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Relationship Between Mixed Venous Oxygen Saturation and Cardiac Index in Patients with Chronic Congestive Heart Failure*

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The use of mixed venous oxygen saturation ($S\bar{v}O_2$) in patients with chronic congestive heart failure (CHF) has been advocated to analyze the action of therapy on cardiac index (CI). To evaluate the relationship between CI and $S\bar{v}O_2$, ten CHF patients (mean age 65 years) were studied before and one, two, three, four (T4), six, eight and 24 hours after oral administration of an angiotensin converting enzyme (ACE) inhibitor (perindopril, 4 mg). At T4, a 12 percent increase in CI ($p < 0.01$) was associated with a 16 percent decrease in arteriovenous oxygen difference ($p < 0.01$), a 13 percent increase in mixed venous oxygen pressure ($P\bar{v}O_2$) ($p < 0.01$), and a 9 percent increase in $S\bar{v}O_2$ ($p < 0.05$) with no significant change in arterial oxygen pressure. There was no correlation between CI and $S\bar{v}O_2$ ($r = 0.22$) and between CI and $P\bar{v}O_2$ ($r = 0.23$). Individual analyses were performed and patients were divided into two groups based on CI versus

$S\bar{v}O_2$, r value; group 1, $n = 6$, $r > 0.65$ (0.65-0.90), group 2, $n = 4$, $r < 0.65$ (0.14-0.20). The lack of correlation in group 2 was due to a drug-dependent increase in oxygen consumption ($\dot{V}O_2$) +18 percent vs -3 percent in group 1 ($p < 0.05$) associated with a lack of increase in $P\bar{v}O_2$ +3 percent vs +14 percent in group 1 ($p < 0.05$) despite a similar increase in oxygen availability +19 percent versus +16 percent. It was concluded that (1) a correlation between CI and $S\bar{v}O_2$ is not found in every patient with CHF; (2) the lack of correlation in four out of our ten patients was due to an associated and significant increase in CI and $\dot{V}O_2$ in group 2; (3) group 2 patients probably had an important oxygen debt before treatment; (4) $S\bar{v}O_2$ cannot be used instead of CI to determine the hemodynamic consequences of the use of cardiovascular drugs.

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Mixed venous oxygen saturation has been reported to be a useful reflection of cardiovascular performance in seriously ill patients.^{1,2} This assumption is valid, in accordance with the Fick equation, when tissue oxygen consumption and arterial oxygen pressure remain stable despite the onset of significant changes in oxygen availability. This potential stability of $\dot{V}O_2$ above the critical threshold of $\dot{D}O_2$, itself the consequence of an inverse relationship between $\dot{D}O_2$ and oxygen extraction ratio, gave sense to a relationship between CI and $S\bar{v}O_2$. Hence, in normal tissues at normal or high levels of delivery, $\dot{V}O_2$ is relatively constant and independent of $\dot{D}O_2$.³ As $\dot{D}O_2$ is gradually reduced, $\dot{V}O_2$ is maintained as tissue ER_{O_2} increases and thus $\dot{V}O_2$ appears to be independent of $\dot{D}O_2$. The lack of relationship between $\dot{V}O_2$ and $\dot{D}O_2$ or rather the relationship between CI and $S\bar{v}O_2$ does not apply to all patients with severely impaired oxygen transport. For example, Danek et al⁴ reported the relationship between $\dot{V}O_2$ and $\dot{D}O_2$ in patients with adult respiratory distress syndrome on mechanical ventilation and Chappell et al⁵ claimed the independence of these two parameters during severe left ventricular failure even in the case of vasodilator therapy. These latter

results were used by several authors to justify the use of $S\bar{v}O_2$ monitoring to evaluate cardiac performance response to various pharmacologic interventions, *ie*, vasodilator or inotropic drugs.^{6,7} The aim of our study, therefore, was to analyze the potential relationship between CI and $S\bar{v}O_2$ in stable patients suffering from left ventricular failure hospitalized for undergoing evaluation of systemic vasodilation with converting enzyme inhibitor.

METHODS

Ten patients with severe chronic CHF (seven men, three women) ranging in age from 41 to 69 years (mean age 65 years) underwent right heart catheterization as part of a protocol to evaluate the effects of ACE inhibitor therapy. The CHF was secondary to ischemic cardiomyopathy in seven and idiopathic in three patients. Five patients were in functional class 3 and 5 in functional class 4 of the NYHA classification. No patient was studied within three months of a myocardial infarction. Patients with valvular disease or elevated plasma creatinine (more than 180 $\mu\text{mol/L}$) were excluded. None had an acute respiratory condition at the time of study. Each patient received, however, 2 to 3 L/min of supplemental oxygen. The study protocol was approved by the hospital ethical committee and informed consent was obtained from all patients prior to study. Patients were fasting, with all medications withheld before testing for at least 24 hours for diuretics and at least six days for vasodilator and digitalis therapy. Right heart catheterization was performed by percutaneous insertion of a 7 F thermodilution, balloon tipped, flow directed catheter into the right jugular vein. An arterial cannula was inserted into the radial artery. Pressures were measured

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Table 1—Hemodynamic Effects of Perindopril 4 mg per Os (n = 10), (Mean ± SEM)

	To	T ₄	T ₂₄
PCWP mm Hg	24.7 ± 1.1	16.1 ± 1.8‡	14.0 ± 1.0‡
MAP mm Hg	88.7 ± 5.9	83.7 ± 4.7	77.0 ± 4.6‡
CO L·min ⁻¹	3.47 ± 0.29	4.04 ± 0.38‡	3.88 ± 0.31†
CI L·min ⁻¹ ·m ⁻²	2.01 ± 0.14	2.34 ± 0.18‡	2.25 ± 0.13†
SI ml·b ⁻¹ ·m ⁻²	23.7 ± 1.9	27.1 ± 2.2‡	28.2 ± 1.6‡
SVR dynes·s·cm ⁻⁵	1942.5 ± 213.7	1692.3 ± 222.9	1592.0 ± 158.4†

To = before treatment, T₄, T₂₄ respectively 4 and 24 hours after treatment; PCWP: pulmonary capillary wedge pressure; MAP: mean arterial pressure; CO: cardiac output; CI: cardiac index; SI: stroke index; SVR: systemic vascular resistance. Comparison T₄, T₂₄ versus To using two way variance analysis complemented by Newman-Keuls test, †p < 0.05, ‡p < 0.01.

with a strain gauge transducer and recorded on a multichannel recorder. Mean arterial pressure and mean pulmonary capillary wedge pressure were obtained by electronic integration with zero reference at the level of the midaxillary line. Heart rate was determined from the simultaneous ECG signal. Cardiac output (CO) was determined in triplicate by thermodilution using a CO computer. Arterial and mixed venous blood samples were collected anaerobically and analyzed using a blood gas analyzer. Blood oxygen content was calculated using the standard formula: O₂ content % saturation × (hemoglobin plasma concentration × 1.39) + (0.003 × P_{O₂}). The CO determination was used to derive oxygen consumption from the Fick equation: $\dot{V}O_2 = CO (CaO_2 - CvO_2)$ where CaO₂ and CvO₂ are arterial and mixed venous oxygen contents respectively. Systemic oxygen availability ($\dot{D}O_2$) was calculated as follows $\dot{D}O_2 = CO \times CaO_2$. The $\dot{V}O_2$ and $\dot{D}O_2$ were normalized for body surface area. The O₂ extraction ratio was calculated as the ratio of $\dot{V}O_2$ and $\dot{D}O_2$. Plasma catecholamine level was determined using the enzymatic method of Brown and Jenner.⁸ After baseline control measurements had been made, a single oral dose of an ACE converting inhibitor, perindopril 4 mg, was given, and then the hemodynamic and gas exchange responses analyzed one, two, five, six, eight and 24 hours after drug intake.

Statistical analysis of the data was performed using a two-way analysis of variance complemented by comparison between time, using a Newman-Keuls test. Linear regression was performed using the method of least squares. When the group of patients was less than five, a nonparametric test (Mann-Whitney) was used. Bilateral hypotheses were used, with an α risk chosen of p < 0.05.

Table 2—Effects of Perindopril 4 mg per os on Gas Exchange (n = 10), (Mean ± SEM)*

	To	T ₄	T ₂₄
PaO ₂ mm Hg	93.7 ± 7.3	92.6 ± 6.6	96.1 ± 6.5
SaO ₂ %	97.3 ± 0.5	97.6 ± 0.5	97.8 ± 0.4
PvO ₂ mm Hg	28.3 ± 1.2	32.1 ± 1.6 ‡	31.2 ± 1.4‡
SvO ₂ %	56.1 ± 2.7	61.6 ± 3.1	61.2 ± 2.7
a $\bar{v}DO_2$ ml·100 ml ⁻¹	6.36 ± 0.36	5.37 ± 0.44‡	5.34 ± 0.34‡
$\dot{V}O_2$ ml·min ⁻¹	221.1 ± 22.7	212.3 ± 23.4	205.9 ± 20.3
$\dot{D}O_2$ ml·min ⁻¹	510.9 ± 40.9	598.2 ± 58.7‡	577.8 ± 51.3†
ERO ₂ %	43.2 ± 2.6	36.4 ± 3.2†	36.5 ± 3.0†

*To before treatment T₄, T₂₄ respectively 4 and 24 hours after treatment; PaO₂ = arterial oxygen pressure; SaO₂ = arterial oxygen saturation; PvO₂ = mixed venous oxygen pressure; SvO₂ = mixed venous oxygen saturation; a $\bar{v}DO_2$ = arteriovenous oxygen difference; $\dot{V}O_2$ = oxygen consumption; $\dot{D}O_2$ = oxygen availability; ERO₂ = oxygen extraction ratio. Comparison T₄, T₂₄ versus To using two way variance analysis complemented by Newman-Keuls test, †p < 0.05, ‡p < 0.01.

RESULTS

Hemodynamic and gas exchange data before, 4 and 24 hours after perindopril administration are shown in Tables 1 and 2. Before treatment, this group of severe heart failure patients was characterized by low CI 2.01 ± 0.14 L·min⁻¹·m⁻² (range 1.40 to 2.50 L·min⁻¹·m⁻²) and high mean PCWP, 24.7 ± 1.1 mm Hg (range 20.0 to 30.0 mm Hg). Basal value of arteriovenous oxygen difference (a $\bar{v}DO_2$) was wide 6.36 ± 0.36 ml·100·ml⁻¹ (range 4.67 to 8.20 ml·100 ml⁻¹) and associated with high level of ERO₂ 41.0 ± 0.04 percent. The hemodynamic effect of oral vasodilator therapy with 4 mg perindopril consisted of a significant decrease in PCWP (p < 0.01): 35 percent at T₄ and 43 percent at T₂₄, and systemic vascular resistance (p < 0.05) 13 percent at T₄ and 18 percent at T₂₄, (p < 0.05) with a significant increase in CI (p < 0.01) 14 percent at T₄ and 12 percent at T₂₄ (p < 0.01). Gas exchange effects of perindopril included a significant increase in P $\bar{v}O_2$ (p < 0.01) 13 percent at T₄ and 10 percent at T₂₄ associated with a significant decrease in a $\bar{v}DO_2$ (p < 0.01) 16 percent at T₄ and 14 percent at T₂₄. Despite the significant increase in $\dot{D}O_2$, $\dot{V}O_2$ did not change significantly, and thus, ERO₂ was reduced (p < 0.05): 12 percent at T₄ and 15 percent at T₂₄. The PaO₂ remained within normal limits. Despite the simultaneous increase in mean CI and P $\bar{v}O_2$, there was neither correlation between CI and P $\bar{v}O_2$ (n = 0.23

Table 3—r Values of Correlation between Cardiac Index and Mixed Venous Oxygen Saturation*

	Group 1 (n = 6) r ≥ 0.65	Group 2 (n = 4) r < 0.65
	1 0.68	2 0.20
	3 0.68	5 0.24
	4 0.65	8 0.17
	6 0.74	10 0.14
	7 0.69	
	9 0.90	

*Calculated for each patient with the eight following measurements before 1, 2, 3, 4, 6, 8 and 24 hours after perindopril 4 mg per os.

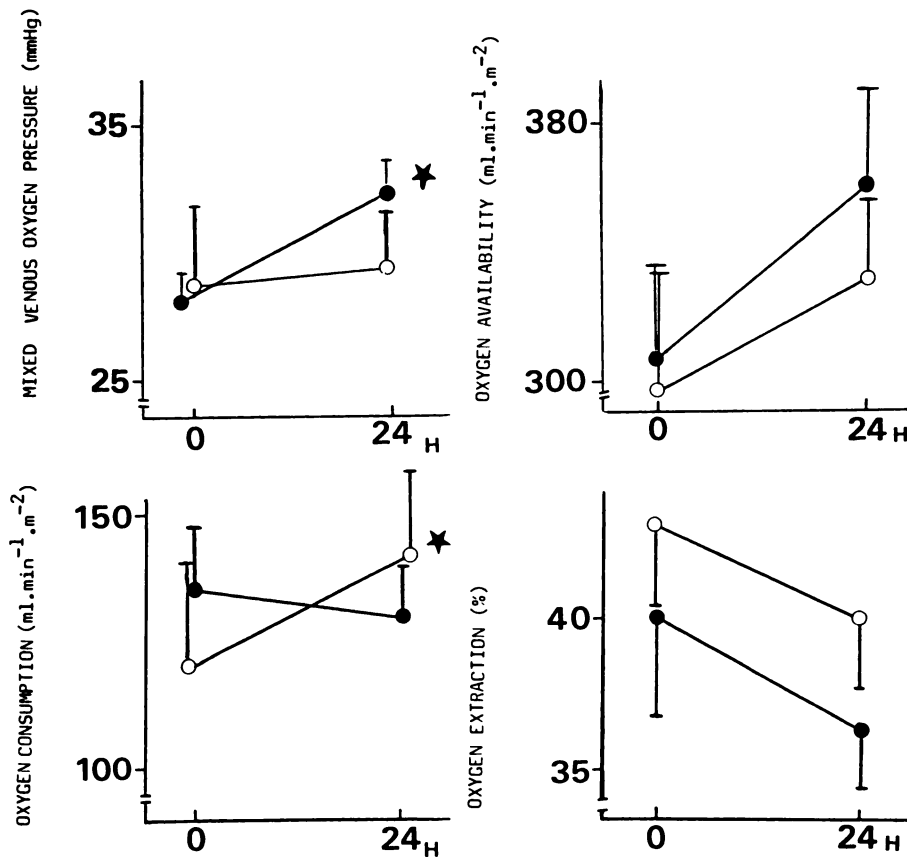


FIGURE 1. Evolution between T₀ (before perindopril) and 24 hours (24 hours after perindopril administration) of mixed venous oxygen pressure, oxygen availability, oxygen consumption and oxygen extraction in group 1 ($r \geq 0.65$) • and group 2 ($r < 0.65$) ○. Comparison T₀ vs 24 hours using Mann-Whitney test (* $p < 0.05$), mean \pm SEM.

df = 68 NS) nor between CI and $\text{S}\bar{\text{v}}\text{O}_2$ when all patients and time controls were pooled. However, individual analyses were performed and patients were divided into two groups, based on CI vs $\text{S}\bar{\text{v}}\text{O}_2$ r value (Table 3)—group 1 $n = 6$, $r > 0.65$ (range, 0.65 to 0.90); group 2, $n = 4$, $r < 0.65$ (range 0.14 to 0.24). The lack of correlation in patients of the group 2 was due to a drug dependent increase in $\dot{\text{V}}\text{O}_2$ (Fig 1) + 18 percent vs -3 percent in group 1 ($p < 0.05$ Mann-Whitney test) associated with a lack of increase in $\text{P}\bar{\text{v}}\text{O}_2$ + 3 percent vs + 14 percent in group 1 ($p < 0.05$) despite a similar increase in $\dot{\text{D}}\text{O}_2$ 19 percent vs 16 percent. The comparison between groups 1 and 2 for hemodynamic, gas exchange and neuroendocrine basal values are shown in Figures 2 and 3. The MAP and mean PCWP were significantly lower in group 2 compared to group 1, being respectively 22 vs 27 mm Hg ($p < 0.05$) and 77 vs 97 mm Hg ($p < 0.05$). The SVR and noradrenaline plasma levels were also slightly reduced in group 2 before treatment, but the difference between groups 1 and 2 was not significant.

DISCUSSION

Despite a simultaneous increase in CO and $\text{P}\bar{\text{v}}\text{O}_2$ after ACE inhibitor administration, our results clearly indicated the lack of global correlation between these two parameters ($r = 0.23$). However, individual analy-

ses using seven sampling points during drug administration showed the presence of a significant correlation between CO and $\text{P}\bar{\text{v}}\text{O}_2$ in six out of ten patients in group 1 (range, r values 0.65 to 0.90) but not in four patients in groups 2 (range, r values 0.14 to 0.24) as a consequence of a significant and associated increase in $\dot{\text{D}}\text{O}_2$ and $\dot{\text{V}}\text{O}_2$ in the latter group.

The acute systemic hemodynamic changes in the ten chronic heart failure patients with low CO and elevated PCWP after ACE inhibition with perindopril (4 mg per os) are consistent with the results of previous studies.^{9,10} They were characterized by a reduction in blood pressure and systemic vascular resistance with a concomitant lowering of the right atrial pressure and PCWP and elevation of depressed cardiac index. These hemodynamic results are associated with the following changes in gas exchange alterations: (1) a significant increase in $\dot{\text{D}}\text{O}_2$ associated with a significant increase in $\text{P}\bar{\text{v}}\text{O}_2$; (2) a significant reduction in arteriovenous oxygen difference without significant change in PaO_2 , and $\dot{\text{V}}\text{O}_2$. Because $\dot{\text{V}}\text{O}_2$ and PaO_2 appear to be unchanged on ACE inhibition with perindopril, the reduction in arteriovenous oxygen difference and increase in $\text{P}\bar{\text{v}}\text{O}_2$ seen in response to perindopril can be obviously attributed to a significant increase in CO associated with a significant reduction of $\text{E}\text{R}\text{O}_2$. The lack of effect of ACE inhibition on PaO_2 has been

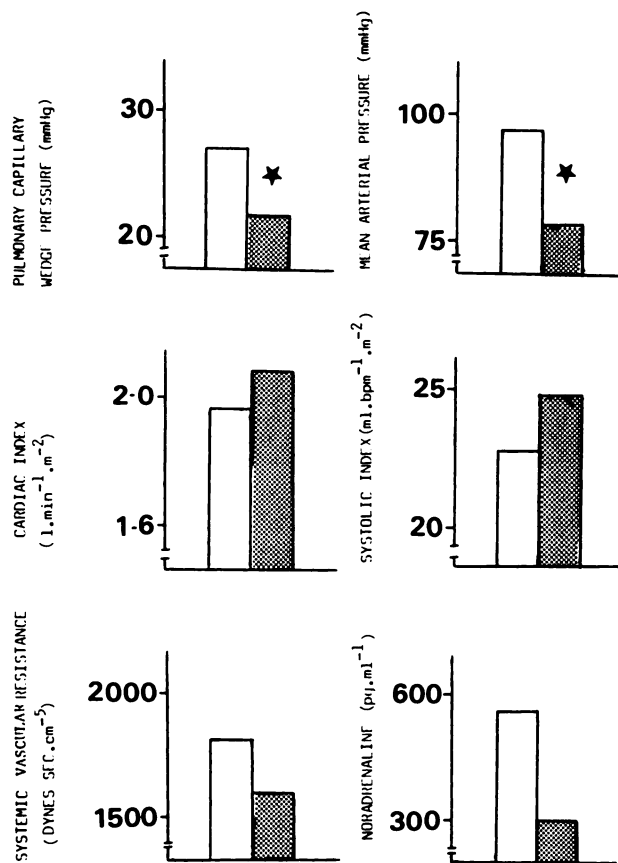


FIGURE 2. Comparison of basal hemodynamic values between group 1 ($r \geq 0.65$)□ and group 2 ($r < 0.65$)■ of pulmonary capillary wedge pressure, cardiac index, systemic vascular resistance, mean arterial pressure, systolic index and of plasma noradrenaline. Between group comparison using Mann-Whitney test * $p < 0.05$.

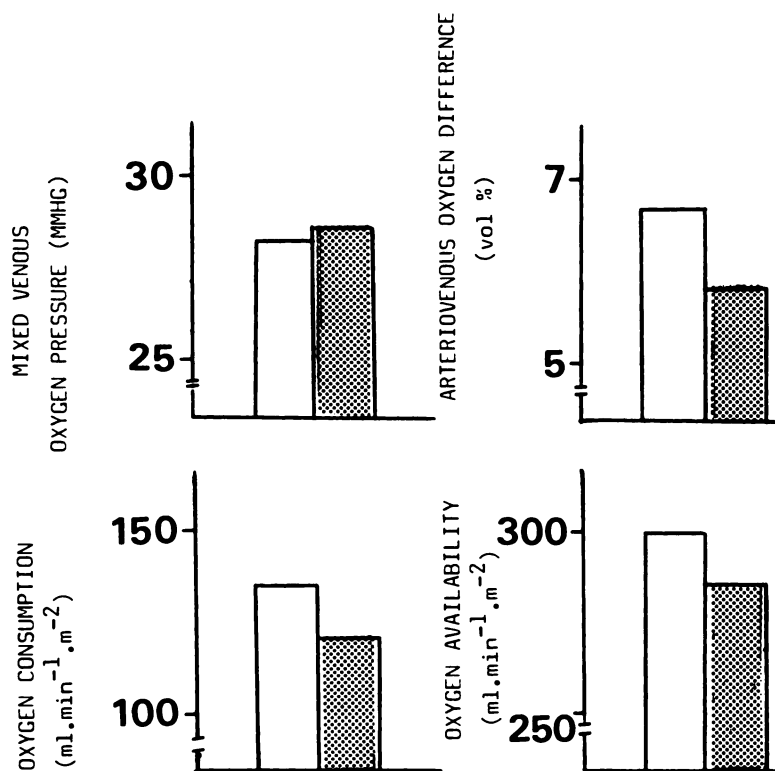


FIGURE 3. Comparison of basal gas exchange values between group 1 ($r \geq 0.65$)□ and group 2 ($r < 0.65$)■ of mixed venous oxygen pressure, oxygen consumption, arteriovenous oxygen difference and oxygen availability. Between group comparison using Mann-Whitney test.

previously reported with captopril and was linked to the absence of effect of these compounds on pulmonary hemodynamics.^{9,11} Accordingly, vasodilators which have prominent pulmonary vascular effects can decrease PaO_2 in patients with CHF, this effect being most likely due to increasing ventilation-perfusion inequalities.¹² Previous studies^{11,13} indicated that, as with other vasodilators and in accordance with our results, ACE inhibitors systematically increased $\dot{\text{V}}\text{O}_2$ as a consequence of CI improvement. Despite the increase in $\dot{\text{V}}\text{O}_2$, $\dot{\text{V}}\text{O}_2$ remained stable at rest and during exercise after administration of an ACE inhibitor.^{10,14}

The $\text{P}\bar{\text{v}}\text{O}_2$ or $\text{S}\bar{\text{v}}\text{O}_2$ has been reported to be a useful reflection of cardiovascular performance in seriously ill patients with low cardiac output states.^{1,2} Thus, $\text{P}\bar{\text{v}}\text{O}_2$ was the best predictor of hyperlactatemia and of death in a variety of cardiopulmonary diseases previously reported by Kasnitz et al.¹⁵ A fall in $\text{S}\bar{\text{v}}\text{O}_2$ indicated inadequate tissue perfusion and was associated with severe clinical deterioration. The use of $\text{S}\bar{\text{v}}\text{O}_2$ determined by intermittent, as in this study, or by continuous measurements has been advocated to emphasize the onset and duration of the effect on cardiac output of inotropic and vasodilator therapy.^{6,7} The reason behind such an hypothesis is the well-known inverse relationship between CO and arteriovenous oxygen difference when $\dot{\text{V}}\text{O}_2$ and CaO_2 remain stable during therapy. In this setting, the reduction in $\text{a}\bar{\text{v}}\text{D}\text{O}_2$ and increase in $\text{S}\bar{\text{v}}\text{O}_2$ observed after therapy might be due to the increase in CO. Gore and Sloan⁶

reported a good correlation between $S\bar{v}O_2$ and CI following the administration of milrinone, a phosphodiesterase inhibitor III, in CHF patients. Chappel et al⁵ also reported a concomitant increase in $\dot{D}O_2$ and $S\bar{v}O_2$ without significant change in $\dot{V}O_2$ after the administration of a potent mixed vasodilator, nitroprusside, in refractory left ventricular failure. However, opposite results have been published previously by Hassan et al.¹⁶ Their results did not consistently demonstrate a strong correlation between CO and $S\bar{v}O_2$ in response to dobutamine used in the treatment of advanced CHF. In our study, despite the global and simultaneous increases in CI and $P\bar{v}O_2$ associated with a stable $\dot{V}O_2$, there was no correlation between these two parameters. However, such a correlation was sought in a separate analysis of each patient and showed two kinds of response to perindopril administration: group 1 with a stable $\dot{V}O_2$ and significant correlation between CO and $P\bar{v}O_2$ ($n=6$); group 2 with a significant increase in $\dot{V}O_2$ and lack of correlation between CO and $P\bar{v}O_2$.

Results from group 1 seem to indicate the lack of dependence between $\dot{D}O_2$ and $\dot{V}O_2$ in 60 percent of the CHF patients treated with perindopril. Similar results were previously reported by Chappell et al⁵ after nitroprusside administration and gave rise to the question of an alteration in oxygen delivery despite an increase in calculated $\dot{D}O_2$ after vasodilators by a drug-induced interference with peripheral vasoregulation. The consequence of such an alteration would be the possibility of shunting blood away from territories with high oxygen requirements to organs with low oxygen extraction, with the risk of a major imbalance between demand and true tissue delivery.¹⁷ This hypothesis can be ruled out in this study for the following reasons: (1) perindopril previously has been shown as being able to homogeneously affect all vascular beds and to particularly vasodilate renal, peripheral, and myocardial circulation;¹⁸ (2) despite the lack of plasma lactate measurement in these patients, the onset of acidosis was not observed (no significant change in pH during treatment), nor were there clinical signs of tissue hypoxia, thus confirming that each patient of this group had a significant clinical and hemodynamic improvement. Finally, the seven sample points per patient may rule out the possibility of an inadequate sample size in order to detect a correlation between $\dot{V}O_2$ and $\dot{D}O_2$.

Our results clearly indicated that in four out of our ten patients (group 2), there was a delivery-dependent oxygen consumption. This result was in marked contrast to the normal control of oxygen metabolism in which uptake is independent of CO and of the oxygen supply above some critically low limiting level.^{3,19,20} Under low level of $\dot{D}O_2$, being variable among the previously reported studies, a dependence between

$\dot{D}O_2$ and $\dot{V}O_2$ was obviously demonstrated.^{2,21,22} A critical level of $\dot{D}O_2$ of 9.8 ml/min/kg was reported in anemic or hypoxic animals studied by Cain et al,²¹ and 8.2 ml/min/kg in anesthetized patients documented prior to coronary artery bypass surgery by Shibutani et al.²² In patients in our study, mean $\dot{D}O_2$ was 510.9 ± 40.9 ml \cdot min⁻¹, ie, an average of 7.5 ml \cdot min⁻¹ kg with no significant difference between group 1 and 2 (Fig 3). Thus, a critical level of $\dot{D}O_2$ could not be predicted from the data and did not explain the discrepancy between the metabolic response of the two groups of CHF patients. Similar results to those observed in group 2 have been previously reported in critically ill patients suffering from adult respiratory distress syndrome who were receiving mechanical ventilation with positive end expiratory pressure with $\dot{D}O_2$ of about 20 ml \cdot min⁻¹kg⁻¹.^{4,23-26} Moshenifar et al¹⁷ reported a relationship between $\dot{V}O_2$ and $\dot{D}O_2$ in CHF patients and hypothesized the loss of capillary density adaptation in response to change in oxygen availability. The consequence of this functional (blood flow shunting) or anatomic (microembolization) loss of capillary bed adaptation was the lack of concomitant and inverse relationship between $\dot{D}O_2$ and ER_{O_2} . A similar hypothesis was proposed by Bihari et al¹⁹ in patients suffering from multiorgan failure. In about 50 percent, there was not a decrease in ER_{O_2} in response to $\dot{D}O_2$ increase with prostacyclin when there was a substantial oxygen debt before drug administration. This theory was previously described by Shoemaker²⁰ as an heterogeneous oxygen transfer state named maldistribution of flow at the microcirculatory level responsible for inadequate tissue oxygenation.

Our results did not clearly demonstrate the severity of patients in group 2, as previously reported by Hassan et al¹⁶ after dobutamine administration in patients with an advanced congestive heart failure. In this study, the dependence between $\dot{V}O_2$ and $\dot{D}O_2$ was observed in the more seriously ill patients with lower stroke volume and the higher pulmonary artery diastolic pressure. The relationship between the clinical and hemodynamic severity of the patients and the $\dot{V}O_2$ - $\dot{D}O_2$ relationship was also emphasized by Bihari et al¹⁹ in multiorgan failure patients since $\dot{V}O_2$ - $\dot{D}O_2$ dependent patients had a reduced survival. Found in patients of group 2 was a significant baseline reduction of the MAP and mean PCWP compared to group 1 and a slight, but nonsignificant reduction in systemic vascular resistance and noradrenaline plasma level. This lower systemic perfusion pressure in group 2 with a possible alteration of compensatory mechanism of vasomotor tone adaptation may modify the distribution of blood flow induced by perindopril within the microcirculation. Finally, this oxygen debt observed in the patients of group 2 was not due to an increase in oxygen demand secondary to hyperventilation

induced by perindopril, since pH and PaCO₂ remain within normal limits and ACE inhibitors have been previously reported to generally decrease ventilation during exercise.¹⁴

Whatever the mechanism of the lack of correlation between CO and P \bar{v} O₂ in CHF patients after administration of an ACE inhibitor, this study indicates that P \bar{v} O₂ or S \bar{v} O₂ cannot be used, as previously suggested,²⁷ instead of cardiac output to determine the hemodynamic consequence of inotropic drugs. It remains to be determined if the presence of a relationship between \dot{V} O₂ and \dot{D} O₂ during treatment with an ACE inhibitor is a potential predictive factor of reduction in survival.

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REFERENCES

- 1 Lee J, Wright F, Barber R, et al. Central venous oxygen saturation in shock. A study in man. *Anesthesiology* 1972; 36:472-78
- 2 Krauss XH, Verdow PD, Hughenoltz PG, et al. On line monitoring of mixed venous oxygen saturation after cardiothoracic surgery. *Thorax* 1975; 30:636-43
- 3 Schumaker PT, Cain SM. The concept of a critical oxygen delivery. *Intensive Care Med* 1987; 13:223-29
- 4 Danek SJ, Lynch JP, Weg JC, Dantzker DR. The dependence of oxygen uptake on oxygen delivery in the adult respiratory distress syndrome. *Am Rev Respir Dis* 1980; 122:387-95
- 5 Chappell TR, Rubin LJ, Markham RV, Firth BG. Independence of oxygen consumption and systemic oxygen transport in patients with either stable pulmonary hypertension or refractory left ventricular failure. *Am Rev Respir Dis* 1983; 128:30-33
- 6 Gore JM, Sloan K. Use of continuous monitoring of mixed venous oxygen saturation in the coronary care unit. *Chest* 1984; 86:757-61
- 7 Birman H, Haq A, Hew E, Aberman A. Continuous monitoring of mixed venous oxygen saturation in hemodynamically unstable patients. *Chest* 1984; 86:753-56
- 8 Brown MJ, Jenner DA. Novel double isotope technique for enzymatic assay of catecholamines, permitting high precision sensitivity and plasma sample activity. *Clin Sci* 1981; 61:591-98
- 9 Faxon DP, Creager MA, Halperin JL, Gavras H, Coffman JD, Ryan TJ. Central and peripheral hemodynamic effects of angiotensin inhibition in patients with refractory congestive heart failure. *Circulation* 1980; 61:925-30
- 10 Kugler J, Maskin C, Frishman WH, Sonnenblick EH, Lejemtel TH. Regional and systemic metabolic effects of angiotensin-converting enzyme inhibition during exercise in patients with severe heart failure. *Circulation* 1982; 66:1256-61
- 11 Richard CH, Ricome JL, Rimailho A, Conrad M, Auzépy PH. Effects of captopril on pulmonary haemodynamics. *Eur J Clin Pharmacol* 1984; 27:35-39
- 12 Pierpont G, Hale KA, Franciosa JA, Cohn JN. Effects of vasodilators on pulmonary haemodynamics and gas exchange in left ventricular failure. *Am Heart J* 1980; 99:209-16
- 13 Rubin SA, Brown HV, Swan HJC. Arterial oxygenation and arterial oxygen transport in chronic myocardial failure, at rest, during exercise and after hydralazine treatment. *Circulation* 1982; 66:143-48
- 14 Crowley AJ, Rowley JM, Stainer K, Hampton JR. Effects of captopril on abnormalities of the peripheral circulation and respiratory function in patients with severe heart failure. *Lancet* 1984; 2:1120-24
- 15 Kasnitz P, Druger GL, Yorra F, Simmons DH. Mixed venous oxygen tension and hyperlactatemia, survival in severe cardiopulmonary disease. *JAMA* 1976; 236:570-74
- 16 Hassan E, Roffman DS, Applefeld MM. The value of mixed venous oxygen saturation as a therapeutic indicator in the treatment of advanced congestive heart failure. *Am Heart J* 1987; 113:743-49
- 17 Mohsenifar Z, Amin D, Jasper AC, Shah K, Koener SK. Dependence of oxygen consumption on oxygen delivery in patients with chronic congestive heart failure. *Chest* 1987; 92:447-50
- 18 Thuillez C, Richard CH, Loueslati H, Perret L, Auzépy PH, Giudicelli JF. Regional and systemic hemodynamic effects of converting enzyme inhibition in patients with congestive heart failure. *Circulation* 1987; 76:444
- 19 Bihari D, Smithies M, Ginson A, Tinker J. The effects of vasodilatation with prostacyclin on oxygen delivery and uptake in critically ill patients. *N Engl J Med* 1987; 317:397-403
- 20 Shoemaker WC. Relation of oxygen transport patterns to the pathophysiology and therapy of shock states. *Intensive Care Med* 1987; 13:230-43
- 21 Cain SM. Oxygen delivery and uptake in dogs during anemic and hypoxic hypoxia. *J Appl Physiol* 1977; 42: 228-34
- 22 Shibutani K, Komatsu T, Kubal K, Sanchala V, Kumar V, Bizzarri D. Critical level of oxygen delivery in anesthetized man. *Crit Care Med* 1983; 2:640-43
- 23 Moshenifar Z, Goldbach P, Tashkin DP, Campisi DJ. Relationship between O₂ delivery and O₂ consumption in the adult respiratory distress syndrome. *Chest* 1983; 84:267-71
- 24 Kariman K, Burns SR. Regulation of tissue oxygen extraction is disturbed in adult respiratory distress syndrome. *Am Rev Respir Dis* 1985; 132:109-14
- 25 Kaufman BS, Rackow EC, Falk JL. The relationship between oxygen delivery and consumption during fluid resuscitation of hypovolemic and septic shock. *Chest* 1984; 85:336-40
- 26 Schoemaker WC, Appel PL, Kram HB. Hemodynamic and oxygen transport effects of dobutamine in critically ill general surgical patients. *Crit Care Med* 1986; 14:1032-37
- 27 Divertie MB, McMichan JC. Continuous monitoring of mixed venous oxygen saturation. *Chest* 1984; 85:423-28

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