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SHORT COMMUNICATION

Paradoxical evolution of arterial blood gases during exercise in patients with chronic obstructive bronchitis

A. Frans*, E. Lampert**, N. Tuo**, Th. Clerbaux*, W. Nullens*, J. Lonsdorfer

Paradoxical evolution of arterial blood gases during exercise in patients with chronic obstructive bronchitis. A. Frans, E. Lampert, N. Tuo, Th. Clerbaux, W. Nullens, J. Lonsdorfer.

ABSTRACT: In more than a thousand chronic obstructive lung disease (COLD) patients we have observed in six cases, that exercise-induced hypercapnia was accompanied by a concomitant improvement in arterial oxygen tension (Pao_2) and a decrease in the alveolo-arterial O_2. This behaviour was not due to technical errors. We explained the increase in Pao_2 during exercise by three nonmutually exclusive mechanisms: 1) an increase in the respiratory quotient; 2) the exercise induced increase in alveolar ventilation, although inadequate to match the increase in CO_2 production, would be redistributed to previously poorly ventilated regions of the lung, these regions therefore receiving enough oxygen to arterialize the blood flowing through the alveolar capillaries; 3) alternatively, perfusion would be redistributed in a more efficient way during exercise, so that even in the presence of hypoventilation, oxygenation would be improved.

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* Cliniques Universitaires St-Luc, Brussels, Belgium.

** Service des Epreuves Fonctionnelles Respiratoires, Hôspices Civils de Strasbourg, Strasbourg, France.

Correspondence: A. Frans, Cliniques Universitaires St-Luc, 10, Avenue Hippocrate, B-1200 Brussels, Belgium.

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In patients with chronic obstructive bronchitis, hypoxaemia and hypercapnia, sometimes observed at rest, are often aggravated during exercise. By reviewing the results of the arterial blood gases performed in more than a thousand patients in our two laboratories (Brussels and Strasbourg), we have observed, in six patients, that an increase in arterial carbon dioxide tension ($Paco_2$) was accompanied by a parallel increase in arterial oxygen tension (Pao_2) during exercise.

An example of this behaviour is given by patient R.M. He was 65 yrs old and was a schoolmaster. He began to smoke at age 14. Since his sixties he had chronic cough, expectoration and breathlessness (stage III). He was obese (83 kg for 1.68 m), cyanotic and had wheezing. His chest X-ray and electrocardiogram were normal. His ventilatory function is given in table 1. O_2 saturation was measured and not calculated. Alveolar P_{O_2} (P_{AO_2}) was calculated by an alveolar air equation. The same measurements were repeated one week later and the same results were obtained.

Our results may not be attributed to technical faults since the blood indices were always measured twice, the arterial saturation was measured and not calculated, and the same experiment was repeated one week later.

It is a common finding that hypoxaemia at rest decreases or disappears in normocapnic patients. This has been shown for the first time by SIMONSSON *et al.* [1] who investigated forty-eight bronchitic patients classified into three groups according to the impairment of their FEV_1. In group II alveolar-arterial O_2 tension difference ($P(A\text{-}a)O_2$) fell from 3.2 at rest to 2.7 kPa during exercise and in group III from 3.9–2.8 kPa. Only two of them were hypercapnic.

The same trend was observed in 12 out of 42 coalminers with simple pneumoconiosis [2], in patients with mild bronchitis or asymptomatic asthma [3], or in hypoxaemic patients without airway obstruction [4]. This behaviour is classically attributed to a more homogeneous distribution of the ventilation-perfusion ratio ($\dot{V}A/\dot{Q}$) [5]. For instance, LEVINE *et al.* [3] divided the lung of their patients into a slow and a fast space. During exercise, alveolar ventilation increases more than cardiac output, thereby increasing the $\dot{V}A/\dot{Q}$ of the lung as a whole. This would tend to increase the ratios of both slow and fast spaces, improving it in the former but worsening it in the latter.

The most striking contribution of our work is to show that alveolar hypoventilation, as evidenced by hypercapnia, may be accompanied by an improvement of the Pao_2. In our subject, a part of the increase of Pao_2 during exercise is due to the increase in the respiratory quotient. This amounts to 0.5 kPa only and cannot explain the decrease in the $P(A\text{-}a)O_2$ by 1.1 kPa.

We have therefore to assume that an increase in alveolar ventilation, although inadequate to match the exercise-induced increase in CO_2 production, would be redistributed to previously poorly ventilated regions of the lung, these regions therefore receiving enough

Table 1. – Ventilatory function of patients R.M.

VC:		2.37 *l*	RV: 3.52 *l*
FEV_1:		0.97 *l*	TLC: 5.89 *l*
FEV_1 After Salbutanol:		0.95 *l*	TLCO: 22.8 $ml \cdot min^{-1} \cdot torr^{-1}$
			: 7.64 $nmol \cdot min^{-1} \cdot kPa^{-1}$

		Rest	Exercise
Sao_2	%	88.9	89.7
Pao_2	torr	56	61
	kPa	7.4	8.1
$Paco_2$	torr	47	53
	kPa	6.2	7.1
R		0.84	0.90
PAO_2	torr	95.4	91.1
	kPa	12.7	12.1
$P(A\text{-}a)O_2$	torr	40	30
	kPa	5.3	4.0
pH		7.41	7.33

VC: vital capacity; FEV_1: forced expired volume in one second; RV: residual volume; TLC: total lung capacity; TLCO: transfer factor for carbon monoxide by the single-breath method; R: respiratory quotient; Sao_2: O_2 saturation of arterial blood; Pao_2: arterial O_2 tension; $Paco_2$: arterial CO_2 tension; PAO_2: alveolar O_2 tension; $P(A\text{-}a)O_2$: alveolar-arterial O_2 tension difference.

oxygen to arterialize the blood flowing through the alveolar capillaries. Alternatively, perfusion may be redistributed in a more efficient way during exercise, so that even in the presence of hypoventilation oxygenation may be improved.

To our knowledge, this is the first description of such an evolution of arterial Po_2 during exercise. This is probably due to the fact that means rather than individual values are generally published. For instance in the third group of patients examined by SIMONSSON *et al.* [1] the mean $Paco_2$ was within normal limits except for two patients who had values below 6.0 kPa at rest. The individual evolution of these two patients during exercise is not given.

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Evolution paradoxale des gaz du sang artériel au cours de l'effort, chez des patients atteints de bronchite chronique obstructive. A. Frans, E. Lampert, N. Tuo, Th. Clerbaux, W. Nullens, J. Lonsdorfer.

RÉSUMÉ: Sur plus de mille fonctions artérielles provenant de patients broncho-emphysémateux nous avons observé six cas où l'effort aggravait l'hypercapnie présente au repos alors que la Pao_2 s'améliorait et que la différence alvéolo-artérielle d'O_2 diminuait. Ce comportement paradoxal n'était par dû à des erreurs techniques. Nous expliquons l'augmentation de la Pao_2 à l'effort chez ces patients par: 1) l'augmentation du quotient respiratoire et par deux autres mécanismes agissant alternativement ou de concert; 2) l'augmentation de la ventilation alvéolaire à l'effort, quoiqu' insuffisante pour éliminer le CO_2 produit par l'effort, se redistribue à des zônes pulmonaires mal ventilées au repos. Ces zônes reçoivent ainsi assez d'oxygène pour artérialiser le sang qui leur parvient; 3) la perfusion se redistribue d'une façon telle à l'effort que l'oxygénation du sang peut se produire même en présence d'hypoventilation.

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