Hemodynamics in Different VSD's

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Ventricular septal defects (VSDs), were first described by Dalrymple in 1847 and account for ~20% of congenital cardiovascular malformations....10% of those in adults

Prevalence estimated at 1.17 /1000 live births

With one exception (subarterial defect), VSDs have no sex preference

They can be associated with ASD’s (35%), PDA’s (22%), RAA (13%) and less often, PS

Multiple VSD’s (4% to 18% of isolated defects) are more prevalent in association with DORV and TOF
Various types of VSD's

- Doubly committed subarterial defect
- Outlet muscular
- Perimembranous
- Muscular
- Inlet/inlet muscular
The direction and volume of an isolated VSD shunt is determined primarily by:

- the size of the defect and the ratio of PVR to SVR
- rather than its location

Associated complications affecting natural history are impacted by location e.g., doubly committed subarterial defect...aortic leaflet prolapse inlet defects associated with LAVV clefts...valve regurgitation
The association between aortic regurgitation and VSD was first reported in 1921 and is more common in young men.

The regurgitation is an acquired lesion seen more with doubly committed subarterial defects than with perimembranous defects.

It results from deficiency or hypoplasia of the outlet septum that leads to abnormal apposition in diastole and prolapse of the poorly supported non- or right coronary cusp through the VSD into the RV.

This results in distortion of the aortic valve and progressive aortic regurgitation.
In the absence of pulmonary stenosis or pulmonary hypertension, the left-to-right shunt results in volume overload of the LA, both ventricles and pulmonary arteries. The volume of the shunt dictates the clinical presentation and ultimately the natural history for the child.
Effect of Size

The size of the defect is crucial

Below a critical size, the defect itself presents a resistance to flow which controls the magnitude .....but not the direction of the shunt

Above this critical size, there is no appreciable resistance to flow

Both magnitude and direction of flow are determined by the level of pulmonary vascular resistance
The effect of size on hemodynamics

When there is a left-to-right shunt and no other associated anomalies (eg., PS), there is equalization of systolic pressures in the 2 ventricles: the defect is unrestrictive

Since ventricles do not contract exactly simultaneously there is always some inequality in the ventricular pressures:

○ throughout the greater part of systole, the pressure difference allows left-to-right shunting

○ during isovolumic relaxation, right-to-left shunting occurs, which is then cleared by the subsequent systole

[Graph showing hemodynamic pressures with annotations for LV, RV, and LA]
What is this critical size anatomically:

A defect diameter of ~1 cm/m² appears to be the hemodynamic dividing line corresponding to an orifice area of ~0.8 cm² /m²

Defects smaller than this are **restrictive** and produced minor or no haemodynamic changes

**Restrictive defects** being about 2/5<sup>th</sup> or 40% of the aortic valve annulus
Size matters:

In a restrictive defect RV and PA systolic pressures < in the LV and Ao

There is a continuum of sizes, where some restrictive defects may allow some elevation of right-sided pressures: to small defects in which the right-sided pressures are normal
Effects of Pulmonary Vascular Resistance: Key points

- The effect of PVR on flow through a restrictive defects is always 2nd to the size of the hole.

- In an unrestricted defect, the PVR (and to a lesser extent SVR) is the controlling factor.

When the PVR is low, the flow through the defect and the flow of blood to the lungs will be high.

Such a high flow is not present at birth.

It takes time for the PVR to fall from the high intrauterine to the normal postnatal levels.
High flow to the lungs $\rightarrow$ LAP $\rightarrow$ cause pulmonary vasoconstriction

Vasoconstriction $\rightarrow$ vascular muscular hypertrophy

When the $\downarrow$ in PVR is delayed $\rightarrow$ the maximal haemodynamic effects of an unrestricted defect may not be reached for some weeks

The time course and extent of the fall in PVR are all variable usually achieved by 6 weeks of age.
The PVR is low enough at this time period to allow a very high PBF, but the fall in PVR may be limited and little flow occurs through the defect.

Such patients may escape detection until the effects of severe PVD have become apparent.
Once the PVR has fallen, it may increase again with the development of the pathological changes of PVD.

This usually occurs only in children where the PAP was high from birth and almost entirely confined to those with an unrestrictive defect.
The level of resistance determines the direction of flow...as the resistance rises.....flow across the defect decreases

When PVR is > SVR, flow changes from left-to-right to right-to-left

Secondary effects occur:

- enlargement of the RV and MPA
- dilation of the PV annulus leading to PI
Effect of Obstruction of the Pulmonary Outflow Tract

If there is co-existing obstruction within the RVOT (valvar or subvalvar), the effect is similar to an elevation of the PVR

The flow through an unrestricted defect and to the lungs, will be limited in proportion to the severity of the obstruction

In restrictive defects, obstruction to the RVOT will result in elevation of RVP also in proportion to the severity of obstruction

The RVP in extreme cases, may be > LV

There will be a reversal in the direction of the shunt in all cases where RVP > LVP
Response of the Heart to Flow between the Ventricles

The cardiac effects depend on the magnitude of PBF

With florid PBF... usually in the setting of **unrestrictive defects**:
- LA volume
- LVED volumes are increased
- LV muscle mass is increased

This results in a marked increase LV work

This leads to LV hypertrophy as a compensatory mechanism, but in **restrictive defects** little RVH
Response of the Heart to Flow between the Ventricles

With extreme ↑ pulmonary flow, there is also an increase in RV size through the following mechanism:

↑ RVP leads ↑ RV work → RVH → development of subvalvar PS

With ↑ PVR or development of RVOTO decreased LV work because of ↓ in left-to-right shunting and PBF

The elevation of RVP leads to RVH, which dominates the picture
In Summary

The haemodynamic affects form a VSD are primarily dependent on:

- the size of the defect
- the PVR
- age of the patient

The natural history is dynamic, and changes in morphology (size) occur with time and determine the need for intervention.
Cám ơn
Thank you
Physiologic Assessment:  
Shunts and Pulmonary Vascular Resistance
Shunts

When there is a communication between the 2 sides of the heart, or GA

- Left to Right
- Right to Left
- Bidirectional
Shunts

When there is a communication between the 2 sides of the heart, or GA

- Left to Right
- Right to Left
- Bidirectional
- QP:QS

Provides estimate of PBF, severity of hemodynamic disturbance, & easy to determine

........................arterial/LV/PV; PA; VC/RA saturations
Shunts: $QP:QS$

Requirements:

- Room air or <30% $O_2$
- Steady-state
- Sample representative of chamber/vessel
- Not contaminated by distal chamber (regurg AV valve)
Shunts: \textbf{QP:QS}

**Practical points**

- If breathing $>30\%$ $O_2$, then have to calculate dissolved $O_2$
- Do not allow sample to equilibrate with room air
- Remember: oximeters are inaccurate if Hgb $>200$ g/l
  
  .................have to use ABG's
- Sample locations:
  - SVC...above azygous
  - RA.....mid lateral wall
  - IVC.....above diaphragm
Shunts: QP:QS

Based on Fick principle

Flow (Q) = \frac{V_{O_2}}{AV \ O_2 \ content \ difference}
Shunts: \( QP:QS \)

Based on Fick principle

\[
\text{Flow (Q)} = \frac{V_{O_2}}{AV \ O_2 \ content \ difference}
\]

To calculate \( O_2 \) content (ml/l):

- Determine \( O_2 \) capacity = \( Hgb \) (g/l) \( \times \) 1.36 (or 1.39) then multiply by the sample saturation
- Example: \( Hgb = 140 \ g/l \)
  
  \( \text{Sat} = 70\% \)
  
  \( 140 \times 1.36 = 190 \text{ ml/l} \ldots \ldots \ldots O_2 \) capacity
  
  \( 190 \times 70\% = 133 \text{ ml/l} \ldots \ldots \ldots O_2 \) content
Shunts: $\frac{QP}{QS}$

As a ratio, content & $V_{O_2}$ cancel

$$\frac{QP}{QS} = \frac{Ao\ sat - MV\ sat}{PV\ sat - PA\ sat}$$
Shunts: $QP:QS$

As a flow ratio, content & $V_{O_2}$ cancel

$$QP:QS = \frac{Ao \text{ sat} - MV \text{ sat}}{PV \text{ sat} - PA \text{ sat}}$$

Obtaining PA & Ao saturations do not present problems
For PV saturation...can assume 98% (no known lung disease)
......or use LV or LA if no right to left shunt
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\( MV \): most distal chamber/site where there is no L to R shunt
Shunts: QP:QS

In general practice: SVC used

.........but true valve somewhere between SVC & IVC values

\[
MV \text{ sat} = \frac{3 \times \text{SVC sat} + 1 \times \text{IVC sat}}{4}
\]

Miller et al BHJ 1974
Shunts: \( QP:QS \)

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MV_{\text{sat}} = \text{SVC sat} - \frac{\text{SVC sat} - \text{IVC sat}}{4}
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$$MV \text{ sat} = SVC \text{ sat} - \frac{SVC \text{ sat} - IVC \text{ sat}}{4}$$

For example: SVC 78%; IVC 70%............

$$MV \text{ sat} = 78 - 70 = 8; \frac{8}{4} = 2; 78 - 2 = 76\%$$
Shunts: \( Q_P:Q_S \)

Limitations:

- absence of steady state (arrhythmia, prolonged collection of samples)
- small shunts poorly detected
- high flow states where MV sat is high decrease shunt detection
MV = 70 - (70-80.5)/4
   = 72.6%

QP:QS = 96.5 - 72.6
      = 3.73:1
Pulmonary blood flow & pulmonary vascular resistance

- In the absence of a shunt PBF=SBF=CO
- Measured by Fick or thermodilution (no shunt)
- Fick - need an estimate of $V_{O_2}$
  charts estimate $V_{O_2}$ based on sex/HR/age
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Example

\[
V_{O_2} = 250 \text{ ml O}_2/\text{min} \\
C_{PA} = 150 \text{ ml O}_2/\text{l} \\
C_{Ao} = 200 \text{ ml O}_2/\text{l}
\]

\[
\frac{250 \text{ ml O}_2/\text{min}}{(200-150) \text{ ml O}_2/\text{l}} = 5 \text{ l/min}
\]
In practice, 'absolute' values of PBF or SBF are less of value than indexed values:

Using indexed $V_O_2 = \text{ml/min/m}^2$

BSA = 2 m$^2$  $V_O_2 = 240 \text{ ml/min}...... V_O_2 = 120 \text{ ml/min/m}^2$
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**infants:**  130 (<3mon) - 170 ml/min/m$^2$

**2-5 years:**  150 - 200 ml/min/m$^2$

**adolescents:**  120 - 180 ml/min/m$^2$

**adult females:**  100 ml/min/m$^2$

**adult males:**  110 - 120 ml/min/m$^2$
Despite deficiencies in assumed $O_2$

- widely used
- probably ‘adequate’ for most situations
- do calculations at upper/lower ranges

Common errors

- assumed $O_2$ notoriously unreliable
- have to add dissolved $O_2$ if breathing enriched $O_2$
  (1000 ml plasma takes up 0.03 ml $O_2$ for each 1 mm Hg $O_2$)
Example

Arterial oxygen content?

arterial saturation = 96%
arterial $P_{O_2} = 81.3$ mm Hg
Hgb = 128 g/l

$O_2$ capacity = $1.39 \times 128 = 177.9$ ml/l
arterial content = $96\% \times 177.9$ ml/l = 170.8 ml/l

Dissolved $O_2$ content =

$P_AO_2 \times 0.03 = 81.3 \times 0.03 = 2.4$ ml/l

$Ao$ content = 170.8 + 2.4 = 173.2$ ml/l
PBF & PVR

**Pulmonary blood flow**

\[
PBF = \frac{V_{O_2}}{\text{Pulmonary AV } O_2 \text{ content difference}}
\]

**Pulmonary vascular resistance**

- **Total pulmonary resistance** (not used anymore)
  \[
  \text{mean PAP} \frac{\text{mean PAP}}{\text{PBF}}
  \]

- **Pulmonary vascular resistance**
  \[
  \text{mean PAP} - \text{mean LAP} \ (\text{or mean PCWP}) \frac{\text{mean PAP} - \text{mean LAP}}{\text{PBF}}
  \]

Wood units (mm Hg/l/min)
Pulmonary vascular resistance

- If PBF indexed then PVR is also indexed as Wood units.m$^2$
- If absolute values used, then smaller patients will have a higher resistance:

Example

BSA 0.5 m$^2$; QP 2 l/min; mPAP 20 mm Hg; mLAP 8 mm Hg

\[
PVR = \frac{(20-8)}{2} = 6 \text{ Wood units,}\]

\[
PVRI = \left(\frac{(20-8)}{2}\right) \times 0.5 = 3 \text{ Wood units.m}^2
\]
Pulmonary vascular reactivity

Assessment important if PVRI elevated in room air
Depends on patient's age
PVRI > 6 Wood units m$^2$ cause for concern
Pulmonary vascular reactivity

Assessment important if PVRI elevated in room air
Depends on patients age
PVRI > 6 Wood units.m² cause for concern

Recognize situations that can increase PVR:

hypoxia, hypercapnia, erythrocytosis, increased sympathetic tone
pulmonary emboli, precapillary pulm edema, lung compression (pleural effusion), mechanical ventilation, positive intrathoracic pressure
Pulmonary vascular reactivity

Assess effect of a pulmonary vasodilator, $O_2/NO$

Common errors:

- Hypoventilation/acidosis producing pulmonary vasoconstriction
- Failure to calculate dissolved $O_2$ otherwise underestimate AV $O_2 \Delta$; overestimate PBF & underestimate PVR
- Assumption that no fall in PAP means no fall in PVR
VSD

Room air

- Hgb = 100 g/l; \( V_{O_2} = 150 \text{ ml/min/m}^2 \)

- Saturations
  - SVC = 70
  - RA = 73
  - IVC = 80
  - RV = 85
  - PA = 80
  - Ao = 95

- Pressures
  - mRA = 6
  - RV = 80/6
  - PA = 80/40/60
  - mL A = 8
  - Ao = 80/50/65
Shunt

\[ \text{QP:QS} = \frac{\text{Ao sat} - \text{MV sat}}{\text{PV sat} - \text{PA sat}} \]

Different ways to calculate MV:

A - \( \frac{3 \times \text{SVC} + \text{IVC}}{4} = 72.5\% \)
B - SVC alone = 70\%
C - \( \frac{\text{SVC} + \text{IVC}}{2} = 75\% \)

\[ \text{QP:QS} = \frac{95 - A, B, C}{95 - 80} \]

A: 1.5:1
B: 1.66:1
C: 1.33:1
PVR calculation - room air

**Capacity** = $1.36 \times 100 = 136 \text{ ml } O_2/l$

**$O_2$ content** =
- \( PA = 136 \times 80\% = 109 \text{ ml } O_2/l \)
- \( PV = 136 \times 95\% = 129 \)

**Pulmonary AV $O_2$ difference** = 20 ml $O_2/l$

\( Q_p = \frac{150}{20} = 7.5 \text{ l/min/m}^2 \)

\( PVR = \frac{(60-8)}{7.5} = 6.9 \text{ Wood units/m}^2 \)
PVR calculation - 100% $O_2$

Capacity = $1.36 \times 100 = 136$ ml $O_2/l$

$O_2$ content =
- $PA = 136 \times 95\% + 0.03 \times 85 = 131.7$ ml $O_2/l$
- $PV = 136 \times 100\% + 0.03 \times 600 = 154$

Pulmonary AV $O_2$ difference = 22.3 ml $O_2/l$

$Q_p = \frac{150}{22.3} = 6.73$ l/min/m$^2$

$PVR = (60 - 8)/6.73 = 7.72$ Wood units.m$^2$

If dissolved $O_2$ not included….PVRI = 2.4 Wood.units.m$^2$

Saturations ($P_{O_2}$)
- $SVC = 75$ ($P_{O_2} 45$)
- $RA = 80$
- $RA = 94$
- $PA = 95$ ($P_{O_2} 85$)
- $Ao = 100$ ($P_{O_2} 600$)
Summary Conclusions

A shunt run requires active assessment of each sample

Shunt flow ratio % PVRI calculations are not complicated but require attention to detail, understanding of sources of error and flow hemodynamics