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Increase in Cardiac Output and PEEP as Mechanism of Pulmonary Optimization*

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The influence of cardiac output (CO) and PEEP on pulmonary shunt (Qs/Qt) has been the subjects of considerable investigation but findings are controversial. The role of CO and PEEP on 19 isolated rabbit lung preparations perfused with hypoxic mixture (6 % CO_2, 10 % O_2, and 84 % N_2), which resulted in a constant oxygen venous pressure (64 ± 5.6 mmHg) has been studied. The first group of 11 preparations were used to study the influence of CO modifications with room air ventilation on the Qs/Qt when the CO rises in 48 %; in the second group simultaneous modifications in CO and PEEP (0.5 and 10 cm H_2O) were performed. A positive correlation ($p < 0.01$) in Qs/Qt (0.048 ± 0.04 to 0.12933 ± 0.09) was found when the CO increased in the first experimental group, the fluid filtration rate (FFR) also increased and the pulmonary vascular resistance (PVR) remained stable. In the second group an increase of 5 and 10 cm H_2O of PEEP at constant CO reduced the Qs/Qt (0.0361 ± 0.02 to 0.0184 ± 0.006) while it increased the arterio-venous oxygen difference, PVR and FFR. During high CO conditions increase of 5 and 10 cm H_2O of PEEP reduced the Qs/Qt (0.099 ± 0.03 to 0.027 ± 0.02) and FFR. These data suggest that when the Qs/Qt is increased, the use of PEEP can compensate the ventilation/perfusion alterations and restore pulmonary gas exchange.

Key words: Cardiac Output, Pv_{O_2}, PEEP, Shunt, Isolated lung, Hypoxia, A-V_{DO_2}.

Abbreviations: A-V_{DO_2}, Arteriovenous oxygen difference; Ca_{O_2}, Cv_{O_2}, Cc_{O_2}, Oxygen concentration in artery, vein and capillary; CO, Cardiac Output; FFR, Fluid Filtration Rate; FiO_2, Inspired oxygen fraction; HPV, Hypoxic pulmonary vasoconstriction; MLAP, Mean Left Atrial Pressure; MPAP, Mean Pulmonary Artery Pressure; PA_{O_2}, Alveolar oxygen partial pressure; Pa_{O_2}, Pv_{O_2}, Arterial and venous oxygen pressure; Paw, Airway Pressure; PB, Barometric pressure; PEEP, Positive end- expiratory pressure; Pa_{CO_2}, Pv_{CO_2}, Arterial and venous carbon dioxide partial pressure; PS, pulmonary shunt; PVR, Pulmonary vascular resistance; Qs/Qt, Shunted blood/total cardiac output; R, Respiratory quotient; RF, Respiratory frequency; S_{O_2}, Hb oxygen saturation.

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The Qs/Qt is a pulmonary blood flow which does not participate in the O_2 exchange. The physiological Qs/Qt is approximately 6-7 %, and arises from bronchial and Tebesian veins, which do not participate in pulmonary oxygenation and by a small percentage from the dependent regions of the lungs. Since KELMAN *et al.* (20), the Qs/Qt has been studied in different experimental situations using pulmonary models, *in vitro* as well as *in vivo*. Research was focused on the effect of the changes of Qt on Qs/Qt performed through different methods: pharmacologic (24), bypass (4), hemorrhage (27, 38) and mechanic (36). In most studies, mixed venous Pa_{O_2} was not a controlled variable which could have led to misinterpretations of the results. Consequently, several studies showed that the Qs/Qt fraction varied directly with changes of cardiac ıtput and indirectly with the Pv_{O_2} and ıe pulmonary vascular resistance. In dis-sed lungs, increases in cardiac output crease the shunt fraction, attributable to ıe perfusion of non ventilated areas. The effect of Pv_{O_2} on Qs/Qt is possibly due to effects on hypoxic vasoconstriction. We wanted to find out why changes in Qt could be an independent variable from the mixed venous Pa_{O_2}, mainly when the Qs/Qt increases by using a room air ventilated rabbit lung, with a total control of the modifications on cardiac output.

It was also considered important to study the effect of positive end PEEP in the shunt fraction after the Qt changes by using isolated rabbit lungs in West's zone II. In most patients PEEP increases oxygen saturation (23), functional residual capacity (13), and reduces or induces the resolution of atelectasias. It has been determined in our laboratory that the optimum PEEP effect is reached when interstitial pressure is positive, in agreement with other investigators (2, 5, 9, 19, 21, 29, 38); this condition could not be developed in our preparation not to interfere in the pulmonary gas exchange and hence in the shunt fraction.

Materials and Methods

Nineteen rabbits with body weight of 2.7 ± 0.3 kg (mean ± SD) were anesthetized with i.p. pentobarbital sodium 30 to 40 mg/kg. A tracheostomy was performed and the lungs ventilated mechanically at a constant tidal volume.

The peak inflation pressure was 15 cm H_2O. A median sternotomy was performed and 2 ml of heparin 1000 IU/ml were injected via cannula in the right ventricle. Two minutes later, the animal was exsanguinated through the same cannula. Approximately 125 ml of blood were obtained, and it was increased to 240 ml using 5 % Dextran solution and 0.9 % NaCl solution. The proportion of Dextran and NaCl solution was set to an oncotic pressure of 22 cm H_2O. The blood so obtained was used to prime the perfusion circuit. The heart and lungs were removed with a minimum handling of the lungs. A silastic perfusion cannula was inserted into the pulmonary artery via an incision in the right ventricle and a second cannula inserted in the left atrium via the left ventricle. These cannulas had both end and side holes and were fixed in place by ligature tied round both ventricles.

This ligature was used to suspend the preparation from a force transducer (Grass type FT-03C) thus permitting small changes in the preparation weight to be sensed (fig. 1). The zero reference for the vascular pressure was the left atrial level and the transducer was set at atrial level. All transducers were repeatedly calibrated by reference to a saline manometer. Since the lungs were suspended vertically, apices were approximately at atrial level and the diaphragmatic surface was about 8 cm below (West's zone II).

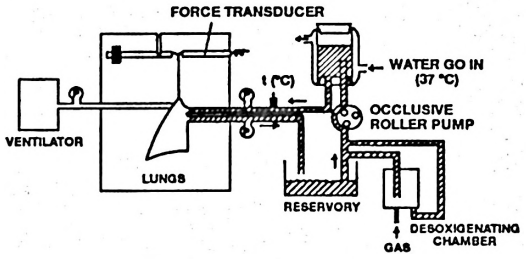

Fig. 1. *Perfusion circuit: Blood is pumped from the left atrial reservoir and desoxigenating chamber through the damping chamber and bubble trap to the pulmonary artery.*
The lungs are suspended from a force displacement transducer fixed to the top of a perpex / box. p = pressure transducer.

Perfusion was commenced within 10 min of exsanguination. The lungs were perfused at constant flow by means of an occlusive roller pump (Watson-Marlow) at a MPAP of 12.85 ± 3.8 mm Hg which resulted in flows of 52 ± 3.1 ml/min. Flow was maintained constantly throughout each experiment. The oscilations produced by the pump were minimized by passing the outflow through an air-filled damping chamber, surrounded by a circular water jacket maintained at 37 °C using a plastic radiator (34).

Once the blood passed from the lungs to the left atrial, it was conduced to a desoxygenating chamber wich had a teflon net containing a gas mix formed by 6 % CO_2, 10 % O_2 and 84 % N_2, with 5 L/min gas flow.

The lungs were room air ventilated and the arterial and venous Pa_{O_2}, Pv_{O_2} and pH were measured in a IL Micro 13. The pH of the perfusate was maintained at normal limits by the addition of small alliquots of sodium bicarbonate. The dose of bicarbonate was calculated according to the arterial pH and Pv_{O_2}.

The Hb and the hematocrit were measured in all experiments at the beginning and at the end using a Hemoglobiter FC-DPR 30.

The blood flow was measured and the PVR calculated at each phase. The perfusion was usually started within 8 to 10 min at the termination of the bleeding. The blood reservoir was set at the same height as the left atrium resulting in a MLAP of –0.1 ± 3.8 mm Hg. After a stabilization period of about 30 min. the gain of the force transducer amplifier was adjusted so that 2 cm deflection was produced when a weight of 1 g was added to the hook from which the preparation was hanging.

FFR was measured using the isogravimetric method described by LUNDE and WAALER (26). With this method, it is possible to separate the pulmonary blood volume change from FFR change using the patron on lung weight traces. In the first, there was marked rapid weight change after a blood volume change. The second shows a more moderate weight change which stabilizes or maintains a constant slope and this one corresponding to FFR.

All the experimental techniques and animal managements were previously approved by the National Institute of Scientific and Technological Research (CDCH National statutes, 1989) and by the Institutional Review Board for the care of animal subjects of the "Instituto de Medicina Experimental" of the Central University of Caracas (Venezuela).

Shunt equation.– To calculate the pulmonary shunt the standard Berggren equation was used (3):

$$Qs/Qt = Cc_{O_2} - Ca_{O_2}/Cc_{O_2} - Cv_{O_2}$$

The Ca_{O_2}, Cv_{O_2} and capillary Cc_{O_2} was obtained through the following gas concentration equation:

$$C\,O_2 = S_{O_2} \times Hb \times 1.34 + (Pa_{O_2} \times 0.0031)$$

Where $C\,O_2$ is the total blood oxygen content (oxy-hemoglobin and oxygen dissolved in plasma), Hb is the hemoglobin

concentration, S_{O_2} is the Hb oxygen saturation, 1.34 is the solubility coefficient, and P_{O_2} is the partial pressure of oxygen. S_{O_2} was determined from the measurement of Pa_{O_2} and the standard oxygen dissociation curve (pH/Pv_{O_2}/temp). To calculate Cc_{O_2}, alveolar and capillary Pa_{O_2} were considered to be equal, since it is not possible to measure Pa_{O_2} through the alveolar membrane. As the interstitial pressure was maintained at low level and no increase in FFR was obtained, the oxygen diffusion would not be affected (7, 8).

$$PA_{O_2} = Fi_{O_2} (PB - 47) - PaC_{O_2} [Fi_{O_2} + (1 - Fi_{O_2})/R].$$

Where PA_{O_2} is the mean alveolar oxygen partial pressure, Fi_{O_2} is the inspired oxygen fraction, PB is the barometric pressure, PvC_{O_2} is the arterial carbon dioxide partial pressure, and R is the respiratory quotient (0.85).

Data analysis.– Statistical differences between experimental values were determined by analysis of variance followed by Student's *t* test. Pearson's coefficient of correlation was also applied. Significance was accepted for $p <$ than 0.05.

Experimental protocol.– Group I (table I): Eleven isolated lung preparations were used to study the influence of cardiac output on pulmonary shunt. Basal conditions of the lungs in the protocol were: pH 7.38 ± 0.06, room air ventilation (15 ml/kg), RF 24 ± 3 breath/min, MPAP 15 ± 2 cm H_2O, Pv_{O_2} 64 ± 5 mm Hg and blood flow 52 ± 3 ml/min.

These preparations were subject to the following blood flow changes (in ml/min): 1. Basal, 52 ; 2. Increased, 73 ; 3. Basal, 53 ; 4. Decreased, 32 ; and, 5. Basal, 52. The former Qt changes were performed interchanging 2 and 4 among the isolated rabbit lungs in order to avoid the effect of time running on the preparation from affecting the results.

Group II (table II): Eight isolated rabbit lungs were used to study the influence of both cardiac output and PEEP on pulmonary shunt. Basal conditions of the lungs were the same as group I. The PEEP was modified by placing the respiratory outflow tube under 5 and 10 cm H_2O.

The Qt and PEEP modifications were maintained for 10 minutes in each of the intervals. In each case, the measurement was done 4 minutes after the modifications were performed in order to allow vascular and perivascular pressure to stabilize (16, 26, 34). A complete set of measurements was recorded after each change in Qt and in the PEEP level, including the Pa_{O_2}, the Pv_{O_2}, and the pH (arterial and venous) as well as the MPAP, MLAP, Paw, FFR, Hb and the pulmonary blood flow.

Results

Effect of changes in cardiac output on pulmonary shunt and hemodynamics variables (Group I).– The effect of changes in CO on PS, FFR and hemodynamic variables is shown in table I. With an increase of the CO in 48 % (52 ± 3.5 – 73 ± 4.1 ml/min) a positive correlation ($p < 0.01$) with the PS (0.06 ± 0.04 – 0.13 ± 0.09) was found, which, suggested that the increase in CO produced an increase in PS. Furthermore a significant decrease in AV difference (50.8 ± 8.2 – 43.2 ± 7.7), with a constant Pv_{O_2} (62.74 ± 5.9 – 63.9 ± 4.3 mm Hg) was observed ($p < 0.01$). The FFR increased (0.06 ± 0.05 – 0.17 ± 0.09 g/min) and the PVR remained stable (0.32 ± 0.07 – 0.29 ± 0.03 mm Hg • min • ml^{-1}).

Contrarywise, a 40 % decrease in CO (53 ± 3.26 – 32 ± 2.3 ml/min) induced a small but not significant increase in Qs/Qt (0.048 ± 0.04 – 0.061 ± 0.07), asso-

Table I. *Variations in hemodynamic and blood gas parameters during the stepwise change in cardiac output.* Cardiovascular measurements: MPAP (Mean Pulmonary Artery Pressure), PVR (Pulmonary Vascular Resistance). FFR (Fluid Filtration Rate), Blood gas tension and pH at each stage of the protocol in normal lungs. Number under each measurement denotes statistically significant differences to the treatment, denoted by numbers *P < 0.05, **P < 0.01 and ***P < 0.001, values are means ± SD.

Cardiac output	1. Normal (52 ml/min)	2. High (73 ml/min)	3. Normal (53 ml/min)	4. Low (32 ml/min)	5. Normal (52 ml/min)
FFR (g/min)	0.06 ± 0.05	0.17 ± 0.09 **1, 3, 4, 5	0.11 ± 0.06	0.08 ± 0.01 **1, 3, 4, 5	0.10 ± 0.06
MPAP (mmHg)	12.90 ± 3.2	18.36 ± 2.41 **1, 3, 4, 5	12.75 ± 3.94	8.19 ± 2.71 **1, 3, 5 ***2	13.12 ± 3.3
PA_{O_2} (mmHg)	121.2 ± 10.6	111.1 ± 13.8 *4 **1, 3, 5	117.4 ± 10.4	114.7 ± 13	120 ± 9.8
$A\text{-}V_{DO_2}$	50.77 ± 8.16	43.16 ± 7.71 *4 **1, 3, 5	51.2 ± 8.33	48.61 ± 6.21	51.8 ± 7.4
Qs/Qt	0.05 ± 0.04	0.13 ± 0.09 *1, 4 **3, 5	0.05 ± 0.04	0.06 ± 0.07	0.04 ± 0.03
PVR (mmHg min ml^{-1})	0.32 ± 0.07	0.29 ± 0.03 *1 **4	0.03 ± 0.03	0.36 ± 0.08 **1, 2, 3, 5	0.31 ± 0.05

ciated with a decrease in FFR (0.11 ± 0.06 – 0.08 ± 0.07 g/min), and a significant rise in the PVR (0.3 ± 0.03 – 0.36 ± 0.08 mm Hg • min • ml^{-1}).

Effect of the change on PEEP on pulmonary shunt (Group II).– The effect of various PEEP levels and CO on PS, FFR and hemodynamic variables is shown in table II. The increase in PEEP to 5 and 10 cm H_2O in basal CO condition reduced the Qs/Qt (0.04 ± 0.02 – 0.02 ± 0.01) and increased the A-V difference (48.3 ± 6.2 – 54.6 ± 7.1), the PVR (0.3 ± 0.04 – 0.04 ± 0.08 mmHg • min • ml^{-1}) and in the FFR (0.07 ± 0.06 – 0.09 ± 0.02 g/min). Furthermore, a rapid positive weight gain is observed immediately following each increase in PEEP, possibly produced by an increase in intravascular volume. When a higher CO was produced the change in PEEP from 0 to 10 cm H_2O increased the A-V difference (39.7 ± 8.2 – 54.8 ± 6.7), reduced the Qs/Qt (0.099 ± 0.03 – 0.027 ± 0.02) and FFR (0.2 ± 0.11 – 0.04 ± 0.03 g/min), which was observed after each increase in PEEP.

Discussion

The isolated lung preparation permits a very controlled environment to study isolated physiological questions relevant to lung pathophysiology; in this study we control the pulmonary blood flow, MPAP, PLAP, Paw, Vt, Pv_{O_2}, PvC_{O_2} and FFR, all of which affect the pulmonary shunt. However, the preparation has its limitations since it differs from normal lungs in several important aspects. First, the lymphatics are cut and little fluid

Table II. *Variations in hemodynamic and blood gas parameters during the stepwise change in cardiac output and PEEP.*
Cardiovascular measurements: MPAP (Mean Pulmonary Artery Pressure), CO (Cardiac Output), FFR (Fluid Filtration Rate). Blood Gas Tension at each stage of the protocol with PEEP and C O modifications. Number under each measurement denotes statistically significant differences to the treatment, denoted by numbers. *P < 0.05, **P < 0.01 and ***P < 0.001, values are means ± SD.

	NORMAL			HIGH			NORMAL		
	1	2	3	4	5	6	7	8	9
PEEP (cm H_2O)	0	5	10	0	5	10	0	5	10
CO (ml/min)	52.8±2.1	52.3±3.4	51.4±3.1	69.8±4.2	71.3±4.0	70.8±3.6	52.1±3.1	51.7±3.2	52.6±3.9
Qs/Qt	0.036±0.02	0.054±0.04	0.018±0.06	0.098±0.03 *2, 5, 8 **1, 7 ***3, 6	0.036±0.03	0.026±0.02 *3, 1, 5, 7 **2, 8 ***4	0.035±0.02	0.039±0.02	0.022±0.015
Pa_{O_2} (mmHg)	119.8±11.3	108.10±10	120.9±9.3	107.3±7.4 *5, 8 **1, 6 ***3, 9	114.3±7.6	118.7±7.31 *8 **2, 4, 7	109.78±9.6	113.6±6.9	118.8±4.08
FFR (g/min)	0.07±0.06	0.08±0.06	0.09±0.02	0.20±0.11 *1, 2, 7 **6, 87	0.11±0.04	0.04±0.08	0.05±0.06	0.046±0.02	0.09±0.05
MPAP (mmHg)	12.83±4.3	15.41±4.17	17.83±3.6	19.5±4.09 *2, 6, 7 **1	23.6±4.7	24.6±3.8 *4 **3, 8, 9 ***1, 2, 7	14.5±3.24	16.6±4.38	19.6±3.98

appears to drain from the lung during the perfusion. Secondly, in the normal chest, lung expansion is limited by the chest wall. Thirdly, since there was no negative intrathoracic expiratory pressure, interstitial pressure fluctuations were avoided. Fourthly, the lungs were perfused with a non pulsatile flow of oxygenated blood, but there is no bronquial circulation. All these factors may affect the FFR, thus the rate of edema formation in isolated lungs may be somewhat different from the rate in an intact lung at the same PEEP level. Our previous experience is that the experimental preparation is stable for up to six hours. Electron microscopic examination shows that there is no discernible ultrastructural alterations of the alveolar capillary membrane after four hours of perfusion (16).

The main advantage of this preparation is that it permits extremely accurate measurements of weight gain in the lungs and also it permits to discern between the changes in weight produced by the increase in insterstitial volume or by pulmonary vascular volume. A rapid change in lung weight, usually in less than 1 minute following sudden change in blood flow, PAW or MLAP, was considered to be produced by changes of intravascular volume (5, 22, 25, 36, 40). This weight change was completely reversible when pressures, flow or PEEPwere returned to preexisting values. Following the abrupt change, a slow change in weight was observed occurring at relatively constant rate, while the above mentioned variables were maintained constant, thus pulmonary edema could be easily measured (5, 22).

In addition, this preparation permits adequate control of mixed venous O_2 pressure through the use of gas mixtures in the deoxygenator, as well as precise oxygen content measurements in the blood draining into the pulmonary artery. Some authors (24, 32, 38) postulated that the increase from 80-100 % in Pv_{O_2} at constant Qt could be the determining factor of a pulmonary shunt increase. On the other hand, BISHOP *et al.* (4, 26) using room air ventilated protocols, reported that the Qs/Qt increase could be produced by either the increase in Qt or the Pv_{O_2} changes.

It is important to consider that any increase in the Pv_{O_2} to more than 60-65 mmHg, produces a relaxation of the HPV (32). This may cause a blood flow redistribution to the poorly ventilated areas and to those areas with diffusion alterations. These two effects explain the Qs/Qt increase, caused by the increase in Pv_{O_2}.

A pulmonary blood flow increase by 48 % caused subsequent rise of Qs/Qt in lung in West's zone II at constant PVR. This finding can be explained by the effects of recruitment of alveolar vessels in regions with poor ventilation, which results in a Pa_{O_2} fall due to an increase in the shunt fraction. A rise in anatomic and precapillary arteriovenous shunts (6, 43), as well as the opening of extraalveolar vessels (18) also determine an increase of Qs/Qt when pulmonary blood flow changes; diffusion alterations due to hydrostatic edema contribute to these results. In constrast, a decrease in CO by 40 % is associated with a small but not significant increase in PS and a significant rise in PVR, which may be explained by a derecruitment of pulmonary vessels in regions with good ventilation.

PEEP has been used during mechanical ventilation to gas exchange improvement in acute hypoxemic respiratory failure (14, 16, 21, 23, 30). By maintaining the airway pressure above the atmospheric level during expiration, PEEP increases functional residual capacity. We believe that PEEP increases rather than decreases lung water in isolated experimental lung, similar findings having been reported by other

investigators (5, 9, 10, 29, 31, 44). There is sufficient evidence to support the concept that interstitial pulmonary water is derived from both intra and extra-alveolar vessels, the latter ones contributing to 63 % of the edema formation under static conditions (1/3 from arterioles and 2/3 from venules). The fluid flux from these vessels varies with the degree of MPAP, pleural pressure (28) and alveolar inflation pressure (3, 9, 11, 34, 42, 45).

Since in our protocol the pleural pressure is constant, FFR change is determined by the change in MPAP (when the blood flow increases) or the change in alveolar pressure (when using PEEP). For that reason we decided to use PEEP to improve pulmonary ventilation. With constant pleural pressure and blood flow increasing PEEP to 5 and 10 cm H_2O reduced PS and increased PVR. These changes can be explained by the rise in FRC. When we apply the same PEEP levels during high blood flow conditions the PS decreased significantly and progressively. These results suggest that when PS increases, induced by rises in pulmonary blood flow, in a lung West's zone II, the use of PEEP, which improves the FRC, can compensate the V/Q alterations. Thus, PEEP at different levels can increase the pulmonary efficiency in high CO conditions.

C. CURIEL, R. MARTÍNEZ, V. PINTO, A. ROSALES, G. D'EMPAIRE y R. SÁNCHEZ DE LEÓN. *Aumento del gasto cardíaco y PEEP como mecanismo de optimización pulmonar.* Rev. esp. Fisiol. (J. Physiol. Biochem.), 51 (1), 7-16. 1995.

La influencia del gasto cardíaco (CO) y PEEP sobre el shunt pulmonar (Qs/Qt) es objeto de numerosas investigaciones siempre, con resultados controvertidos. Se estudia el papel del CO y del PEEP en 19 preparaciones aisladas de pulmón de conejo perfundidas con mezcla hipóxica (CO_2 6 %, O_2 10 % y N_2 84 %) que da lugar a una presión de oxígeno venosa constante (64 + 5,6 mmHg). En un primer grupo de 11 preparaciones se estudia la influencia de las modificaciones de CO con ventilación de aire ambiental sobre Qs/Qt cuando el CO sube a 48 %; en un segundo grupo se realizan modificaciones simultáneas en CO y PEEP (0,5 y 10 cm H_2O). Se encuentra una correlación positiva ($p < 0,01$) en Qs/Qt (0,048 + 0,04 to 0,12933 + 0,09) cuando se incrementa el CO en el primer grupo experimental, aumenta también la tasa de filtración de líquido (FFR) y permanece estable la resistencia vascular pulmonar (PVR). En el segundo grupo el incremento de 5 y 10 cm H_2O de PEEP y CO constante reduce el Qs/Qt (0,0361 + 0,02 a 0,0184 + 0,006) mientras que incrementa la diferencia arteriovenosa de oxígeno, PVR y FFR. Durante las condiciones de alto CO, el aumento de 5 y 10 cm H_2O de PEEP reduce el Qs/Qt (0,099 + 0,03 a 0,027 + 0,02) y el FFR. Estos datos sugieren que cuando se incrementa la Qs/Qt el uso de PEEP puede compensar las alteraciones de la ventilación/perfusión y restaurar el intercambio de gas pulmonar.

Palabras clave: Gasto cardíaco, Pv_{O_2}, PEEP, Shunt, Pulmón aislado, Hipoxia, A-V_{DO_2}.

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