in patients with a variety of serious disorders,[9-11] we were anxious to establish whether both tests were free from postural influences. Our results indicate that measurements of hypoxic response in the supine posture are comparable with measurements made in the sitting posture conventionally used in this method.

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RÉSUMÉ

On sait déjà que la réponse ventilatoire au gaz carbonique ne subit pas l'influence des changements de posture. Dans le but de comparer la relation entre les changements posturaux et la réponse à l'hypoxie, sept sujets ont été étudiés en position dorsale et assise à une P_{CO_2} constante située à mi-chemin entre la P_{CO_2} de fin d'expiration et celle du sang veineux mêlé. La ventilation-minute fut trouvée plus élevée en position assise qu'en position couchée ($P < 0.01$) à ce degré d'isocapnie et cette élévation est attribuable à une augmentation du volume courant ($P < 0.05$). Cependant, on n'a pas trouvé de différences significatives entre les deux postures lorsque furent étudiées la réponse ventilatoire, la réponse du volume courant et la réponse de la fréquence à l'hypoxie.