Cavopulmonary assist: long-term reversal of the Fontan paradox

Short title: Cavopulmonary assist for long-term Fontan reversal

Mark D Rodefeld MD, a Alison Marsden PhD, b Richard Figliola PhD, c Travis Jonas d, Michael Neary e, Guruprasad A Giridharan PhD, f

From a the Section of Cardiothoracic Surgery, Department of Surgery, Indiana University School of Medicine and James Whitcomb Riley Hospital for Children, Indianapolis, IN, b The Department of Bioengineering and Pediatrics, Stanford University, Stanford, CA, c Department of Mechanical Engineering, Clemson University, Clemson SC, d Mechanical Solutions Inc., Whippany, NJ, e Rotor Bearing Technology and Software Inc, Phoenixville, PA, f The Department of Bioengineering, University of Louisville, Louisville, KY

Article word count: 3485/3500

Supported in part by National Institutes of Health Bioengineering Research Grant R01 HL098353, Big Hearts to Little Hearts, Children’s Heart Foundation, Matthews Hearts of Hope.


This is the author’s manuscript of the article published in final edited form as:

Address for correspondence: Mark D Rodefeld MD, Professor of Surgery, Section of Cardiothoracic Surgery, Indiana University School of Medicine, Emerson Hall 215, 545 Barnhill Drive, Indianapolis, Indiana 46202. Tel: 317-944-7150. Fax: 317-274-2940. Email: rodefeld@iupui.edu.
Structured Abstract:

Objective: Fontan circulatory inefficiency can be addressed by replacing the missing subpulmonary power source to reverse the Fontan paradox. An implantable cavopulmonary assist device is described that will simultaneously reduce systemic venous pressure and increase pulmonary arterial pressure, thereby improving preload and cardiac output, in a univentricular Fontan circulation on a long-term basis.

Methods: A rotary blood pump based on the von Karman viscous pump was designed for implantation into the total cavopulmonary connection (TCPC). It will impart modest pressure energy to augment Fontan flow without risk of obstruction. In the event of rotational failure, it is designed to default to a passive flow diverter. Pressure-flow (H-Q) performance was characterized in vitro in a Fontan mock circulatory loop using blood analog.

Results: The pump performed through the fully specified operating range, augmenting flow in all 4 directions of the TCPC. Pressure rise of 6-8 mmHg was readily achieved, ranging up to 14 mmHg at highest speed (5600 RPM). Performance was consistent over a wide range of cardiac output. In the stalled condition (0 RPM), there was no discernible pressure loss across the TCPC.

Conclusions: A blood pump technology is described that can reverse the Fontan paradox and may permit a surgical strategy of long-term biventricular maintenance of a univentricular Fontan circulation. The technology is intended for Fontan failure in which right-sided circulatory inefficiencies predominate and ventricular systolic function is preserved. It may also apply prior to clinical Fontan failure as health maintenance to preempt the progression of Fontan disease.

Abstract word count: 249/250
Central Message: An implantable blood pump that can safely and reliably reverse the Fontan paradox long-term may enable a physiologically curative strategy for single ventricle heart disease. (Characters including spaces: 174/200)

Perspective Statement: Fontan palliation is associated with a chronic circulatory inefficiency that culminates in secondary disease, failure, and attrition. We report feasibility of an implantable cavopulmonary assist device that can safely replace the missing subpulmonary power source and reverse the Fontan paradox long-term. This may enable a physiologically curative strategy for single functional ventricle. (characters including spaces: 390/405)

Central picture Legend: Cavopulmonary assist device: flow augmentation through the total cavopulmonary connection. (Characters including spaces: 90/90)

Glossary of Abbreviations

- MCS, mechanical circulatory support
- TCPC, total cavopulmonary connection
- SVC, superior vena cava
- IVC, inferior vena cava
- RPA, right pulmonary artery
- LPA, left pulmonary artery
- VAD, ventricular assist device
Repair of single functional ventricle currently consists of staged surgical approach that terminates in a univentricular Fontan circulation. Since its introduction, Fontan palliation has dramatically improved the outcome for all forms of single ventricle heart disease [1,2]. However, Fontan survivors are committed for the remainder of their lives to a state of chronic circulatory inefficiency due to the lack of a subpulmonary ventricle.

This results in coexisting elevated systemic venous pressure and reduced cardiac output, known clinically as the Fontan paradox [3]. Combined, these problems have been clearly shown to progress to late Fontan failure and attrition [4]. Despite extensive clinical and research effort, therapies to prevent or reverse this circulatory decline remain extremely limited with no breakthroughs for decades [5]. With an increasing number of late Fontan survivors, Fontan failure is now a routinely encountered and challenging clinical problem.

We have theorized that replacement of the missing subpulmonary power source would reverse the Fontan paradox and shift the univentricular circulation toward more stable biventricular equivalency [6]. It would decongest the systemic venous and lymphatic circulations, while simultaneously improving cardiac output. If applied as durable long-term support, it could in theory preempt Fontan-associated disease progression by maintaining biventricular equivalency.

Despite the simplicity and attractiveness of this concept, the technologic considerations are complex and dissimilar to any other mechanical circulatory support (MCS) application. We have found that existing MCS technologies are ill-suited and unmodifiable to address Fontan circulatory inefficiency. Alternatively, we have focused on the development of an anatomically-specific device that can replace the missing subpulmonary power source and reverse the Fontan
paradox [7]. This report describes the computational and in vitro basis for an implantable
cavopulmonary assist device concept that has potential to safely and durably restore biventricular
equivalency in a univentricular circulation.

Methods:

Pump design: In previous studies, we have demonstrated that a remotely powered rotary
pump, based on the von Karman viscous pump, can provide 4-way flow augmentation through
the total cavopulmonary connection (TCPC) [7]. A biconical impeller suspended in the midst of
the TCPC functions as a 2-sided centrifugal pump, drawing inflow from the superior and inferior
vena cavae (SVC, IVC), and augmenting outflow to the left and right pulmonary arteries (LPA,
RPA) at physiologic pressure. A modest pressure step-up of 6 mmHg has been shown in vitro
and in vivo to restore the univentricular Fontan circulation to biventricular equivalency under
normal physiologic conditions [6,7]. We have also demonstrated that a cavopulmonary assist
device based on the von Karman viscous pump will not obstruct the systemic venous pathway in
the event of rotational dysfunction, and will instead reduce kinetic energy loss in the TCPC [7].

Because an external power source would limit such a device to temporary use, a
permanent power source was incorporated in the form of an outrunner brushless DC (BLDC)
motor. In an outrunner motor, the rotating component is outside rather than inside (Figure 1). For
an in-series pump situated in the Fontan venous pathway, this configuration is key to eliminating
the risk of venous pathway obstruction because it allows preservation of a wide passage between
the impeller and housing. As a result, the pump can serve in 2 distinctly different capacities:
rotating, it functions as a pump; non-rotating, it functions as a static flow diverter (Figure 2). Thus, it is intended to be beneficial even if it fails.

A biconical stator, containing iron laminations and copper windings, is located centrally within the impeller, and has no blood contact or moving parts. It is surrounded by a biconical rotor with embedded magnets as the only moving component. Rotor surface vanes provide low-pressure, high-volume augmentation of cavopulmonary flow (Video 1). A secondary (internal) flow path between the rotor and the stator is an inherent centrifugal pump and integrates several key functions: 1) electromagnetic flux transfer for motor torque; 2) heat dissipation; 3) bearing lubrication; 4) rotordynamic stabilization (Video 2). The housing is a double inlet, double outlet thin-walled hardcase shell that serves only to direct TCPC flow and structurally suspend the pump. Wiring from the stator travels through the central shaft to exit the housing via shaft struts. Because there is no external component bulk, the pump is no larger than the native TCPC, and is suitable for in situ implantation.

An advanced prototype was designed and fabricated at adult scale (25 mm dia inlets, 20 mm dia outlets) (Figure 3). Using computational fluid dynamic (CFD) modeling, impeller dimension and motor size were determined based on the torque required to achieve a pressure rise of 6-8 mmHg under physiologic conditions. Non-circumferential conical bearings support the rotor at each end. The impeller surface vane height is only 0.7 mm. The pump was tested initially in an open circuit, with no baseline imposed flow, using blood analog (water/glycerin 63/37, physiologic density 1060 kg/m$^3$, 3.2 cP). Testing was then performed in a static loop for pressure-flow (H-Q) performance, and lastly in vitro in a mock loop of a Fontan circulation for physiologic performance. For static performance, the pump was operated at 0-5600 RPM against 5 different resistances. Steady-state pressure head and flow rates were plotted to characterize
hydraulic (H-Q) performance. Inlet partial and complete occlusion performance was assessed.

For physiologic performance testing, a Fontan mock circulatory system was utilized that includes compliance and resistance elements [7].

**Results:**

All design objectives were met, including: 1) pressure-flow performance of 0-14 mmHg pressure rise over 0-5600 RPM; 2) mean scalar shear stress < 100 Pa, transit time < 0.01s; 3) non-cavitating performance; 4) acceptable recirculation; 5) no pressure loss in the static condition (0 RPM). Heat dissipation was excellent, confirmed by reduction of core temperature at higher speed. Hydraulic performance was consistent over a wide range of cardiac output (Figure 4). A peak pressure of 14 mmHg was achieved at the highest speed. The pump generated 5 L/min of flow and 6 mmHg pressure head at 4000 RPM, and 5 L/min of flow and 8 mmHg pressure head at 5600 RPM. The in-vitro pump performance was consistent with the design specifications and CFD modeling (6-8 mmHg pressure rise under physiologic conditions). The pump generated inflow from both the SVC and IVC equally to both pulmonary arteries for SVC:IVC inflow ratios ranging from 30:70 to 50:50. The pump effectively mixed inflow from both inlets with symmetric outflow distribution (hepatic factor, Video 1). The power draw was less than 6W at maximal speed, and is expected to improve with further optimization. Because the surface vane profile is so low, there is flexibility to increase vane height for higher pressure performance without compromising the no-obstruction constraint.

In *in vitro* physiologic mock loop testing, the device effectively reversed the Fontan (Figure 5). With increasing RPM, ventricular end-diastolic pressure and aortic pressure
increased. At 5000 RPM, the pump converted a cavopulmonary pressure head of positive 2.8 mmHg to negative 4.8 mmHg, replacing a small energy loss with a proportionally larger gain. It boosted pulmonary arterial pressure by 6 mmHg, and increased preload sufficient to increase cardiac output by 12.8%. Vena caval pressure was reduced 1.4 mmHg, which is significant in this segment of the circulation where even small reduction of pressure is beneficial to splanchnic health. Increase in pump speed beyond 5000 RPM caused a larger negative cavopulmonary pressure head but did not lead to a measurable increase cardiac output. When the pump was stopped (0 RPM), there was no discernible pressure drop, validating the no-obstruction design constraint. Importantly, in the stopped condition, TCPC turbulence was reduced compared to the condition with no pump installed.

Discussion:

Amongst the many significant advances in the treatment of congenital heart disease over the past 5 decades, single ventricle palliation has had perhaps the largest impact. Nonetheless, it was predicted by its’ innovators that it may be incompatible with a normal lifespan and require improvement or modification [9-11]. As predicted, Fontan-associated disease now represents one of the most significant challenges in the field. Although Fontan palliation is lifesaving, it carries a lifelong chronic disease burden that has no primary preventive therapy and is ultimately life-limiting [12,13]. As a reflection of its palliative nature, late Fontan attrition is relatively constant suggesting that immutable factors play a role, and 30-year survival is only 43-70% [14-16].

Solutions to address Fontan-associated disease have been largely incremental and of unclear benefit, and we may have reached a plateau. Fontan optimization can at best only
prolong an inherently inefficient system because it does not address the underlying physiologic
deficit: the lack of a subpulmonary ventricle. To date, little effort has focused on mechanical
replacement of a subpulmonary power source, presumably because a comprehensive solution is
unobvious and highly complex. However, replacement of the missing subpulmonary power
source is physiologically compelling: it will normalize systemic venous pressure, decongest the
lymphatic circulation, and normalize ventricular loading conditions, emulating biventricular
efficiency. Given the ongoing problems with Fontan-associated disease, it is reasonable to
reconsider the current paradigm. Rather than Fontan perpetuation, a preferable strategy may be
to reverse the Fontan [17].

Fontan failure is largely non-ventricular failure: Although those with failing Fontan
circulations may exhibit classic features of congestive heart failure, the primary cause is not
typically ventricular systolic dysfunction. Prior to end-stage Fontan disease, ejection fraction is
preserved in >70% of patients; diastolic dysfunction predominates [18]. While the mechanisms
are complex and multi-factorial, Fontan diastolic dysfunction has been largely attributed to
chronic preload deprivation [19,20]. Over time, abnormal loading conditions (decreased preload,
increased afterload) result in cardiomyopathic remodeling and fibrosis [21], and eventual systolic
dysfunction. Thus, it can be said that the Fontan circulation predisposes the single ventricle to
fail. Furthermore, efforts that target improvement in ventricular function as a solution to Fontan
disease may be misguided; it may be of greater benefit to provide cavopulmonary assistance.

Cavopulmonary assist specifically addresses the lack of a subpulmonary power source,
and will in turn normalize ventricular loading conditions and improve function. In the setting of
diastolic dysfunction but preserved systolic function, a modest increase in preload (~1-3 mmHg) will improve myocardial performance and cardiac output (opposite primary myocardial failure).

Transplantation is, in a sense, a definitive therapy for Fontan failure in that it reinstates a subpulmonary ventricle, but it does so at the cost of a different disease process. There aren’t sufficient donor organs available to transplant all single ventricle patients. Due to listing criteria, most Fontan patients won’t receive a donor organ until they have end-stage disease with advanced co-morbidities. Transplant survival of <50% at 10 years is arguably no better than the natural history of late Fontan attrition [22]. For these reasons, transplantation is not a comprehensive solution and represents end-stage therapy.

Fontan optimization falls short: Strategies to optimize Fontan hemodynamics have logically targeted the TCPC, where anatomic factors may result in significant power loss [23-25]. These have included various vena caval offset configurations, as well as a Fontan ‘Y’ conduit to reduce power loss in the inferior vena cava distribution [26]. Although these modifications have been applied clinically, they have not had significant impact. The maximal pressure gain that can be derived from passive flow optimization (~1-2 mmHg) does not fully correct the circulatory deficit and, as a result, does not meaningfully improve circulatory status. By comparison, consider normal biventricular physiology in which the right ventricle provides a cavopulmonary pressure step-up of ~6-8 mmHg. To make a clinically meaningful improvement in Fontan, it is necessary to add pressure energy at the TCPC by a similar amount.
Limitations of current MCS technology applied to Fontan: The application of existing MCS devices for Fontan failure is limited, highly variable, and associated with poor outcomes [27-30]. It has generally focused on the application of systemic support in end-stage Fontan disease due to the nature of existing technologies. Nearly all MCS technologies are intended for systemic support. However, depending on the circumstances, the device is likely mismatched to Fontan circulatory needs, i.e. left-sided support to address a right-sided deficit. Anecdotally, existing MCS appears to be better suited for ventricular “pump” failure in Fontan, rather than for Fontan failure secondary to lack of a subpulmonary ventricle. In the setting of preserved systolic function, systemic MCS in Fontan is redundant; it may needlessly congest the right-sided circulation where the circulatory bottleneck exists and exacerbate Fontan disease.

Existing MCS applied strategically to the right side of the Fontan circulation has been reported [31]; however, it is logistically impractical for several reasons: 1) the Fontan must be taken down to accommodate a single inflow, single outflow device; 2) a systemic MCS device is not optimized to the very low pressure rise desired for cavopulmonary assist, increasing risk for device failure; 3) the right-sided circulation is dependent on the device, making device dysfunction lethal; 4) device operational lifespan is limited, and therefore not a long-term solution. Given these considerations, the use of existing MCS technology in Fontan is limited to end-stage disease as bridge therapy, if it is used at all.

Technical considerations for long-term Fontan reversal: Dedicated right-sided circulatory support of the Fontan circulation presents a number of caveats that are uniquely challenging (Table 1). Technically, a cavopulmonary assist device is not a ventricular assist device (VAD)
because it provides support where no ventricle exists. It is intended to function as a low-input auxiliary right-sided pump to maintain low systemic venous pressure and preload to the systemic ventricle, identical to the essential function of the right ventricle in a biventricular circulation [32].

Although comparatively a weak pump, the inherent performance a von Karman viscous pump is ideal for the Fontan circulation. As a dynamic pump, it can provide consistent pressure rise over a wide range of cardiac output, making it responsive to physiologic demand. Low hydraulic efficiency also makes it less likely to generate suction, vein collapse, and cavitation, and tolerant to physiologic variation in systemic venous return. Similarly, it is unlikely to generate excessive downstream pressure, reducing risk for lung perfusion injury. The majority of existing MCS devices are displacement pumps that must vary RPM to vary flow rate, making them susceptible to suction, and unresponsive to physiologic demand thereby limiting exercise capacity.

The TCPC is the best-known geometry for passive cavopulmonary flow [23]. Thus, our strategy has been to retain the TCPC as a default flow path, and design an anatomically-specific pump to add pressure energy within it. A central diverting body at the TCPC intersection will split incoming flow toward each outlet, significantly reducing turbulent energy loss [33,34]. By rotating a central stabilizing body, suspended within the TCPC independent of the vessel walls, fluid pressure and velocity is increased, transforming it from a static flow diverter to a pump. We also anticipate that a permanently implanted device will eventually fail. In Fontan, a pump situated in the center of the flow path cannot impede flow (Figure 6). This is a critical safety consideration in young Fontan patients with decades of life expectancy ahead of them.
The ideal pressure rise for a cavopulmonary assist device is not yet clinically determined. We have shown in this study, and in other in vitro mock circulatory studies of a Fontan circulation that 6 mmHg pressure step-up will restore biventricular equivalency under normal physiologic conditions [7,8]. This pressure rise correlates with normal circulatory physiology. The presence of increased pulmonary vascular resistance in Fontan may impact the magnitude of pressure rise required. Extremely elevated pulmonary resistance is incompatible with late Fontan survival; therefore, we believe the need for a pressure increase significantly greater than ~15 mmHg is unlikely. Further, in the cavopulmonary circulation, the margin to apply an aggressive pressure step-up is limited by baseline systemic venous pressure. For example, the application of a 20 mmHg pressure step-up in the TCPC in which baseline systemic venous pressure is 16 mmHg will induce negative (-4 mmHg) systemic venous pressure, leading to suction collapse and flow disruption.

In general, MCS devices are not placed in line or in series with the circulation, but are rather placed in parallel (i.e. paracardiac) due to risk of device obstruction of the native flow path. This concern is amplified for a Fontan pump situated in the lowest pressure segment of the circulation, where seemingly trivial obstruction can induce hemodynamic instability. An implantable Fontan pump must have no potential for mechanical obstruction in the event of dysfunction.

Emerging technologies: Single functional ventricle is a final frontier for MCS therapy, and concepts for dedicated Fontan circulatory support are currently evolving [8,35]. To date, nearly all have focused on modification of existing technology (unidirectional axial or centrifugal VADs) to operate in the low-pressure Fontan environment. Although these may suffice as bridge
therapy, they lack the technologic rigor required for safe and durable long-term support. They are incapable of supporting flow in all 4 limbs of the cavopulmonary connection, and will induce back-pressure elevation in the opposing systemic venous territory. They are capable of mechanical obstruction, a prohibitive issue when the right-sided circulation is dependent on the device. Due to motor configuration, they may be bulky and not fit within the confines of the TCPC. Lastly, if the Fontan pathway is modified to accommodate a unidirectional flow device, the resulting pathway will not support passive cavopulmonary flow in the event of device dysfunction. With respect to safety and durability, a Fontan pump that offers multi-directional flow, in situ anatomic placement, and a contingency plan for failure is preferable.

Clinical implications: Cavopulmonary assist reframes single ventricle palliation from a problem with no solution to a problem with an obvious solution. The challenge lies in creating a technology that can safely and durably implement it. If successfully translated, it would represent a first-ever targeted therapy for Fontan failure. It would also permit biventricular equivalency to be maintained in a univentricular circulation prior to clinical failure, preempting Fontan disease. For Fontan patients who have few options to alter their inevitable disease progression, this concept has exciting potential to shift the paradigm for single ventricle therapy from palliation to cure.

The clinical vision for a Fontan-specific cavopulmonary assist device is much different from current MCS strategy. Rather than reserving MCS for end-stage salvage, it may be better to apply it preemptively for biventricular health maintenance. Thus, the device may not necessarily be limited to advanced Fontan failure, and may be considered earlier. For Fontan patients, the implications are starkly different: a lifetime of chronic disease progression with systemic MCS
as end-stage therapy versus cavopulmonary assist to enable long-term biventricular health and wellness, potentially for decades.

**Limitations:** This report presents early stage electromechanical feasibility for an implantable cavopulmonary assist device concept. Such a therapy has never been implemented clinically; therefore, discussion of clinical application and duration of support is theoretical. Substantial technologic development remains to be performed, and significant obstacles may arise in future design iteration. Thrombogenicity performance remains to be determined, and device durability is unknown. The power source has not yet been determined, although it is envisioned to be powered wirelessly. Until a device is fully developed, regulatory approved, and enters clinical trial, the translational impact of long-term Fontan reversal will remain speculative.

**Conclusions:** After Fontan repair of single functional ventricle, patients are trapped for the remainder of their lives in a cycle of chronic circulatory inefficiency. Despite dramatic improvement in surgical technique, perioperative care, and long-term surveillance, late Fontan failure and attrition remains an intractable problem for which there is no primary or preventive therapy. An early stage implantable cavopulmonary assist device shows promising potential to reverse the Fontan paradox and preempt Fontan-associated disease as a curative therapy. It represents a significant opportunity to fundamentally shift the paradigm for single ventricle care to one based on biventricular health.
Table 1. Desirable technical features for a long-term cavopulmonary assist device:

1. Simplicity. A single pump, with one moving part, that serves as a low-input primer for the systemic ventricle.
2. Multi-directional flow. Flow augmentation in all 4 distributions of the TCPC (SVC/IVC inflow, RPA/LPA outflow).
3. Low pressure performance. Low pressure (~6-8 mmHg), high volume flow augmentation, similar to normal right ventricular hemodynamics.
4. Wide performance range. Consistent pressure rise over a wide range of cardiac output and variable physiologic demand, independent of pump speed.
5. Unobstructive. An in-line pump in the Fontan venous pathway must account for pump failure, with minimal or no obstruction risk as a failure default.
6. TCPC as the preferred default flowpath. The failed (0 RPM) condition should ideally optimize passive TCPC flow as an unsupported Fontan circulation.
7. Permissive preload and afterload performance. Low hydraulic efficiency to reduce risk of excessive negative upstream pressure (suction, vein collapse, cavitation), and excessive positive downstream pressure (perfusion lung injury). It will also permit tolerance to the natural inflow instabilities that occur in systemic venous return (supine vs upright posture, cough, Valsalva).
8. Mixing. Symmetric hepatic factor distribution to prevent pulmonary arteriovenous malformation (Video 1).
9. No barrier to recirculation. To reduce thrombogenicity and obstruction risk.

10. Small size. In situ implantation to avoid physical encroachment on adjacent structures (e.g. aorta, common atrium, pulmonary veins).

References:


Figure Legend:

**Figure 1:** Outrunner brushless DC motor: A. Traditional motor design with inner rotating component. B. Outrunner motor design with outer rotating component and stationary central component.

**Figure 2:** Implantable cavopulmonary assist device concept. A central impeller contains the motor (impeller support shaft and struts not shown). The external housing is a hard case, thin-walled shell that has no component bulk and serves only to direct Fontan flow. The wide gap between the impeller and the housing must support passive TCPC flow in the event of device dysfunction.

**Figure 3:** Advanced prototype, adult scale. A: Design schematic showing housing with mounted impeller. Wiring travels from the stator via the central shaft and strut to exit the housing. The only moving component is the rotor. A secondary flow path allows for inflow at each axial end, with outflow at the equatorial gap (Video 2). B: Design schematic showing patulous channel to prevent obstruction in the event of rotational failure. Rotor surface shows 0.7 mm height curved surface vanes. C: Assembled prototype as tested and demonstrated in video.

**Figure 4:** Hydraulic performance. H-Q curve demonstrates pressure and flow performance.

**Figure 5:** In vitro mock loop parameter showing reversal of the failing Fontan circulation with cavopulmonary assist. Fontan baseline includes pump in TCPC in stalled condition.
VIP, viscous impeller pump; HR, heart rate; SV, stroke volume; CO, cardiac output; CO%
increase, percent increase from baseline Fontan cardiac output; VCP, vena caval pressure;
PAP, pulmonary artery pressure; CPPH, cavopulmonary pressure head (pump ΔP) = vena
caval pressure – pulmonary artery pressure; AoP, aortic pressure.

Figure 6: Pump implantation (conceptual). The cavopulmonary assist device is implanted in
situ in the TCPC and serves to modestly augment cavopulmonary flow. The device is
compatible with either lateral tunnel and extracardiac conduit Fontan construction. Graft
extensions allow for suturing, with a total of 4 anastomoses. Grafts can be extended or
tailored to address anatomic issues. Two pumps are depicted. The systemic ventricle
performs the majority work load. It is served by a low-input cavopulmonary assist device to
normalize preload to the systemic ventricle, in addition to reducing systemic venous pressure.
(TCPC: total cavopulmonary connection; SVC: superior vena cava; IVC: inferior vena cava;
PA: pulmonary artery)

Video legend:

Video 1: Primary (external) flow path. Open static circuit; flow is induced by the pump.
Yellow and blue contrast represent systemic venous inflow. The green outflow represents left
and right pulmonary arterial flow. Mixing indicates symmetric hepatic factor distribution.

Video 2: Secondary (internal) flow path. Open static circuit; flow is induced by the pump.
Contrast injection flows selectively through the internal flow path, with dispersion of outflow at
the equatorial midline. Internal path flow is essential to pump function for heat dissipation, bearing flushing, and to reduce risk of recirculation, stasis, and thrombosis.
<table>
<thead>
<tr>
<th>Case</th>
<th>VIP Speed (RPM)</th>
<th>HR (beats/min)</th>
<th>CO (L/min)</th>
<th>CO % increase</th>
<th>VCP (mmHg)</th>
<th>PAP (mmHg)</th>
<th>CPPH (mmHg)</th>
<th>AoP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fontan Baseline</td>
<td>0</td>
<td>75</td>
<td>3.9</td>
<td>-</td>
<td>17.5</td>
<td>14.7</td>
<td>2.8</td>
<td>98.3/57.4</td>
</tr>
<tr>
<td>Fontan + VIP</td>
<td>3000</td>
<td>75</td>
<td>4.1</td>
<td>5.1%</td>
<td>17</td>
<td>16.6</td>
<td>0.4</td>
<td>102.3/60.5</td>
</tr>
<tr>
<td>Fontan + VIP</td>
<td>4000</td>
<td>75</td>
<td>4.3</td>
<td>10.3%</td>
<td>16.8</td>
<td>18</td>
<td>-1.2</td>
<td>111.9/66.7</td>
</tr>
<tr>
<td>Fontan + VIP</td>
<td>5000</td>
<td>75</td>
<td>4.4</td>
<td>12.8%</td>
<td>16.1</td>
<td>20.8</td>
<td>-4.8</td>
<td>113.6/69.0</td>
</tr>
</tbody>
</table>
Cavopulmonary assist device for long-term Fontan reversal

Mark Rodefeld MD, Guruprasad Giridharan, Alison Marsden, Travis Jonas, Michael Neary