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In-Parallel Attachment of a Low Resistance Compliant Thoracic Artificial Lung under Rest and Simulated Exercise

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Abstract

Background—Previous thoracic artificial lungs (TALs) had blood flow impedances greater than the natural lungs, which could cause abnormal pulmonary hemodynamics. New, compliant TALs (cTALs), however, have an impedance lower than the natural lung.

Methods—In this study, a new cTAL design was attached between the pulmonary artery and left atrium in five sheep (60.2 ± 1.9 kg). A distal pulmonary artery band was placed to control the percentage of cardiac output routed to the cTAL. Rest and exercise conditions were simulated using a continuous dobutamine infusion of 0 and 5 mcg/kg/min, respectively. At each dose, a hemodynamic data set was acquired at baseline (no flow to the cTAL) and 60, 75, and 90% of CO shunted to the cTAL.

Results—Device resistance did not vary with blood flow rate, averaging 0.51 ± 0.03 mmHg/(L/min). Under all conditions, cardiac output was not significantly different from baseline. Pulmonary system impedance only increased above baseline with 5 mcg/kg/min of dobutamine and 90% of cardiac output diverted to the cTAL.

Conclusion—Results indicate minimal changes in pulmonary hemodynamics during pulmonary artery – left atrium cTAL attachment for high device flows under rest and exercise conditions.

Keywords

artificial organs; transplantation; lung; ventricle; right; pulmonary vascular resistance/hypertension

Introduction

The only long term solution for chronic lung disease is lung transplantation; however, organ donation is limited and cannot supply the demand [1]. There is a need for a device which can serve as a bridge to lung transplant for patients with end-stage lung disease. Thoracic artificial lungs (TAL) are being developed for these patients and would ideally allow patients to be awake and ambulatory while awaiting lung transplant.

TALs are attached to the pulmonary circulation, and thus their blood flow is provided by the right ventricle (RV). It is essential that these devices have low blood flow resistance to prevent overloading the RV and causing decreases in cardiac output (CO). Several studies

have shown that CO decreases linearly with the pulmonary system zeroth harmonic pulmonary input impedance, Z_0 [2,3] a measure of RV afterload [4]. During TAL use, Z_0 is influenced by the TAL attachment mode and the resistance of the TAL, device inlet graft anastomosis, and native lung [5,6].

The most commonly proposed TAL attachment modes are in parallel or in series with the natural lungs. During in parallel attachment blood flow is routed from the pulmonary artery (PA), through the TAL and then returned to the left atrium (LA). In patients with pulmonary hypertension this attachment mode is ideal, as it reduces Z_0 for the combined TAL and natural lung system and thus unloads the right ventricle. In this setting, TAL attachment should ideally result in a pulmonary system Z_0 that is as close as possible to the healthy natural lung. To accomplish this, the combined resistance of the TAL and the anastomoses used to attach it must also be close to that of the healthy natural lung.

This paper presents the first *in vivo* test of a new, ultra-low resistance compliant TAL (cTAL) that is designed to meet that goal. The device has a gradual blood inlet and outlet and a compliant housing that reduces blood flow recirculations. The housing is combined with a low-resistance fiber bundle design to arrive at an overall low device resistance. This study examined pulmonary hemodynamics and cTAL function during in parallel attachment in sheep with up to 90% of CO through the cTAL both at rest and during simulated exercise.

Methods

Compliant Thoracic Artificial Lung

A cTAL, consisting of a compliant Biospan (DSM PTG, Berkeley, CA) housing and polypropylene fiber bundle, was used in this study (Figure 1). In this device, blood flows into the inlet conduit, expands into the inlet manifold, flows through the fiber bundle at the center of the device, then travels through the outlet manifold and exits through the outlet conduit. To create the fiber bundle, woven mats of polypropylene fibers with a fiber diameter of 210 μm were wound into compact bundles with porosity, path length, and frontal area of 0.75, 0.038 m, and 0.013 m^2 , respectively.

Experimental Procedure

A total of five male sheep averaging 60.2 ± 1.9 kg were used in this study. All sheep received humane care in compliance with the “Guide for the Care and Use of Laboratory Animals” and all methods were approved by the University of Michigan Committee for the Use and Care of Animals. Anesthesia was induced with 6-9 mL/kg of propofol and then maintained after endotracheal intubation with 1-3% inhaled isoflurane. Sheep were mechanical ventilated with oxygen using a Narkomed 6000 ventilator (North American Dräger, Telford, PA). The ventilator was set to a tidal volume of 10 mL/kg and a frequency of 12 breaths/min and was adjusted to maintain an arterial partial pressure of carbon dioxide (PaCO_2) between 35 and 45 mmHg. A carotid arterial line and left jugular venous line were placed and then connected to fluid coupled pressure transducers (Hospira, Inc., Lake Forest, IL) for the continuous monitoring of arterial and central venous pressures (P_{Art} and P_{CV}).

A muscle sparing left thoracotomy was performed and the main pulmonary artery and left atrium (LA) were identified. Dacron vascular grafts (Terumo, Ann Arbor, MI) for artificial lung attachment were attached to the PA (between the pulmonary valve and bifurcation) and LA. An ultrasonic perivascular flow probe (Transonic 24AX, Transonic Systems, Inc., Ithaca, NY) was placed around the PA, proximal to the device inlet graft. This probe was connected to a flow meter (T400, Transonic Systems, Ithaca, NY) to allow for the measurement of PA flow (Q_{PA}). A pressure catheter (Becton Dickinson and Co., Franklin Lakes, NJ) was then inserted at the proximal PA and connected to a transducer for the

display and recording of the PA pressure (P_{PA}). A Rommel tourniquet was placed around the distal PA to allow for the adjustment of flow through the cTAL. Prior to device attachment, 1g of methylprednisolone (Solu-Medrol, Pfizer, New York, NY) was administered and the animal was anticoagulated with 100 IU/kg of intravenous sodium heparin (Baxter Healthcare Corp., Deerfield, IL) to maintain active clotting times of above 300 seconds.

The cTAL was primed with heparinized saline (10 U/mL) and then connected to the PA (device inlet) and LA (device outlet) grafts (Figure 2). An ultrasonic flow probe (Transonic 14PXL, Transonic Systems, Inc., Ithaca, NY) was placed around the inflow conduit and connected to a flow meter (T400, Transonic Systems Inc., Ithaca, NY) in order to measure device flow (Q_{cTAL}). Pressure transducers were connected to the device inlet and outlet to acquire device inlet and outlet pressure (P_{in} and P_{out}). A suction line was attached to the cTAL gas outlet and 95:5% O₂:CO₂ with 2-3% vaporized isoflurane was used as the sweep gas through the gas inlet. Sweep gas flow was adjusted during cTAL use to maintain PaCO₂ between 35 and 45 mmHg. A Hoffmann clamp was placed around the cTAL outlet conduit to restrict flow through the device. After cTAL attachment, clamps on the cTAL inlet and outlet conduits were removed and the Hoffmann clamp was slowly loosened until Q_{cTAL} = 1 L/min was achieved. This flow was maintained for 10 minutes for equilibration of fluid volumes and any inflammatory response. After device attachment, CVP decreased related to the systemic inflammatory response and 500 mL of hetastarch and 500 – 1000 mL of crystalloid were administered to restore CVP back to baseline values. Thereafter, the device conduits were clamped off and baseline data was taken, marking the start of the experiment.

A hemodynamic data set of Q_{PA} , Q_{cTAL} , P_{art} , P_{CV} , P_{PA} , P_{in} , P_{out} were digitally acquired for 10 seconds at a sampling frequency of 250 Hz through a BIOPAC data acquisition system (BIOPAC, Goleta, CA). Before attaching the cTAL, an animal baseline data set was acquired. Rest and exercise conditions were simulated using a continuous dobutamine (Hospira Inc., Lake Forest, IL) infusion of 0 and then 5 mcg/kg/min, respectively. At each dose, the hemodynamic data set was acquired at baseline (Q_{cTAL} = 0 L/min) and conditions of 60, 75, and 90% of CO shunted to the cTAL ($100 * Q_{cTAL} / Q_{PA}$), created by tightening the Rommel tourniquet around the PA. At each condition, 10 minutes were allowed for equilibration before data was taken. Also, at each condition, a device exit gas sample was taken along with blood samples from the animal, device inlet and device outlet.

Data Analysis

The zeroth harmonic pulmonary input impedance modulus, Z_0 , was calculated at each flow condition:

$$Z_0 = \frac{P_0}{Q_0} \quad (1)$$

where P_0 is the mean PA pressure and Q_0 is the mean PA blood flow rate. Device resistance, R , was calculated using the formula:

$$R = \frac{P_{in} - P_{out}}{Q_{cTAL}} \quad (2)$$

where P_{in} is the cTAL inlet pressure, P_{out} is the cTAL outlet pressure, and Q_{cTAL} is cTAL flow. Comparisons were performed on the rest and exercise data sets separately with SPSS 19 (SPSS, Chicago, IL). A mixed model was used with sheep number as the subject variable and flow condition (percentage of flow to the cTAL) as the fixed, repeated-measure

variable. All data is reported as mean $\pm$ standard error with a p-value of 0.05 or less being considered statistically significant.

Results

Animal Physiology

Before cTAL attachment, average animal baseline CO, mean arterial pressure (MAP), mean PA pressure (mPAP), central venous pressure (CVP), and Z_0 was 6.4 ± 0.59 L/min, 87.7 ± 8.08 mmHg, 18.2 ± 1.34 mmHg, 6.83 ± 1.22 mmHg and 2.99 ± 0.41 mmHg/(L/min), respectively. The effect of cTAL attachment on the sheep's CO is shown in Figure 3. Without dobutamine, the baseline CO was 5.5 ± 0.65 L/min and was maintained at or above this level until 90% flow to the cTAL. At 90% flow to the cTAL, there was a negligible, 0.33% decrease in CO from baseline which was not significant ($p=0.94$). At 5 mcg/kg/min of dobutamine, baseline CO was 7.23 ± 0.70 L/min and was maintained above this level until it decreased to 6.88 ± 0.69 L/min at 90% flow to the cTAL. This 5.6% drop was also not significant ($p=0.36$).

Figure 4 displays Z_0 for increasing flow to the cTAL. For both rest and dobutamine-simulated exercise conditions, Z_0 decreases from baseline with 60% flow to the cTAL. Without dobutamine, Z_0 remains below baseline at all conditions. At 5 mcg/kg/min of dobutamine, baseline Z_0 decreases due to increased blood flow to the natural lungs. As more flow is diverted to the TAL, Z_0 is initially relatively constant but begins to rise at 75% flow. As the natural lung is increasingly excluded, Z_0 then rises towards the same Z_0 as without dobutamine. At 90% flow, Z_0 has increased 50% above the 5 mcg/kg/min dobutamine baseline and is almost equivalent to all 0 mcg/kg/min conditions. This increase was significant ($p=0.049$) when compared to baseline.

MAP and mPAP for both dobutamine levels are displayed in Table 1 at each target and actual percent flow to the cTAL. The mean baseline PA pressure at 0 mcg/kg/min dobutamine was 25.1 ± 5.31 mmHg with no significant change at 60, 75 and 90% flow to the cTAL ($p = 0.35, 0.46, \text{ and } 0.86$). At 5 mcg/kg/min dobutamine, mPAP increased from 20.5 ± 1.48 mmHg at baseline to 29.4 ± 3.61 mmHg at 90% flow to the cTAL; however, this increase approached but was not significant ($p=0.09$). Baseline MAP was 78.4 ± 12.6 mmHg with no dobutamine and decreased slightly to 73.1 ± 7.82 mmHg at 90% flow to the cTAL. With 5 mcg/kg/min of dobutamine, baseline MAP was 78.5 ± 4.98 mmHg and decreased to 67.3 ± 6.92 mmHg at 90% flow to the cTAL; however, this decrease was not significant ($p=0.16$).

Device Performance

Device resistance at various flow rate ranges is shown in Figure 5. Resistance of the cTAL remains relatively constant at all tested flow rates. Device resistance averaged 0.51 ± 0.01 mmHg/(L/min), ranging from 0.50 ± 0.01 mmHg/(L/min) at 2-3 L/min to a maximum of 0.55 ± 0.02 mmHg/(L/min) at 5-6 L/min. Accordingly, device flow did not significantly effect resistance ($p=0.29$). Though this was not primarily a gas exchange experiment, the cTAL also exchanged gas effectively. Arterial PO_2 , PCO_2 and pH, along with cTAL O_2 and CO_2 gas transfer rates (VO_2 and VCO_2) are displayed in table 2 for each device flow condition. The device outlet oxyhemoglobin saturations were above 99% at every flow condition, although venous conditions were not sufficient to challenge the device. A typical hemoglobin and device inlet PO_2 was 7.5 and 80 mmHg, respectively. Even with this higher inlet PO_2 , VO_2 rates ranged from 85.7 ± 7.7 mL/min at 0 dobutamine and 60% CO to the cTAL to 155.5 ± 8.8 mL/min at 5 dobutamine and 90% CO to the cTAL. Lastly, at the conclusion of the experiment, there was no visible clot formation in the device.

Comment

To date, there is no commercially available TAL. Over 18 years of development, these devices have always featured excellent gas exchange and research has thus focused on developing devices with progressively lower blood flow resistance and improved hemodynamics during *in vivo* attachment [7,8,9]. Ideally, TALs should be able to provide the majority of the gas exchange while being able to maintain normal pulmonary hemodynamics.

As there is no commercial device for this application, a few groups have investigated clinical PA-LA attachment in cases of severe pulmonary hypertension using the lowest resistance gas exchanger on the market, the Novalung ILA. Two of these patients had primary pulmonary hypertension and four had pulmonary veno-occlusive disease. Five of the patients (87%) were successfully bridged. Despite this success, the Novalung was not designed for this application. Its intended use is arterio-venous CO₂ removal, which features much lower blood flows of 2 L/min and higher driving pressures. As a result, the Novalung's gas exchange capabilities are low for the application and the resistance is approximately 5-6 mmHg/(L/min) at blood flows of 2-2.5 L/min [10,11,12]. To support COPD, IPF, and septic patients, greater gas exchange will be required. Moreover, in cases of marked pulmonary hypertension, it would be ideal to be able to fully unload the RV and eliminate the need for inotropes.

In the current study, the new cTAL design was tested in healthy animals with normal PA pressure. Therefore, the goal for this study was to maintain normal PA pressures and CO with up to 90% of CO diverted to the cTAL. Results indicate that these goals were met. cTAL attachment caused only minimal, statistically insignificant decreases in CO and increases in PA pressure from baseline at all conditions. At the most extreme condition, simulated exercise with 90% of the CO diverted to the cTAL, CO decreased 5.6% and PA pressure increased 8.8 mmHg. This, however, represents an extreme case. A more typical, and advisable [13], condition would likely be 75% of CO. Here, CO was identical to baseline values, and PA pressure decreased at rest and increased a small amount during simulated exercise.

Maintenance of normal pulmonary hemodynamics was the result of very low cTAL resistance. Average resistance for the range of flows tested was 0.51 ± 0.01 mmHg/(L/min), well below that of the natural lung. Moreover, the resistance of the device did not change significantly as flow increased. The low cTAL resistance, in turn, maintains low Z_0 . Previous studies have shown that Z_0 is the dominant variable affecting CO during TAL attachment, with CO decreasing as Z_0 increases [5,6,14]. In this study, Z_0 is lower than baseline at 60% flow to the cTAL due to the second, parallel flow path provided by the cTAL and the minimal banding of the PA at that condition. As the PA is banded further, the natural lung portion of the system is closed, the percentage of flow to the cTAL increases, and Z_0 increases slightly. However, since device resistance remains low, the resulting Z_0 remains small and similar to that of healthy natural lungs. At 5 mcg/kg/min of dobutamine, however, high CO and PA pressure leads to lower baseline Z_0 . As flow is diverted to the cTAL, Z_0 then increases to the same level seen with in the 0 dobutamine case.

Based on these results, the cTAL will be able to completely unload the right ventricle *in vivo* during PA-LA attachment in patients with any degree of pulmonary hypertension. To examine this one can use the equation $P_{PA} = CO * R + P_{LA}$, where P_{PA} is the PA pressure, P_{LA} is the left atrial pressure, and CO is cardiac output. R is the resistance of the parallel artificial and natural lung system. If there is no PA banding, $R = [R_N * (R_T + R_A)] / [R_N + R_T + R_A]$, in which R_N is the natural lung resistance, R_T is the TAL resistance, and R_A

is the resistance of the TAL anastomoses or cannulae. For $CO = 6$ L/min, $P_{LA} = 6$ mmHg, $R_T = 0.5$ mmHg/(L/min), and $R_A = 0.87$ mmHg/(L/min), and $R_N = 9$ mmHg/(L/min) [14], the PA pressure prior to attaching the TAL would be 60 mmHg. With the cTAL, it falls to 13 mmHg. In comparison, under the same conditions, the Novalung ILA ($R_T = 6$ mmHg/(L/min)) would reduce PA pressure to 29 mmHg.

The other experimental TAL in development is the Biolung® (MC3, Ann Arbor, MI). The hard-shell Biolung was tested in a study similar to this one [15]. The only exception is that Akay *et al* simulated rest (no dobutamine), ambulatory (2 mcg/kg/min dobutamine), and exercise (5 mcg/kg/min dobutamine) conditions. Results showed that CO was maintained as the percentage of CO to the device increased at resting and ambulatory conditions. At exercise conditions, CO decreased with increasing flow to the TAL up to $23 \pm 5\%$ at 90% flow diverted through the TAL. Direct comparison between these two studies is difficult, as the baseline Z_0 and baseline cardiac response to dobutamine varied greatly. In the Biolung study, baseline Z_0 were lower and, as a result, baseline CO was greater at the same dobutamine doses. As a result, the easiest means of comparison between these studies is the Z_0 with 90% of cardiac output through the artificial lung. Here, hemodynamics are largely unaffected by natural lung resistance. At 90%, Z_0 ranged from 4.5-4.9 in the Biolung study and 4.1-4.5 in the cTAL study. This small difference is similar to what one would predict by the difference in TAL resistances in these studies. The cTAL resistance is approximately 0.3 mmHg/(L/min) smaller with flow rates of 2-3 L/min and approximately 0.65 mmHg/(L/min) lower at flow rates of 5-6 L/min. Thus the Biolung should provide slightly less but similar unloading.

Ultimately, this study suggests that the cTAL is capable of being used clinically, in parallel with the native lungs and under high flow conditions. Minimal decreases in CO were seen at 90% flow to the device at both dobutamine levels, indicating exercise would be possible during cTAL attachment. Since cTAL resistance is small, flow should route preferentially through the device. This attachment mode could be used with any patients with chronic respiratory insufficiency but would be ideal for patients with high pulmonary vascular resistance (PVR) since the device resistance would be much lower than the native lung resistance. Approximately 57% of CO went through the cTAL with no PA banding in these sheep with normal PVR. Assuming $PVR = 2.25$ mmHg/(L/min) in patients with pulmonary hypertension, about 70% of the CO would flow through the cTAL with no PA banding.

Further long-term testing (14 days) of in parallel attachment of the cTAL is necessary in healthy sheep and sheep with pulmonary hypertension. These studies would focus on the biocompatibility of the device in a similar fashion as the studies of Sato *et al.* with the Biolung [16,17]. Those studies utilized simple heparin anticoagulation with ACTs from 180-220s. That said, future studies with this device should examine various surface coatings as well as different anticoagulation strategies, including aspirin to inhibit platelets as sometimes used with the Novalung ILA [10]. Although farm sheep tend to be more procoagulant than humans, these studies may help to define clinical anticoagulation strategies.

Conclusion

The cTAL resistance is lower than the natural lung, averaging 0.51 ± 0.03 mmHg/(L/min). Use of the cTAL in a PA-LA configuration caused no change in CO under rest and exercise conditions. Thus, the cTAL can provide PA-LA respiratory support without significant changes in pulmonary hemodynamics in healthy sheep and will be capable of unloading the RV in subjects with pulmonary hypertension.

Acknowledgments

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List of Abbreviations

CO	Cardiac output
CVP	Central venous pressure
cTAL	Compliant thoracic artificial lung
LA	Left atrium
MAP	Mean arterial pressure
mPAP	Mean pulmonary artery pressure
PA	Pulmonary Artery
PVR	Pulmonary Vascular Resistance
RV	Right Ventricle
TAL	Thoracic artificial lung
Z₀	Zeroth harmonic pulmonary input impedance

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Figure 1.
Blood flow path in the cTAL

Figure 2.
PA-LA cTAL attachment and instrumentation

Figure 3.
Cardiac output at varying percentages of cardiac output diverted to the cTAL for dobutamine doses of 0 and 5 mcg/kg/min

Figure 4.
Zeroth harmonic impedance modulus, Z_0 , at varying percentages of cardiac output diverted to the cTAL for dobutamine doses of 0 and 5 mcg/kg/min

Figure 5.
cTAL resistance at varying blood flow rate ranges

Table 1

Mean pulmonary artery pressure and mean arterial pressure for varied percentages of the cardiac output to the cTAL

Percentage of CO to cTAL				
Dobutamine Dose	Goal	Actual	mPAP (mmHg)	MAP (mmHg)
0 mcg/kg/min				
	BL – 0%	0 ± 0%	25.1 ± 5.3	78.4 ± 12.6
	60%	62 ± 1%	21.6 ± 4.2	72.3 ± 6.6
	75%	74 ± 2%	22.0 ± 4.6	73.5 ± 7.8
	90%	91 ± 2%	24.3 ± 5.2	73.1 ± 7.8
5 mcg/kg/min				
	BL – 0%	0 ± 0%	20.5 ± 1.5	78.5 ± 5.0
	60%	61 ± 2%	20.5 ± 2.6	73.2 ± 2.6
	75%	76 ± 2%	26.5 ± 3.2	73.7 ± 6.1
	90%	93 ± 2%	29.4 ± 3.6	67.3 ± 6.9

BL = baseline; CO = cardiac output; cTAL = compliant thoracic artificial lung; MAP = mean arterial pressure; mPAP = mean pulmonary artery pressure

Table 2

Arterial PO₂, PCO₂ and pH, and cTAL O₂ and CO₂ gas transfer rates (VO₂ and VCO₂) for varied percentages of CO to the cTAL.

Dobutamine Dose	% CO to cTAL	Arterial PO ₂ (mmHg)	Arterial PCO ₂ (mmHg)	Arterial pH	cTAL VO ₂ (mL/min)	cTAL VCO ₂ (mL/min)
0 mcg/kg/min						
	60%	439 ± 57	38.9 ± 1.3	7.40 ± 0.01	85.7 ± 7.7	213.6 ± 17.1
	75%	460 ± 49	38.7 ± 1.3	7.41 ± 0.01	96.0 ± 6.8	238.6 ± 58.1
	90%	442 ± 35	37.1 ± 3.6	7.40 ± 0.01	109.1 ± 8.2	277.5 ± 21.5
5 mcg/kg/min						
	60%	399 ± 37	43.6 ± 1.6	7.37 ± 0.02	103.3 ± 11.1	326.0 ± 16.6
	75%	442 ± 35	45 ± 1.8	7.36 ± 0.02	120.7 ± 10.9	369.5 ± 31.4
	90%	422 ± 38	44.4 ± 1.3	7.36 ± 0.01	155.5 ± 8.8	424.0 ± 31.5

CO = cardiac output; cTAL = compliant thoracic artificial lung; VCO₂ = CO₂ gas transfer rate; VO₂ = O₂ gas transfer rate