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Total Inversion of the Left Lung Circulation: Morphologic and Functional Analyses

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ABSTRACT

Background: An experimental model for total inversion of left lung circulation was developed. With this model, the authors demonstrate that it is possible to reverse the pulmonary circulation and preserve the normal function and morphology of the lung.

Methods: Eight dogs had their left pulmonary circulation reversed. The blood from the pulmonary artery trunk was diverted to the pulmonary veins, and returned from the pulmonary artery into the left atrium. In order to monitor the flow through the reversed system, color Doppler echocardiography was performed on the ninth postoperative day. The dogs were reoperated after 15 days for re-evaluation. Blood gas analyses from the aorta and the pulmonary artery were used to study the functional status of the lung in both operations. The morphology was studied by comparing biopsies of the lung performed before and after reversal of flow.

Results: Blood gas analysis showed no significant difference between the samples from the aorta and pulmonary artery. Color Doppler echocardiography was a reliable method for the study of the inverted circulation. The histological study showed no differences in the morphology of the lung after the reversed circulation.

Conclusions: Pulmonary function and morphology remained normal after left pulmonary circulation was fully reversed.

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INTRODUCTION

The complex pulmonary circulatory system is made up of two independent arterial circulations—the pulmonary circulation and the bronchial circulation. The pulmonary arteries take deoxygenated blood from the right ventricle into the pulmonary capillaries for gas exchange. The normal pulmonary circulation has a high compliance, low resistance and is a low-pressure system. The branches of the pulmonary arteries follow the same pattern of division as the bronchi. Small pulmonary arteries adjacent to the alveoli respond to alveolar gas composition. Alveolar hypoxia causes pulmonary artery hypoxia and vasoconstriction in contrast to the systemic circulation where hypoxia produces vasodilation [Guyton 1989, Zin 1995]. The bronchial artery circulation originates from the aorta and is responsible for only about 1% of total pulmonary blood flow.

Although the pulmonary artery circulation does not have atherosclerotic impairment in most cases, there are several other lesions that may make blood flow difficult in these vessels. These injuries may be classified as primary and secondary. Primary injuries result from intrinsic changes in the pulmonary circulatory tree, usually at the level of the capillaries. Secondary disorders are more frequently related to cardiac disease or embolic disorders, which in general causes secondary pulmonary hypertension [Andrade 1995]. Other lesions of the pulmonary vascular system are less frequently encountered and have not been fully studied. The clinical management with various drugs, either by inhalation or intravenous, represents a great challenge and has been studied extensively [Alpert 1994].

There are two different groups of pulmonary circulatory disorders: those reversible by known procedures, as in congenital diseases, and those that are currently considered irreversible, such as primary pulmonary hypertension and pulmonary hyper-resistance.

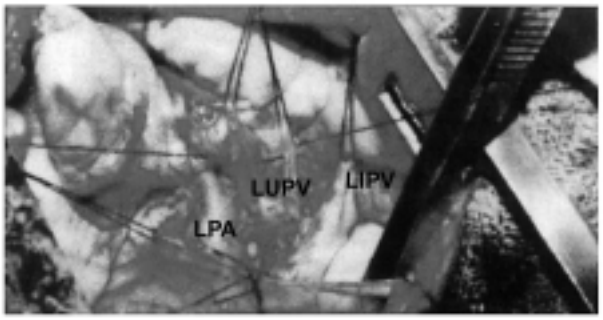

Figure 1. LIPV = left inferior pulmonary vein; LUPV = left upper pulmonary vein; LPA= left pulmonary artery

The cause of primary pulmonary hypertension (PPH) is unknown and is observed mostly in young women. It is characterized by a continuous progression of right heart failure with a mean survival time of three years following diagnosis. There are several medical treatment approaches with unstable and sometimes unsatisfactory results. When clinical therapy does not work, heart-lung transplantation is an option. Lung transplant still has high failure rates, in contrast to transplant procedures of other organs, such as kidneys, heart and liver [Palevsky 1991, Rich 1995].

Several animal models have been created to study the response of the pulmonary circulation to various stimuli. In most cases, they are animal models designed to develop changes in the pulmonary vasculature leading to pulmonary hypertension, so that the physiologic study might evaluate the efficacy of various therapy options [Prielipp 1991]. With the increasing interest in the development of pulmonary circulation research, an experimental model of left pulmonary circulation reversal is suggested.

A few areas in which experimental research with blood flow inversion is used clinically are: reconstructive plastic surgery using arterialized venous patches [Inoue 1990]; peripheral vascular surgery with revascularization of the plantar venous arch [Lengua 1995]; and cardiovascular surgery for myocardial protection with retrograde cardioplegic solution [Menasché 1990, Salermo 1991], and for protection of the central nervous system with retrograde cerebral perfusion [Kouchoukos 1994]. In the experimental field, there are other areas where blood flow inversion has been studied: in coronary venous system revascularization; the use of the pulmonary venous system in pulmonary preservation for transplants; and in the treatment of pulmonary emboli reaching the more distal arterial segments [John 1995, Varela 1997].

The present study is aimed at developing a canine experimental model for the physiologic study of reversed pulmonary circulation. This model will be useful in the evaluation of the effectiveness of reversed circulation in gas exchange. Finally, this study evaluates possible morphologic changes in the lung resulting from reversed circulation.

MATERIALS AND METHODS

In order to develop this model a complete pulmonary circulation reversal had to be achieved. In the normal physiology, blood flows from the right ventricle through the pulmonary trunk to the lungs via the main pulmonary arteries. Upon arrival to the lung, blood is oxygenated and then returns to the left atrium via the pulmonary veins. Therefore, for a complete inversion of this flow, all of the blood coming from the left pulmonary artery has to be directed to the pulmonary veins and returned via the left pulmonary artery connected to the left atrium. For the blood from the left pulmonary artery to get to the pulmonary veins a small segment of tubular graft was placed, and the return was enabled by connecting the pulmonary artery directly to the left atrium.

Eight mixed-breed adult dogs weighing from 13 to 23 kg obtained from the kennel of Curitiba's City Hall were used. The animals were numbered according to the chronological order of surgery dates. They were sent to the Experimental Surgery Laboratory on the morning of the surgery day, where a trichotomy of the left thoracic region and both front legs was carried out. After weighing, the animals were anesthetized and ventilated with a volume ventilator, with an FiO_2 of 100%. A cardioscope and an invasive pressure monitor were used for hemodynamic monitoring.

A left postero-lateral thoracotomy was used for surgical access. The left lung was pushed to the side and the left pulmonary artery and pulmonary veins were identified. All of the vessels were dissected, released from the neighboring tissues and encircled with a cotton thread, size 0 (see Figure 1 ◉). Subsequently, heparin sodium was administered intravenously at 1 mg/kg of body weight.

After the pulmonary trunk was isolated, the left pulmonary artery was clamped between two straight vascular clamps and cut at its origin. The left atrium and origins of the pulmonary veins were isolated and clamped with a Satinsky vascular clamp. The left atrium was then sectioned leaving a small patch of left atrial wall as a cuff along with the openings of the pulmonary veins to facilitate anastomosis. Atrial closure was then carried out using a two-layer suture with 5-0 polypropylene suture (Prolene®).

Vascular anastomoses were carried out with a continuous 6-0 polypropylene suture (Prolene®). The same procedure was used in all of the dogs. First, to establish pulmonary blood flow back to the heart the hilar portion of the divided left main pulmonary artery was anastomosed to the left auricle in an end-to-end manner. Next, the hilar segment of the pulmonary veins (two or three) were anastomosed to one end of an expanded 8 mm polytetrafluorethylene (PTFE) prosthesis. The inferior pulmonary vein was anastomosed in an end-to-end fashion with the prosthesis, and the remaining veins attached in an end-to-side fashion. The PTFE prosthesis was finally anastomosed end-to-end to the proximal trunk of the left main pulmonary artery, thus reestablishing the pulmonary flow in the reversed direction (see Figures 2, 3, and 4 ◉).

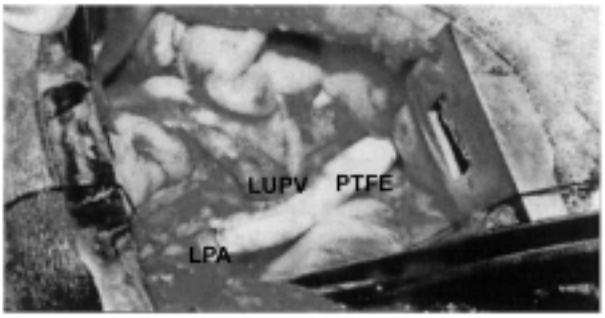

Figure 2. PTFE = PTFE graft; LUPV = left upper pulmonary vein; LPA = left pulmonary artery

After all anastomoses were finished, heparin was reversed with 5 ml of protamine intravenous for each 1 ml of heparin. Careful control of hemostasis was performed. The lung was then manually inflated to remove atelectasis.. After normal ventilation was restored, arterial blood gas samples were collected from the left pulmonary artery about two centimeters away from the anastomosis with the left auricle and from the aorta. These samples were immediately sent for biochemical evaluation.

The dogs were sent to a veterinary clinic for postoperative care (analgesia and treatment of the surgical wound). Analgesia was maintained with antiinflammatory drugs such as diclofenac sodium (Voltaren®) or tenoxicam (Tilatil®) intramuscular as required. The dogs were examined daily and the status of the surgical wounds and respiratory patterns were recorded. They were followed-up for 14 days.

On the ninth postoperative day, color Doppler echocardiography was carried out. The animals were sedated with chloral hydrate at 400 mg/kg of body weight to facilitate the procedure. An SD-800 Phillips ultrasound machine with a 3.5 MHz transducer was used. The status of the PTFE prosthesis, the left pulmonary artery, and the flow direction were identified in order to record the presence of flow in the anastomosed vascular structures.

On the fourteenth postoperative day, the animals were returned to surgery, anesthetized with thiobarbiturate ethyl sodium (Thionembutal®) 15 mg/kg, and instrumented for hemodynamic monitoring as in the first surgery. A new left postero-lateral thoracotomy was performed. The adhesions from the previous surgery were carefully released and the vascular structures of the left pulmonary hilum were identified. As in the first surgery, a biopsy of the left lung was carried out. The macroscopic aspects were studied and recorded and new blood gas samples were collected. An animal was then sacrificed with 20 ml of 19.1% potassium chloride.

For histological evaluation, two pulmonary samples were collected from each dog, one as a baseline sample from the first operation prior to flow inversion and another after the "redo" operation two weeks later. Both sets of specimens were preserved in 10% formaldehyde solution. The pieces were identified and analyzed by a pathologist following the standards for pulmonary optical microscopy.

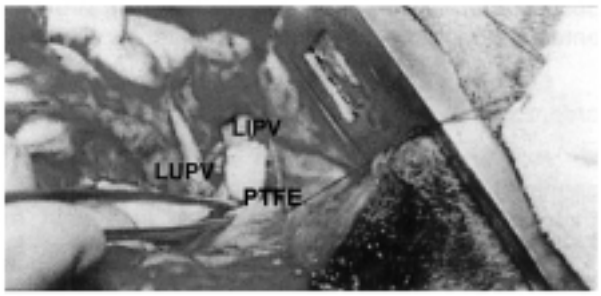

Figure 3. PTFE = PTFE graft

Three staining methods were used: hematoxylin-eosin, trichrome, and silver for reticular fibers.

RESULTS

All eight dogs underwent the described procedure without difficulties in the surgical handling. None had evidence of disease (such as active pneumonia), and all were in good clinical condition. All of them survived the procedure without major complications. No difficulties were encountered with orotracheal intubation, muscular relaxation, or any other aspect of the anesthetic technique used.

Mean blood pressure during the procedure ranged from 40 to 130 mmHg and heart rate from 65 to 160 beats per minute. The volume of crystalloid solution infused for hydration and volume restoration ranged from 12 ml/kg/hr to 45 ml/kg/hr with a mean of 32 ml/kg/hr.

The return to spontaneous ventilation after the procedure was adequate, and the dogs were extubated within an average of 30 minutes (10-55 minutes) postoperatively. The mean operation time was 2 hours and 15 minutes, ranging from 2 hours to 3 hours and fifteen minutes. No complications resulting from the surgical procedure were observed.

All eight dogs survived the two week postoperative follow-up period One dog (Dog #5) experienced dyspnea after exercise and cough with hemoptysis after the first postoperative day. No changes of the respiratory pattern were observed in the other animals.

Echocardiographic evaluation was carried out in the eight dogs. Sedation with chloral hydrate was satisfactory, and the animals were cooperative during examination. Of the eight echocardiographic examinations, one did not show flow in

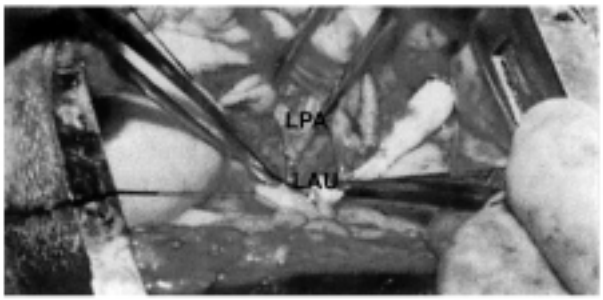

Figure 4. LAU = left auricle; LPA = left pulmonary artery

Table 1. Blood Gas Analysis from Inverted Pulmonary Circulation

	pH	pO_2	pCO_2	HCO_3	% Sat
Mean					
Ap	7.46	331.63	30.24	20.46	99.56
Ao	7.47	331.25	29.81	20.74	99.73
ao2	7.49	297.29	28.33	29/0.89	99.71
aao2	7.49	283.29	28.64	20.14	99.73
Standard deviation					
Ap	0.09	72.24	10.36	5.19	0.41
Ao	0.10	61.15	8.96	5.49	0.20
ao2	0.09	85.62	7.42	3.08	0.22
aao2	0.12	55.24	9.23	3.49	0.18

Ap: gasometry of the pulmonary artery at operation
Ao: gasometry of the aorta at operation
ao2: gasometry of the pulmonary artery at reoperation
aao2: gasometry of the aorta at reoperation

the PTFE graft. In the seven remaining dogs, circulation was observed in the graft and reversed flow was observed in the left pulmonary artery. The findings were compared to the invasive analysis showing a specificity of 100%.

The macroscopic observations at reoperation showed atelectasis of the left lung in only one animal (#5). The lungs of the remaining animals were fully expanded. Tissue perfusion was considered adequate in seven animals. Only one animal (#5) had signs of hypoperfusion in the pulmonary tissues. No differences were observed in left and right lung compliance when the grafts were patent. The dog with the occluded graft had a dense and atelectatic lung.

Mean blood gas values results showed no significant difference between left pulmonary artery values and aorta values (see Table 1 ◉).

The biopsies were evaluated by pathologists using three staining methods. No histologic differences between tissues of the preoperative and postoperative periods of reversed pulmonary circulation were observed.

DISCUSSION

The study of reversed systemic venous circulation has been of great interest to the medical community. This is reflected in the large number of publications describing the search for surgical alternatives for such complex issues. The proposed procedures have not been well established, and classic therapy has not achieved satisfactory results [Inoue 1990, Menasché 1990, Salerno 1991, Kouchoukos 1994, John 1995, Lengua 1995, Varela 1997].

Dogs were chosen for the proposed experimental model, since the anatomy of their circulatory and pulmonary system is similar to human anatomy. Blood pressure and heart rate control were considered quite satisfactory with the use of inhaled anesthesia. A consistent nebulized volume-hemodynamic change ratio was obtained in all animals. With this kind of monitoring the amount of anesthetic drug was fully used , and further confirmed by the animal's recovery into spontaneous respiration after the surgical procedure. The combination of several drugs allowed a balanced anesthesia, and the amount used did not result in undesirable effects.

No difficulties were encountered in the handling of the anastomoses or in the control of eventual hemorrhage. This supports the technical feasibility of working in this area of the circulatory system. We also noted that the venous structures were converted into a system of greater pressure, which could have led to an even greater risk of bleeding. However, this was not observed.

The anastomotic disposition found after restructuring the pulmonary hilar vessels to the PTFE graft was adequate. It did not result in complications such as obstruction of flow to the lungs or of blood return to the heart once the vessels were perfused and postoperative pressure was normal. The macroscopic evaluation of the reversed pulmonary circulation was always very similar to the contralateral lung. The patency rate of the PTFE graft was considered good, especially since there is a generally high occlusion rate for synthetic grafts in low pressure systems. In the pulmonary reversal operation, however, flow rates through the graft are quite high.

The major limiting factor for an adequate gas exchange in the lung with reversed circulation would be the interstitial edema. However, early postoperative blood gas analysis and those done at the time of reoperation showed perfect gas exchange in the operated lung. The histologic study also confirms the lack of morphologic change in the lung parenchyma.

In conclusion, a model of fully reversed pulmonary circulation was therefore achieved and no undesirable results were observed. Gas exchange remained the same as with the normal circulation. Echocardiographic studies showed a perfect correlation with the direct examination of the conduits and confirmed the presence of flow through the graft. Histologic data with pre-inversion samples and samples obtained 14 days after the surgery show that the lungs maintained their normal characteristics, and no alterations of the vascular tree or edema were observed.

This experimental model may be used for the research of pulmonary circulation inversion, providing a new research field for respiratory physiology and stimulating future interest in the surgical treatment of pulmonary circulation disorders.

REFERENCES

1. Alpert MA, Concannon MD, Mukerji B, Mukerji V. Pharmacotherapy of chronic pulmonary arterial hypertension: value and limitations. Part II: secondary pulmonary hypertension. Angiology 45: 775-60, 1994.
2. Andrade GN, Costa AF. Hipertensão pulmonar e cor pulmonale. In: Bethlem N. Pneumologia. São Paulo: Atheneu, 817-29, 1995.
3. Guyton AC. Textbook of medical physiology. 6th ed. Philadelphia: Saunders, 1989.
4. Inoue G, Maeda N, Suzuki K. Resurfacting of skin defects of the hand using the arterialised venous flap. Brit Plastic Surg 43: 135-9, 1990.

5. John LC, Awad WI, Anderson DR. Retrograde pulmonary embolectomy by flushing of the pulmonary veins. Ann Thorac Surg 60: 1404-5, 1995.
6. Kouchoukos NT. Adjuncts to reduce the incidence of embolic brain injury during operations on the aortic arch. Ann Thorac Surg 57: 243-5, 1994.
7. Lengua F, Cohen R, L´Huillier B, Buffet JM. Arteriovenous revascularization for lower limb salvage in unreconstructible arterial occlusive disease (long term outcome). VASA 24: 261-9, 1995.
8. Menasché P, Subayi JP, Piwnica A. Retrograde coronary sinus cardioplegia for aortic valve operations: a clinical report on 500 patients. Ann Thorac Surg 49: 556-64, 1990.
9. Palevsky HI, Fishman AP. The management of primary pulmonary hypertension. JAMA 265: 1014-20, 1991.
10. Prielipp RC, McLean R, Rosenthal MH, Pearl RG. Hemodynamic profiles of prostaglandin E1, isoproterenol, prostacyclin, and nifedipine in experimental porcine pulmonary hypertension. Crit Care Med 19: 60-7, 1991.
11. Rich A. Medical treatment of primary pulmonary hypertension: a bridge to transplant? Am J Cardiol 75: 63a-6a, 1995.
12. Salerno TA, Houck JP, Barrozo CAM, et al. Retrograde continuous warm blood cardioplegia: a new concept in myocardial protection. Ann Thorac Surg 51: 245-7, 1991.
13. Varela A, Montero CG, Cordoba M, et al. Improved distribution of pulmonary flush solution to the tracheobronchial wall in pulmonary transplantation. Eur Surg Res 29: 1-4, 1997.
14. Zin W. Fisiologia do sistema respiratório. In: Bethlem N. Pneumologia. São Paulo: Atheneu, 39-53, 1995.

REVIEW AND COMMENTARY

Commentary by Mark M. Levinson, MD

This study by Mulinari and colleagues raises some unique questions. It would be even more revolutionary if the authors had answered some of the questions which they raised when creating this fascinating model. It is hoped that the publication of their report will stimulate the authors or others to repeat this experiment with additional data to support the underlying concepts.

At first reading, the purpose of reversing the pulmonary circulation is not obvious. Why do this? The authors do not provide a full discussion of their reasons for undertaking the reversal operation, but it becomes more apparent as their data is analyzed. Many forms of right heart failure are due solely to increased pulmonary vascular resistance. If the arterial circulation is damaged to the point that the microcirculation loses its capacitance and the total resistance is increased, pulmonary hypertension and right heart failure result. In general, the prognosis and treatment alternatives for this situation are poor. There are many etiologies, including congenital heart disease with pulmonary overcirculation, mitral valve disease with chronic atrial hypertension, recurrent pulmonary emboli, severe emphysema, and primary pulmonary hypertension. The authors do not discuss the differences between these various etiologies in their proposed operation, but clearly they are not all the same. For example, the cause of pulmonary hypertension from repeated emboli is very different than the cause in congenital heart disease with overcirculation. None-the-less, the concept of using the unobstructed pulmonary venous bed to circumvent resistance disease in the pulmonary arterial bed is both unique and potentially revolutionary in the treatment of end-stage pulmonary vascular resistance diseases.

Although the etiology of primary pulmonary hypertension (PPH) is not known, the potential to treat this entity without transplantation is one of the spin-offs of this article. As PPH progresses, hypoxia and right heart failure become terminal events. Failure of the right ventricle mandates replacement of both the lungs and heart (i.e., heart-lung transplantation). In patients who do not manifest severe right-sided heart failure, a pulmonary transplant can lower the pulmonary vascular resistance and provide enough gas exchange for survival. The natural question after reviewing the paper by Mulinari is whether the pulmonary reversal operation would accomplish the same end without the need for a donor organ or post transplant antirejection therapy. The answer to this question will likely be found by reviewing the pathology of explanted lungs from PPH patients undergoing heart-lung or lung transplantation. If the venous vessels are not involved with the primary pathologic process and the venous resistance is normal, then the reversal operation of Mulinari could be a potential "cure". However, if the venous vessels are fibrotic or the venous resistance is also elevated (in a passive flow ex vivo test), then the reversal operation would not offer anything beneficial.

Mulinari et al. provided an intriguing question about the future of patients with PPH, but unfortunately, this cannot be easily answered in an animal model. It is difficult to simulate PPH in animals. One possible means to do so is high altitude heart failure which develops in some animals (cattle) taken to hypoxic altitudes. Unlike high altitude pulmonary edema, this is a chronic form of right heart failure presumably from continuous low alveolar paO_2 and hypoxia with secondary pulmonary vascular changes. Animals with this condition may be a good model for testing the hypothesis that pulmonary circulation reversal will correct the pulmonary hypertension and right heart failure associated with PPH.

The pulmonary reversal operation may have some role in chronic pulmonary emboli if the newly established drainage through the pulmonary artery branches is not obstructive. The capacitance of the pulmonary arteries to drain the entire pulmonary blood flow in the face of major embolic obstruction has not been demonstrated. Again, an animal model would be difficult to obtain.

One of the brighter prospects for the Mulinari operation is the potential to treat certain forms of congenital heart disease with pulmonary reversal. Children with overcirculation (single ventricle with septal communications, ventricular septal defect [VSD], adult patent ductus arteriosus [PDA]) may be one population that can be served with this procedure. For example, one of the chief determinants of failure of the Fontan correction for single ventricle is the presence of eleveated pulmonary vascular resistance. Children with chronic aorto-pulmonary shunts

gradually develop increased pulmonary resistance with time. Although this is multifactorial (with a component of LV dysfunction contributing), changes of chronic pulmonary vascular obstructive disease (PVOD) can render these patients inoperable for a Fontan. The presence of Eisenmengers physiology (reversal of left-to-right shunt into a right-to-left shunt) is a sign of inoperability in lesions that are otherwise correctable, such as VSD or PDA. Although uncorrected VSDs and PDAs are now rare due to excellent early childhood diagnostics, there are cases of adults with Eisenmengers physiology. At the present state of knowledge, these patients cannot be corrected because of PVOD. Inversion of the pulmonary circulation may convert these patients into candidates for surgical correction of their intracardiac defect. In the case of VSD with Eisenmengers, could the patient have a pulmonary reversal and VSD closure? In the case of a child with single ventricle or truncus with PVOD, could the child have a pulmonary reversal and then later become a candidate for palliation or correction?

It is intriguing to speculate how the Mulinari operation would work in other states, such as cor pulmonale from end stage emphysema. It is likely that such patients would not tolerate any surgery of any kind, but what if pulmonary circulation reversal improved both gas exchange and pulmonary vascular resistance? My opinion is that the parenchymal process that causes cor pulmonale will affect the venous capacitance vessels and the alveolar-capillary membrane as well the arterioles and thus mitigate against a beneficial result, but this is speculative. As there are millions affected by chronic obstructive pulmonary disease, investigating the possibility of relief using pulmonary reversal would need to be done at some point. Mulinari et al. did demonstrate that reversal could be accomplished without cardiopulmonary bypass, which would be necessary in this very frail population.

Unfortunately, the data from this initial report does not complete the picture of pulmonary reversal. First, the patency of the 8 mm diameter PTFE graft is a serious question. It is encouraging that 7 of 8 grafts in the experiment were patent, but this may not be achievable in humans where small PTFE grafts do not have a high long term patency rate. However, it is assumed that humans would require a much larger graft. Another natural extension of their concept would be a tube constructed of native or bovine pericardium to bridge the distance between the main pulmonary artery and the hilar venous structures. Pericardium is easier to work with and less thrombogenic [Pires 1999].

Also, their report did not address the possible contributions of the other remaining (normal) lung in this canine experiment. Sampling of blood gas values from the aorta will not prove the efficacy of gas exchange in the reversed lung. Normal blood gases in the aorta could have been seen even if the reversed lung had failed since the contralateral was entirely normal. The author did not perform chest radiographs which would have helped to document the vasculature and ventilation.

The authors should repeat their experiment, but perform the second surgery through a sternotomy and obtain differential blood gas analysis by sampling the venous effluent from each lung separately. This would permit the calculation of the capillary exchange ratio, or $AaDO_2$ of each lung. Other data that would be needed include the following from each lung (reversed and non-reversed): 1) calculation of the pulmonary vascular resistance, 2) weight and water content, and 3) pulmonary compliance (ventilation pressure/volume curves). These data were not reported in the current study and would be best obtained with bilateral instrumentation through a sternotomy at the reoperation experiment. If the results demonstrate equal performance of the reversed and non-reversed lung, then their concept would indeed be validated.

Despite the brevity of this report and the data that was not captured, the potential of their concept invites further research. One of the current limitations of cardiac surgery is the inability to correct PVOD and chronic pulmonary hypertension. The Mulinari operation brings new potential to this arena and I encourage him and his colleagues to continue development and documentation of this bold concept.

REFERENCES

1. Pires AC, Saporito WF, Leão, LEV, Forte V, Cardoso SH, Ramaciotti O. Bovine Pericardium Used as a Cardiovascular Patch. Heart Surgery Forum #1999-16834; 2(1):60–69, 1999.