Parallel Circulation

Karim Rafaat, M.D.
The basic issue with “parallel” circulation is achieving the proper balance between the pulmonary and systemic circulations.

Qp:Qs

Today, we focus on how to do it prior to definitive surgical correction.

Before that, however, we have to understand some basics:

- How to calculate Qp and Qs from a cath diagram
- How to calculate Qp:Qs ratio
I have to apologize for the speed with which I am going to go over the different cardiac lesions.

My focus will be only how they lay in the spectrum of complete mixing lesions, and so, how we must deal with the problems they present with relation to their systemic and pulmonary circulations.
An amount, I, of an indicator, can be added to an unknown fluid quantity, Q. The concentration before the indicator is added is $C_i$, and after, $C_f$. 
\[ Q \times C_f - Q \times C_i = I \]

the amount of added indicator is equal to the amount of stuff in the fluid after minus the stuff in the fluid before

\[ Q = \frac{I}{C_f - C_i} \]
Upstream sample, Indicator concentration

CI

Indicator infusion (constant over time)

I

Steady state flow, Q

Cf

Downstream sample, Indicator concentration

This is the basic form of the Fick Principle.

\[ Q = \frac{I}{C_f - C_i} \]
Fick principle

Used to calculate flow when it cannot be measured directly

We can measure it indirectly by way of an indicator

There are a few ways to use this concept, depending on the indicators that are used
Thermodilution Method

The indicator is temperature

Bolus injected in RA, temp measured by thermistor in PA (usually)

Degree of cooling is inversely proportional to the magnitude of flow and directly proportional to amount of “cold”
As an aside…

- The sharper the curve
- the quicker the return to normal blood temp
- so the less and quicker influence of the set amount of “cold”
  - Cf – Ci (integrated over time) is smaller….
- therefore, the higher the cardiac output.
When oxygen is the indicator, the Fick Principle is more directly applicable.

The rate of change of the indicator (I) is Oxygen Consumption.

- Can be directly measured (impossible practically)
- Commonly taken from a table

The concentration is the Oxygen Content.
Then, to calculate Qp or Qs, you have to pick your points around which you are going to measure the “change in concentration”
So, for

- Qp: pulmonary veins – pulmonary arteries
- Qs: aorta – mixed venous
Oxygen Content = 
(13.6 \times \text{Hgb} \times \% \text{sat}) + (0.003 \times \text{pO2})

1.36 is O2 carrying capacity of hgb in mL/g
Hgb is in g/dL
O2 content, though, is measured in ml/L
So, you use 13.6 in the equation above…
Qp = Oxygen Consumption
\[ \frac{\text{C}_{\text{pulmonary vein}} \text{O}_2 - \text{C}_{\text{pulmonary artery}} \text{O}_2}{\text{C}} \]

Qs = Oxygen Consumption
\[ \frac{\text{C}_{\text{aorta}} \text{O}_2 - \text{C}_{\text{mixed venous}} \text{O}_2}{\text{C}} \]
DORV
Let’s calculate some ratios, then….

\[ Q_p = \frac{\text{Oxygen Consumption}}{\text{C}_{\text{pulmonary vein}} O_2 - \text{C}_{\text{pulmonary artery}} O_2} \]

\[ = \frac{172}{(13.6 \times 16 \times 0.94) - (13.6 \times 16 \times 0.84)} \]

\[ = \frac{172}{204 - 183} \]

\[ = 7.9 \text{ L/min/m}^2 \]
Qs = Oxygen Consumption
    \[ \frac{C_{\text{aorta}} O2 - C_{\text{mixed venous}} O2}{172} \]
    \[ \frac{(13.6 \times 16 \times 0.72) - (13.6 \times 16 \times 0.49)}{156 - 106} \]
    = 3.4 L/min/m²
So, Qp:Qs
7.9/3.4 = 2.4

Math trick.....
Why is this important?

✅ In lesions with parallel circulation, the total CO of the usually single ventricle is shared between pulmonary and systemic circulations

✅ The *ratio* of Qp:Qs describes the relative amount of pulmonary and systemic blood flow

✅ The absolute value, however, is a representation of total cardiac output
Why is this important?

Physiology with a high Qp:Qs brings with it a relatively low systemic oxygen delivery.

Low systemic DO2 leads to tissue hypoxia, anaerobic metabolism, and eventual end organ damage.
The goal, then, of our management, is the optimization of systemic oxygen delivery (DO2) 

Not maximize SaO2

This requires the maintenance of cardiac inotropy while balancing Qp:Qs.
Why is Qp:Qs important?

- This is a theoretical graph of systemic O2 availability versus Qp:Qs, at different CO’s.
- The dashed line represents the max O2 delivery.

Fig. 4. Systemic oxygen availability (mL O2/min) as a function of pulmonary/systemic blood flow ratio (Qp/Qs) at different levels of cardiac output. Pulmonary venous oxyhemoglobin saturation (SpvO2) is assumed to be 96%, and oxygen consumption (VO2) is assumed to be 18 mL O2/min. (From Bamea O, Austin EH, Richman B, et al. Balancing the circulation: theoretic optimization of pulmonary/systemic flow ratio in hypoplastic left heart syndrome. J Am Coll Cardiol 1994;24:1376–81; with permission.)
Why is this important?

- Max O2 delivery occurs at a Qp:Qs between 0.5 and 1
  - Not at 2.3…
- Increasing CO, increases systemic O2 delivery
  - With more of a difference made at Qp:Qs ratios less than 1

Fig. 4. Systemic oxygen availability (mL O2/min) as a function of pulmonary/systemic blood flow ratio (Qp/Qs) at different levels of cardiac output. Pulmonary venous oxyhemoglobin saturation (SpvO2) is assumed to be 96%, and oxygen consumption (VO2) is assumed to be 18 mL O2/min. (From Bamea O, Austin EH, Richman B, et al. Balancing the circulation: theoretic optimization of pulmonary/systemic flow ratio in hypoplastic left heart syndrome. J Am Coll Cardiol 1994;24:1376–81; with permission.)
The slope of each curve is steepest around point of maximal O2 delivery. This suggests small changes in Qp:Qs can be associated with large changes in oxygen delivery.
With complete mixing lesions, the ventricular output is the **SUM** of Qp and Qs

Cause there’s, effectively, one ventricle

The higher the ratio, the higher the demand on the heart

So, a Qp:Qs of 2.3 means that the heart is pumping about 3 “cardiac outputs”

It must maintain such a high output in an attempt to allow for acceptable systemic oxygen delivery
What does this mean?

Ventricular wall tension and myocardial oxygen demand are increased in the dilated, volume overloaded ventricle

- Leads to myocardial dysfunction and AV valve regurgitation

Prolonged increased pulmonary volume will lead to pulmonary vascular bed remodeling

- can lead to increased pulmonary vascular resistance, which makes single ventricle surgical repair impossible
When must we think of these things?

A wide variety of lesions, usually associated with atresia of an AV valve, have the common physiology of complete mixing.

While most of them are commonly dealt with in the NICU, there are a few that we will routinely see.
The most common is Hypoplastic Left Heart Syndrome

- 1. PFO
- 2. hypoplastic aorta
- 3. Patent PDA
- 4. aortic atresia
- 5. Hypoplastic left ventricle

Mixing occurs via a patent PDA
We see this lesion usually after the stage 1 Norwood operation:

- BTS supplies pulmonary flow
- Atrial septectomy
- Pulmonary trunk disconnected from MPA
- MPA and Aorta anastomosed to form a neo-aorta
Double Outlet Right Ventricle
- Both the aorta and pulmonary artery arise from the RV
- Accompanied by a VSD
D-TGA with VSD

- Aorta and Pulmonary Artery arise from the wrong ventricle
- Mixing occurs through the VSD
CAVC

**Complete AV Canal**
- atrial septal defect
- abnormal tricuspid valve
- abnormal mitral valve
- ventricular septal defect
Truncus Arteriosus

- single large arterial trunk arises from both ventricles,
- large VSD just below the trunk
Tetralogy of Fallot

- Ventricular septal defect (VSD)
- Pulmonary (or right ventricular outflow tract) obstruction
- Overriding aorta.
- Right ventricular hypertrophy
So, what determines our ratio?

**OHM’S LAW:**

\[ V = I \times R \]

- \( V \) is voltage, or, another way, driving force
- \( V = \) Pressure difference
- \( I \) is current or flow
- \( I = CO \)
- \( R \) is, in both cases, resistance
Rearranged:

\[ I = \frac{V}{R} \]

or

\[ Q = \frac{\tilde{E}P}{R} \]

\( \tilde{E}P \) can be affected by way of inotropy, but this has little effect on the ratio of pulmonary to systemic flow

The resistances of the two circuits are separate, and can thus be manipulated in a way that can effect flow differentially
Resistance to Pulmonary flow is determined by:

- Valvar or subvalvar pulmonary stenosis
- Pulmonary arteriolar resistance
- Pulmonary venous and left atrial pressure

In part determined by:
- amount of pulmonary blood flow
- restriction of outflow through left atrioventricular valve
Resistance to systemic flow determined by:

- Presence of anatomic obstructive lesions
  - Aortic valve stenosis
  - Arch hypoplasia or coarctation
  - Subaortic obstruction
- Systemic arteriolar resistance
Since the most easily alterable aspects are the resistances of the respective vascular beds

The problem of balancing the flows can be somewhat simplified to balancing the ratio of PVR:SVR

Useful, as the majority of therapies available to us that affect flow differentially do so by way of manipulation of the resistance of the respective vascular beds
Remember, we are going for a Qp:Qs that will optimize DO2.

That occurs most effectively between a Qp:Qs of 0.5 and 1.
Pulmonary Vascular Resistance

Qp can be effectively decreased by increasing PVR
- This is the issue in most lesions we deal with, with the exception of unrepaired Tet

This can be accomplished by PEEP
- Has to be in excess of that required to maintain FRC to increase PVR
Increasing PCO2

- Mild hypercapnia will increase pulmonary vascular resistance
- This can be done by way of hypoventilation in the sedated, intubated patient, or, one can bleed in 2%-5% CO2 into the ventilator circuit

Decrease pH

- Mostly, ensure that one is not alkalotic, which causes a decrease in PVR
Decrease FiO2

This increases PVR by way of hypoxic pulmonary vasoconstriction

FiO2 can be decreased to less than 21% by adding nitrogen to the inspired mixture
Systemic Vascular Resistance

Qs can be effectively increased by decreasing SVR
This can be accomplished by
- Sedation
  - Decrease sympathetic output
- Paralysis
  - See above.....
Pharmacologic manipulation

Nitroprusside – NO donor, causes both venous and arteriolar vasodilation, lowers SVR

Milrinone – increases intracellular cAMP, causing vasodilation, lowers SVR

Alpha adrenergic receptor antagonists

When the SVR is low, increases in CO will further increase DO2
How do we assess the efficacy of our interventions?
Remember, we want a Qp:Qs of 0.5 to 1.0
We can use the Fick principle to make quick estimates...

Fig. 4. Systemic oxygen availability (mLO₂/min) as a function of pulmonary/systemic blood flow ratio (Qₚ/Qₛ) at different levels of cardiac output. Pulmonary venous oxyhemoglobin saturation (SpvO₂) is assumed to be 96%, and oxygen consumption (VO₂) is assumed to be 18 mLO₂/min. (From Bamea O, Austin EH, Richman B, et al. Balancing the circulation: theoretic optimization of pulmonary/systemic flow ratio in hypoplastic left heart syndrome. J Am Coll Cardiol 1994;24:1376–81; with permission.)
We can make some assumptions:

- In single ventricles, aortic and pulmonary artery saturations are the same
- Lungs are usually healthy, so pulmonary vein sats are assumed to be 96%
- Assume a normal systemic A-VO2 difference of 25%

\[
Qp:Qs = \frac{\text{Sat aorta} - \text{Sat mixed venous}}{\text{Sat pulmonary vein} - \text{Sat pulmonary artery}}
\]
The equation then becomes:

\[
\frac{Q_p}{Q_s} = \frac{25}{96 - \text{SaO}_2}
\]

A \( \frac{Q_p}{Q_s} \) of 1, then can be assumed to occur at a sat of around 70%.

There are problems with this, however...
The first is that this is an assumption...

If the systemic A-VO2 difference is any higher than normal (indicating poor DO2), measured Qp:Qs will be much higher for a given SaO2.
A better idea would be to follow those variables that are more closely related to a match of systemic oxygen supply and demand:

- Base deficit
- Lactate levels

These are indicators of anaerobic metabolism, and so, increases in either of them indicate that DO2 is inadequate, and that, presuming adequate CO, Qp:Qs is too high.
Mixed venous oxygen saturation can be used as a marker.

Hoffmann et al showed that mixed venous oxygen saturations in neonates following a Norwood stage 1 operation closely mirrored tissue oxygen levels.

SvO2 of 50-55% led to a less than 5% risk of anaerobic metabolism...
Their main conclusion, however, was that, after linear regression of those factors they monitored pre-op (MAP, SvO2, BE, Qp, Qs, Hgb etc. etc.) the odds ratio of anaerobic metabolism at an SvO2 of less than 30% is 8.

This was NOT apparent when MAP, SaO2 and base deficit are examined, even in combination.

Svo2, therefore, is deemed to be an essential part in monitoring patients in whom a Qp:Qs balance is essential.

### Table 4. Anaerobic Risk at SvO2 Strata

<table>
<thead>
<tr>
<th>SvO2</th>
<th>n</th>
<th>BE (mean)</th>
<th>BElo (risk)</th>
<th>ΔBElo (risk)</th>
<th>Anaerobic Risk Unadjusted</th>
<th>Anaerobic Risk Adjusted</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 30%</td>
<td>42</td>
<td>-0.82 ± 0.71</td>
<td>0.238 ± 0.36</td>
<td>0.071 ± 0.60</td>
<td>0.262 ± 0.35</td>
<td>0.294 ± 0.39</td>
</tr>
<tr>
<td>31–40%</td>
<td>154</td>
<td>1.82 ± 0.28</td>
<td>0.039 ± 0.42</td>
<td>0.019 ± 0.58</td>
<td>0.052 ± 0.36</td>
<td>0.048 ± 0.38</td>
</tr>
<tr>
<td>41–50%</td>
<td>469</td>
<td>2.11 ± 0.18</td>
<td>0.075 ± 0.18</td>
<td>0.021 ± 0.32</td>
<td>0.092 ± 0.16</td>
<td>0.063 ± 0.19</td>
</tr>
<tr>
<td>51–60%</td>
<td>828</td>
<td>3.91 ± 0.13</td>
<td>0.023 ± 0.23</td>
<td>0.026 ± 0.28</td>
<td>0.034 ± 0.19</td>
<td>0.024 ± 0.21</td>
</tr>
<tr>
<td>&gt; 60%</td>
<td>581</td>
<td>4.15 ± 0.12</td>
<td>0.015 ± 0.34</td>
<td>0.002 ± 1.00</td>
<td>0.017 ± 0.32</td>
<td>0.014 ± 0.33</td>
</tr>
</tbody>
</table>
A combination of SaO2, SvO2 and base excess measurement, then, seems to be the best approach

So what is the best strategy of management?
Steih et al, in a retrospective evaluation of 72 patients with HLHS, found that in hospital mortality decreased from 65% to 13% with a shift in preoperative management from ventilation to increase PVR, to pharmacologic management to decrease SVR.

Preoperative organ dysfunction was higher in those patients who were ventilated versus those who received afterload reduction in the form of phenoxybenzamine.
Chang et al, Pediatric Cardiac Intensive Care, LWW, 1998


