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The role of physiological deadspace and shunt in the gas exchange of patients with pulmonary hypertension: a study of exercise and prostacyclin infusion

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The role of physiological deadspace and shunt in the gas exchange of patients with pulmonary hypertension: a study of exercise and prostacyclin infusion. B. Otulana, T. Higenbottam.

ABSTRACT: Haemodynamic and gas exchange measurements were made at rest, on supine exercise and on acute vasodilatation with intravenous prostacyclin in eight patients with pulmonary hypertension. This enabled an assessment of the contribution of $\dot{V}/\dot{Q}$ imbalance to the abnormal gas exchange on the condition. At rest arterial oxygen tension, Pao_2 (mean 8.1±1.7 kPa) and mixed venous oxygen tension, $P\bar{v}o_2$ (3.6±0.4) were reduced. The physiological shunt, $\dot{Q}s/\dot{Q}t$ (15±17%) and the dead space, Vd/Vt (0.47±0.11) were elevated above normal. Exercise produced an increase in cardiac index, a fall in $P\bar{v}o_2$, no significant change in Pao_2 and also no appreciable changes in the Vd/Vt and $\dot{Q}s/\dot{Q}t$. Intravenous prostacyclin increased the cardiac index and raised the Pvo_2 and the Pao_2 but again with no significant changes in Vd/Vt and $\dot{Q}s/\dot{Q}t$. We conclude that ventilation-perfusion imbalance as shown by increased Vd/Vt and $\dot{Q}s/\dot{Q}t$, contributes significantly to the abnormal gas exchange in pulmonary hypertension. But neither index was altered by exercise or vasodilation; the latter improves the hypoxemia by increasing the $P\bar{v}o_2$ from an increase in the cardiac output.

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Pulmonary hypertension from plexogenic pulmonary arteriopathy [1], peripheral thrombotic arteriopathy [2] and proximal thromboembolic disease [3] results from obstruction and "obliteration" of the pulmonary vascular bed [4]. Abnormal pulmonary gas exchange is a probable consequence and has been well described in pulmonary hypertension [5, 6]. A widened alveolar-arterial oxygen gradient $D(A\text{-}a)o_2$ occurs at rest and small increases in work during exercise are accompanied by worsening hypoxia. This probably accounts for the exercise intolerance characteristic of this disorder. Two pathophysiological processes are believed to contribute to the widened $D(A\text{-}a)o_2$. Ventilation-perfusion imbalance occurs, as demonstrated by an enlarged deadspace together with an increased right to left intrapulmonary shunt [7, 8]. The effects of this imbalance of ventilation and perfusion are accentuated by a low mixed venous oxygen content ($P\bar{v}o_2$) [9, 10] caused by a reduced cardiac output ($\dot{Q}t$) [11]. During exercise increased hypoxia appears to be due to the fall in $P\bar{v}o_2$ and its impact on the end-capillary tension of the shunt [7].

There remains, however, uncertainty as to the relative contribution of the ventilation-perfusion imbalance and the low $P\bar{v}o_2$ to the widened $D(A\text{-}a)o_2$. This uncertainty reflects the differences in methods used to study the extent of ventilation-perfusion imbalance [7, 9], as well as considerable heterogeneity in the severity of the disease in the patient groups studied [9, 10, 12].

As a result of this uncertainty and because the effectiveness of treatment is judged by the ability to improve exercise tolerance, we have undertaken further studies of pulmonary gas exchange in patients with pulmonary hypertension due to pulmonary vascular disease. The patients, who had all been considered for heart-lung transplantation, represent a uniform group with a poor three year prognosis of only 17% [13]. Using the methods of RILEY *et al.* [14] we studied the effects of exercise and pulmonary vasodilatation, achieved with intravenous prostacyclin (PGI_2) [15, 16], on the size of the physiological deadspace and the right to left intrapulmonary shunt.

Patients and methods

Five patients with primary pulmonary hypertension (PPH), without evidence of proximal pulmonary artery thromboembolism on ventilation-perfusion scintigraphy and three patients with pulmonary angiographic evidence of proximal thromboembolic disease were studied. Each had given written informed consent, and the study had the approval of the local ethics committee. No patient had evidence of intracardiac shunt on previous catheter study. Dynamic lung volumes were measured using a dry wedge spirometer (Vitalograph Ltd, Buckingham, England). Single-breath gas transfer for carbon monoxide was also recorded (Transfer test, PK Morgan, Chatham, Kent).

A triple lumen right heart catheter was inserted *via* the internal jugular route into the pulmonary artery and positioned under fluoroscopic control. The patient had fasted for eight hours before cardiac catheterization. Recordings of right atrial pressure ($\bar{P}ra$), mean pulmonary artery pressure ($\bar{P}pa$) and pulmonary artery wedge pressure (Ppaw) were made in the supine position. Cardiac output was measured in triplicate by thermodilution. Mean systemic artery presssure ($\bar{P}sa$) was measured from an arterial cannula. Arterial and mixed venous blood samples were taken for direct measurement of oxygen tension using an ABC 3 Radiometer gas analyser (Copenhagen, Denmark). Oxygen content of arterial and venous blood were estimated in the standard fashion [17].

Rate of oxygen consumption ($\dot{V}o_2$) and rate of carbon dioxide output ($\dot{V}co_2$) were measured using analysis of mixed expired gas. The patient breathed through a low resistance valve (Otis-McKerrow) to a five litre baffled box sampled with a mass spectrometer (Centronic MGA 200). A combination of pneumotachograph, differential manometer and integrator (PK Morgan, Chatham, Kent, England) was used to record expired volume. The concentrations of O_2 and CO_2 and the expired volume were recorded on a four channel recorder (Gould Electronics Ltd, Essex). Standard equations were used to calculate $\dot{V}o_2$ and $\dot{V}co_2$.

Exercise consisted of four minutes of alternate leg raising in a supine posture. The technique of intravenous infusion of prostacyclin has been described previously [15]. The dose was increased until either a 20% fall in pulmonary vascular resistance occurred or the Pas fell by 20%. Two patients were considered too ill to exercise and one patient did not receive prostacyclin.

The wasted ventilation fraction Vd/Vt was calculated using a modified Bohr formula [18]:

$$Vd/Vt=\frac{Paco_2-Peco_2}{Paco_2}$$

where $Paco_2$=arterial carbon dioxide tension and $Peco_2$=expired gas carbon dioxide tension.

Physiological shunt $\dot{Q}s/\dot{Q}t$ was calculated as follows:

$$\dot{Q}s/\dot{Q}t\times100\%=[(Cc'o_2-Cao_2)/(Cc'o_2-C\bar{v}o_2)]\times100$$

where $\dot{Q}s$=shunt in $l\cdot min^{-1}$; $\dot{Q}t$=cardiac output; $Cc'o_2$=oxygen content in pulmonary capillary; Cao_2=arterial oxygen content; $C\bar{v}o_2$=mixed venous oxygen content.

The $Cc'o_2$ was derived from the alveolar oxygen tension which was calculated using the alveolar gas equation.

The alveolar-arterial oxygen difference $D(A\text{-}a)o_2$ was calculated as:

$$D(A\text{-}a)o_2=\frac{(Pio_2-Paco_2)-Pao_2}{R}$$

where Pio_2=ideal compartment oxygen tension and was derived from the alveolar gas equation and R=measured respiratory exchange ratio.

It has been shown previously [19] that application of the concept of an ideal alveolar gas for estimating $D(A\text{-}a)o_2$ in both steady and non-steady state exercises does not give significantly different results.

We were able to check the presence of intracardiac shunts by inspecting the kidney for uptake of ^{99m}Tc macroaggregated albumin (MAA) during lung scintigraphy in all patients; and in three patients who subsequently died and two patients who underwent heart-lung transplantation the heart was inspected for intracardiac shunts.

Paired t-testing was used to establish the significance of the changes in haemodynamic and gas exchange measurements compared to resting values.

Results

Only one patient had minimal evidence of airflow obstruction on spirometry (table 1). All had reduced values of pulmonary carbon monoxide transfer factor (TLco). All were hypoxic at rest (table 2) and had elevated pulmonary artery pressure with reduced cardiac index (CI).

No patient showed evidence of kidney accumulation of ^{99m}Tc MAA at lung scintigraphy. In the five patients where macroscopic examination was possible during necropsy or pathological examination of transplant material, no intracardiac shunt could be demonstrated (table 1).

At rest, there was considerable heterogeneity in the degree of ventilation-perfusion imbalance. The Vd/Vt ranged from 0.32 to 0.66, (mean 0.47±0.11 SD). The shunt fraction of cardiac output ranged from 2.64 to 55.42% (mean 15.2±9.0 SD) (table 2). In all patients the $P\bar{v}o_2$ was reduced at rest (table 2).

Gas exchange data at rest, on exercise and on intravenous prostacyclin infusion are shown in table 3. The mean maximum $\dot{V}o_2$ achieved during the supine exercise was 25.4±7.5 $mmol\cdot min^{-1}$ (table 3). This exercise produced a fall in $P\bar{v}o_2$ ($p=0.005$) and Pao_2 (though not statistically significant, $p=0.57$) with a widening of the $D(A\text{-}a)o_2$. However, overall there was no significant change in Vd/Vt ($p=0.05$) or $\dot{Q}s/\dot{Q}t$ ($p=0.44$) (fig. 1). The mean maximum dose of prostacyclin was 6.7±0.9 $ng\cdot kg^{-1}\cdot min^{-1}$. With these doses, cardiac output rose ($p<0.001$) and $P\bar{v}o_2$ increased ($p=0.02$) but this increase in cardiac output, unlike that during exercise, was achieved without a rise in $\bar{P}pa$ (fig. 2). In line with the rise in $P\bar{v}o_2$ the Pao_2 increased, this time quite significantly ($p<0.001$). The $D(A\text{-}a)o_2$ narrowed but again there were no significant changes in Vd/Vt ($p=0.25$) or $\dot{Q}s/\dot{Q}t$ ($p=0.07$) (fig. 2).

Discussion

Our results support the view [7] that the ventilation-perfusion imbalance is not minimal in patients with pulmonary hypertension. In our patients Vd/Vt and $\dot{Q}s/\dot{Q}t$ exceed normal values [20]. We found no support for the view that vasoconstriction promotes matching between ventilation and perfusion, since PGI_2-induced vasodilatation failed to alter either shunt or deadspace consistently. However, we confirm the central role of the

Table 1. – Resting pulmonary function data in the patients

Subject	$\dot{V}/\dot{Q}$ Scan	Age yrs	Sex	% Pred FEV_1	% Pred FVC	FEV_1/FVC %	% Pred TLCO
JM	Normal	34	M	80	92	67	63
GW	Normal	32	M	100	100	76	61
DO*	Abnormal	17	M	90	80	76	63
AF*	Normal	39	F	125	119	81	26
IC*	Normal	32	F	85	110	95	33
AC*	Abnormal	40	M	88	79	86	50
MT	Abnormal	35	M	87	88	75	65
PD*	Normal	25	M	83	120	83	68

*: Patient had the heart inspected for shunt following heart-lung transplantation or death. Abnormal $\dot{V}/\dot{Q}$ scan implies finding of significant proximal segmental defects.

Table 2. – Baseline cardiopulmonary indices in the patients

Subject	$\bar{P}pa$ kPa	CI $l \cdot min^{-1} \cdot m^2$	Pa_{O_2} kPa	Pa_{CO_2} kPa	$P\bar{v}_{O_2}$ kPa	$D(A\text{-}a)O_2$ kPa	$\dot{Q}s/\dot{Q}t$ %	Vd/Vt
JM	14.0	2.1	7.2	3.2	3.2	7.6	16.5	0.44
GW	11.7	1.8	9.1	2.8	3.2	6.9	5.9	0.37
DO	10.7	1.9	10.5	4.4	3.7	2.8	2.6	0.66
AF	8.3	2.0	5.2	4.0	3.5	10.1	55.4	0.53
IC	6.7	1.7	7.9	3.2	3.6	7.5	11.1	0.53
AC	7.1	2.2	7.5	4.0	3.5	6.4	15.6	0.48
MT	7.3	1.3	9.6	5.1	3.9	6.3	9.3	0.32
PD	5.2	1.8	7.5	3.9	4.3	7.9	5.4	0.42
Mean	8.9	1.85	8.1	3.8	3.6	6.9	15.2	0.47
SD	3.0	0.3	1.7	0.7	0.4	2.1	16.9	0.11

Abbreviations as defined in the text.

Table 3. – Gas exchange data at rest, on exercise and following intravenous prostacyclin infusion

	Rest			Exercise			Prostacyclin		
	$\dot{V}_{O_2}$	$\dot{V}_{CO_2}$	R	$\dot{V}_{O_2}$	$\dot{V}_{CO_2}$	R	$\dot{V}_{O_2}$	$\dot{V}_{CO_2}$	R
JM	10.5	6.5	0.62	26.6	16.3	0.61	9.4	5.8	0.62
GW	6.7	4.8	0.72	-	-	-	9.5	7.1	0.75
DO	11.6	6.6	0.57	-	-	-	12.2	8.6	0.70
AF	7.4	6.3	0.86	11.0	13.3	1.21	-	-	-
IC	13.8	9.3	0.68	24.9	18.3	0.74	13.9	8.9	0.64
AC	20.9	13.6	0.65	27.4	17.7	0.65	14.8	9.1	0.62
MT	14.7	20.3	1.22	31.0	47.7	1.54	15.2	11.4	0.75
PD	11.4	9.4	0.83	31.3	31.3	1.00	11.9	10.7	0.90
Mean	12.1	9.6	0.77	25.4	24.1	0.96	12.4	8.8	0.71
SD	4.5	5.1	0.21	7.5	13.1	0.37	2.4	1.9	0.10

Abbreviations as defined in the text. $\dot{V}O_2$ and $\dot{V}CO_2$ are in $mmol \cdot min^{-1}$. Subjects GW and DO were too ill to exercise; AF did not receive prostacyclin.

low value of $P\bar{v}_{O_2}$ in determining the widened $D(A\text{-}a)O_2$ [12] in patients with pulmonary hypertension.

The increased deadspace is not surprising as obstruction of pulmonary blood flow with normal ventilation should produce "wasted" ventilation [21]. In explaining previously reported low values for deadspace in patients with pulmonary hypertension, it has been argued that the "wasted" ventilation could be reduced by two physiological adaptations [9]: hypocapnic bronchoconstriction in underperfused regions was thought to reduce ventilation [22] and alveolar capillary perfusion maintained by means of collateral circulation [7].

Circumstantial evidence in our patients suggested that neither of these physiological adaptations was important.

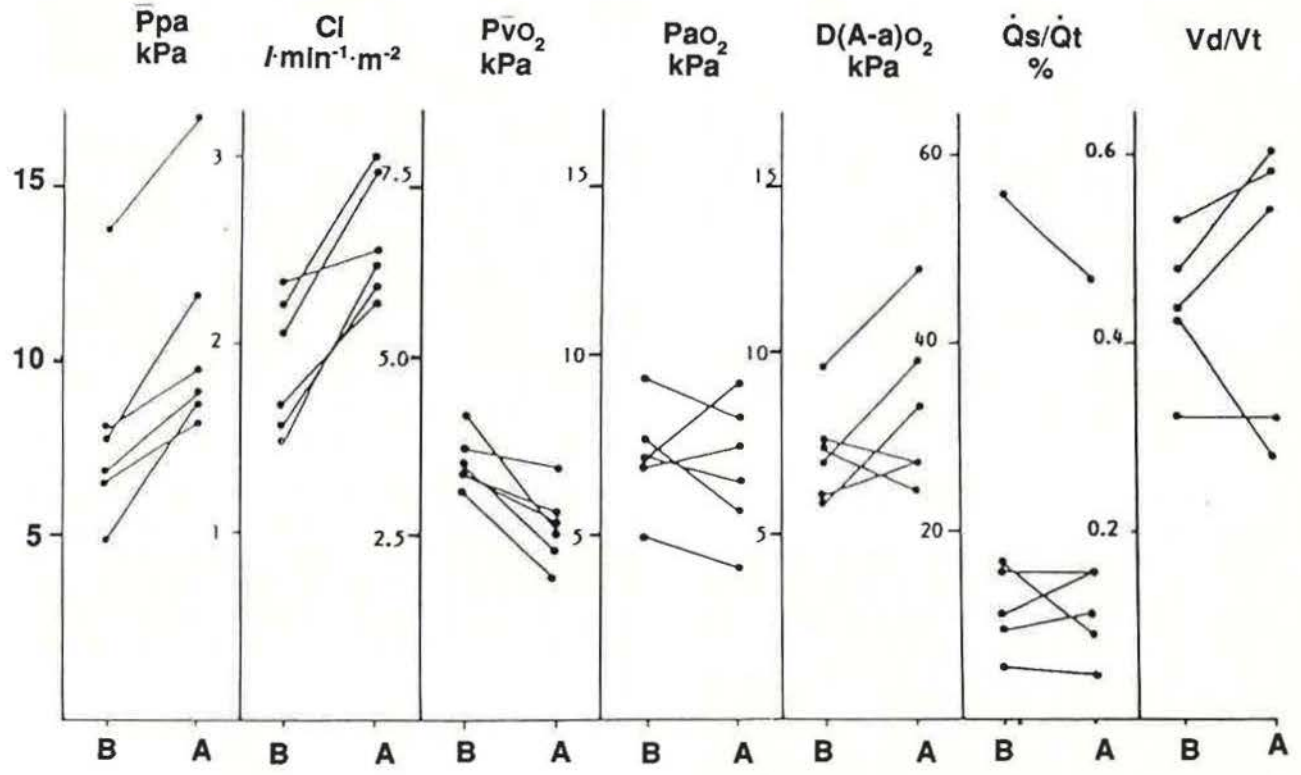

Fig. 1. - A plot of the changes in the various indices from rest to exercise in the individual subject; B: before exercise, A: after exercise.

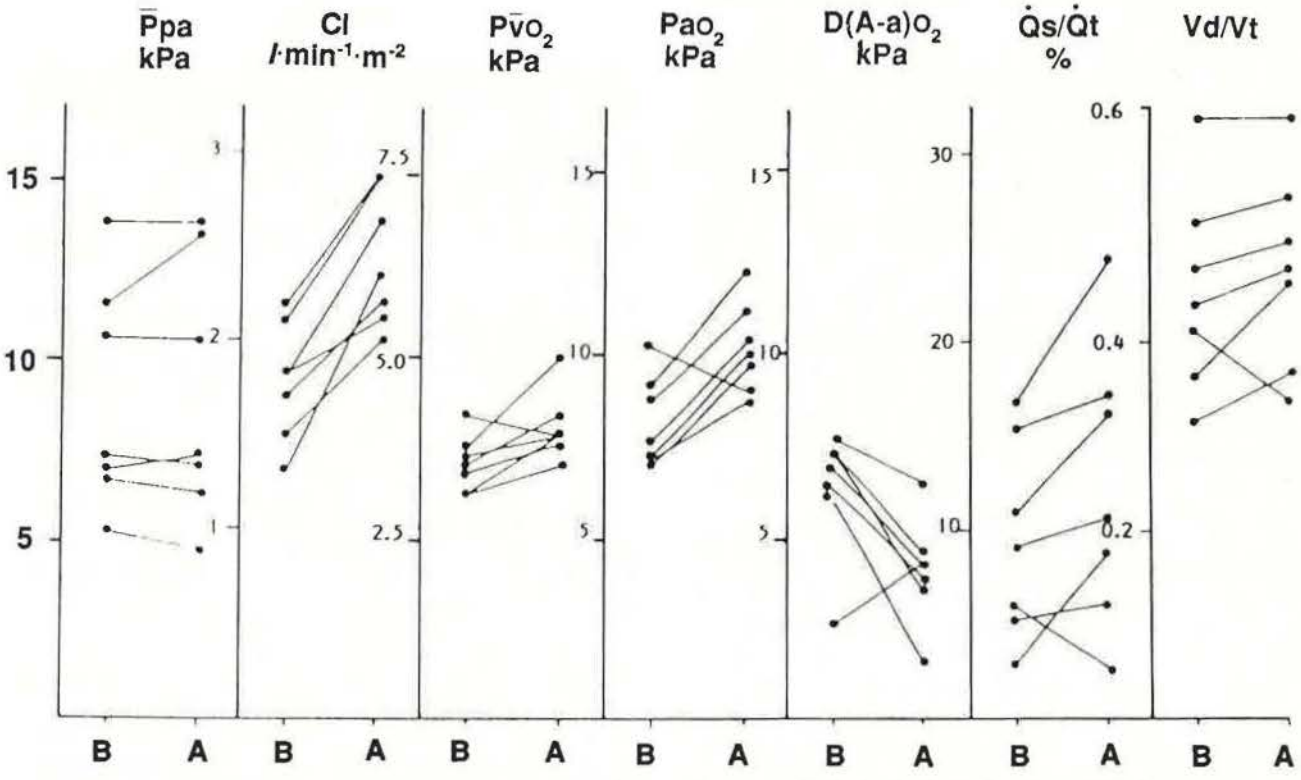

Fig. 2. – A plot of the changes occurring after intravenous infusion of prostacyclin in the individual subject. B: before prostacyclin infusion; A: after prostacyclin infusion.

Only one patient had evidence of airflow obstruction as measured by a reduced forced expiratory volume in one second/vital capacity (FEV_1/VC) ratio and this patient did not have the lowest value of Vd/Vt. The TLCO was reduced in all our patients, which implied a reduction of alveolar capillary volume [23]. This argued against

significant enhancement of collateral circulation to the alveolar capillaries.

We consider that those patients severely affected by pulmonary hypertension do indeed have an enlarged deadspace [7]. The wide variation between patients in the degree of ventilation and perfusion imbalance seen in our present study and earlier work [9, 10, 12] supports this view. The work of DANTZKER and co-workers, using the multiple inert gas elimination technique, suggested only minimal increases in deadspace [9, 10]. However, the patients studied, in terms of their reduction in $P\bar{v}o_2$, were less severely affected than our patients. Also, in a later study of patients with reductions in $P\bar{v}o_2$ comparable to our subjects DANTZKER and co-workers observed values for deadspace size and shunt fraction similar to our values [12]. Furthermore, in this later study [12] both Riley's method and the multiple inert gas elimination technique of Wagner gave very comparable estimates for deadspace and shunt.

The size of the right to left shunt was by no means modest in our patients but is similar to the later study of DANTZKER *et al.* [12]. There is no reason to believe that any of our patients had an intracardiac shunt. Previous findings at earlier catheter studies reduced the chance of this and we observed no accummulation of ^{99m}Tc MAA in the kidneys following lung ventilation-perfusion ($\dot{V}/\dot{Q}$) scintigraphy. Also, after death or transplantation the hearts of five patients were inspected and no septal defects were found. This included the one patient with a shunt fraction of 0.55.

A number of explanations for intrapulmonary shunting have been proposed. Increased blood flow through the remaining normal pulmonary vessels as a result of obliteration of a large portion of the pulmonary vascular bed, is one suggestion [9, 10]. Further contributions could be made by the development of interstitial oedema [24]. This could occur if the pulmonary artery pressure is high enough [20] and could lead to airway closure and an increase in the number of low $\dot{V}a/\dot{Q}$ units. An alternative explanation involves the commonly found histopathological abnormality in pulmonary hypertension, *i.e.* the dilatational lesions which are observed in proximity to the obstructed and narrowed arteries [25]. These dilated venous-like structures represent adaptive changes which facilitate pulmonary blood flow despite a restricted vascular bed [4]. Some of these vessels could provide the route for right to left intrapulmonary shunts.

We were unable to confirm that vasoconstriction in pulmonary hypertension lessens the ventilation-perfusion imbalance [10]. We used a titrated dose of intravenous prostacyclin (PGI_2) to optimally vasodilate the pulmonary vessels in each patient [15, 16]. Cardiac index increased as a result but with little or no effect on Ppa: a phenomenon best explained by pulmonary vasodilatation. Despite this we were unable to demonstrate a significant increase in $\dot{Q}s/\dot{Q}t$, although the general trend was upward, the individual changes were small. Also there was no change in deadspace. These observations do not lend strong support to the idea that vasoconstriction limits ventilation-perfusion imbalance.

The PGI_2 infusion caused not only a rise in cardiac index but also, as anticipated, a rise in $P\bar{v}o_2$ [12]. In parallel the Pao_2 also rose. This emphasized the central importance of $P\bar{v}o_2$ in determining the widened $D(A\text{-}a)o_2$ of these patients.

During exercise the downward trend in Pao_2 also appears to be a consequence of the fall in $P\bar{v}o_2$. Despite the rise in cardiac index there was a fall in $P\bar{v}o_2$ because in exercise the tissues invariably increase their oxygen extraction to meet metabolic demands. A low $P\bar{v}o_2$ is thought to significantly reduce the Pao_2 by decreasing the end-capillary tension of lung units with $\dot{V}a/\dot{Q}$ less than unity and in shunts [26]. This probably accounts for the widened $D(A\text{-}a)o_2$ observed during exercise in all but two patients, even in the absence of significant changes in either deadspace or shunt.

In conclusion, our observations confirm the central role of $P\bar{v}o_2$ in determining hypoxaemia in patients with pulmonary hypertension [12]. However, as anticipated from the pathology of the condition, the imbalance of ventilation and perfusion is large [7] not minimal [9, 10]. The improvement of symptoms with vasodilator treatment is likely to be the consequence of increased cardiac output and the resulting rise in mixed venous oxygen content.

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RÉSUMÉ: Huit malades sans shunts intracardiaques démontrables ont été étudiés afin d'établir les contributions du shunt physilogique et de l'espace mort à l'échange de gaz anormal dans l'hypertension pulmonaire due à la maladie pulmonaire vasculaire. Les mesures hémodynamiques et d'échange de gaz au moyen de prostacycline intraveineuse ont été effectuées au repos, au cours d'exercice en supination, et sur vasodilation aiguë. La tension oxygénique arterielle (Pao_2) (moyenne 8,1±1,7 kPa) et la tension oxygénique veineuse mélangée ($P\bar{v}o_2$) (moyenne 3,6±0,4 kPa) étaient réduites au repos. Les shunts physiologiques ($\dot{Q}s/\dot{Q}t$) (moyenne 15,2±16,9%) et l'espace mort (Vd/Vt) (moyenne 0,47±0,11) étaient élevés. L'exercice a provoqué une augmentation de l'index cardiaque ($p=0,002$), une chute de la $P\bar{v}o_2$ ($p=0,005$), sans changements significatifs de la Pao_2 ($p=0,56$) ni changements appréciables du Vd/Vt ($p=0,45$) ni du $\dot{Q}s/\dot{Q}t$ ($p=0,43$). La prostacycline intraveineuse tout en augmentant l'index cardiaque ($p<0,001$) et a élevé la $P\bar{v}o_2$ ($p=0,02$) et la Pao_2 ($p<0,001$) encore sans changements significatifs des Vd/Vt ($p=0,25$) et $\dot{Q}s/\dot{Q}t$ ($p=0,07$). Nous en concluons que le déséquilibre ventilation/perfusion tel qu'il est démontré par des Vd/Vt et $\dot{Q}s/\dot{Q}t$ augmentés contribue fortement à l'échange de gaz anormal dans l'hypertension pulmonaire, mais ces indexes restent inchangés par l'exercice ou la vasodilation provoquée par les médicaments; cette dernière améliore l'hypoxémie en augmentant la $P\bar{v}o_2$ suite à l'exaltation du débit cardiaque.