Neonatal Shock

Holger Michel

Department of Neonatology, University Children's Hospital Regensburg (KUNO), Hospital St. Hedwig of the Order of St. John, University of Regensburg, Regensburg, Germany

European School of Neonatology

The ESN is a project by the European Society for Paediatric Research

EAPS 17-20 October, Vienna & Online

The 10th Congress of the EUROPEAN ACADEMY OF PAEDIATRIC SOCIETIES

No conflicts of interest to declare

EAPS 17-20 October, Vienna & Online
The 10th Congress of the EUROPEAN ACADEMY OF PAEDIATRIC SOCIETIES

Overview:

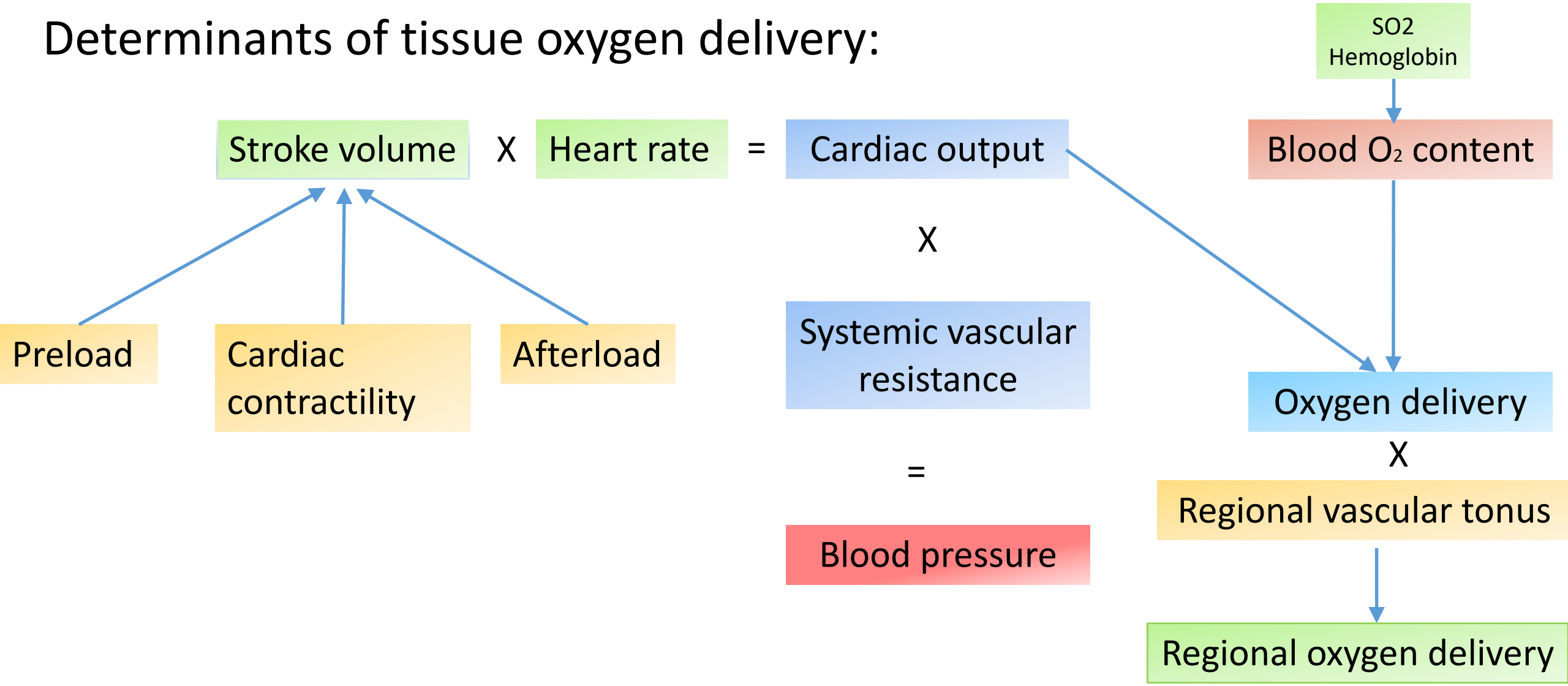
- Defining Shock
- Pathophysiology of Shock
- Assessment of neonatal Shock
- Echocardiography
- Aspects of treatment
- Back to the ward

Defining Shock:

- State of significant systemic **reduction of tissue perfusion**
- **oxygen delivery** does not meet the demand.
- This results in a cellular **depletion of energy** (ATP synthesis)

  - Cell membrane ion pump dysfunction
  - Intracellular edema
  - Leakage of intracellular contents into extracellular space
  - Inadequate regulation of intracellular pH

Determinants of tissue oxygen delivery:

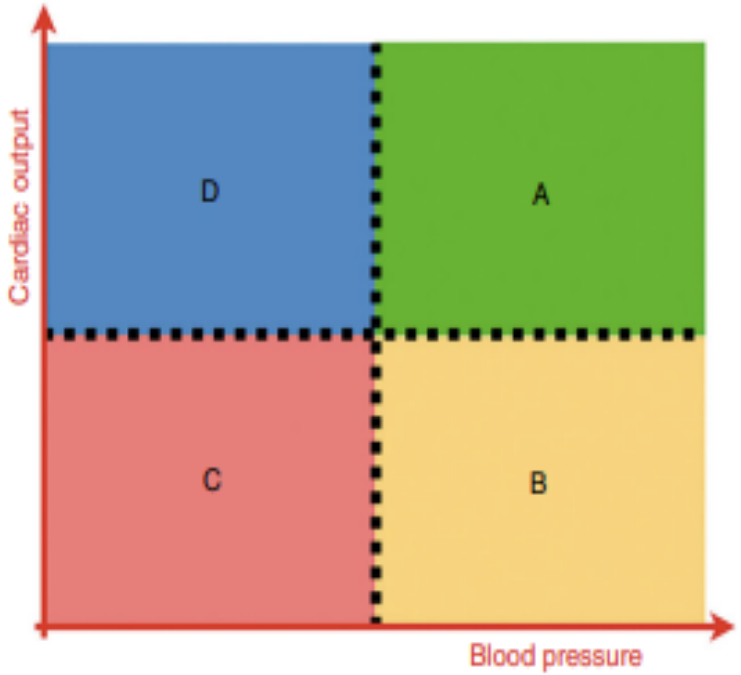


Stages of shock

- **Compensated shock**: Oxygen delivery and perfusion towards vital organs is maintained

- **Uncompensated shock**: perfusion and oxygen delivery of the vital organs fails

Stages of shock



	Cardiac output	Blood pressure	Situation
A	Normal/ High	Normal/ High	Normal
B	Low	Normal/ High	Compensated shock
C	Low	Low	Uncompensated shock
D	Normal/ High	Low	Hyperdynamic circulation

Willem P. de Boode et al. , PediatricResearch 2018

Definition shock: What we really need to know is **Blood Flow** (oxygen supply)

Whats the role of **Blood Pressure** ?

EAPS 17-20 October, Vienna & Online The 10th Congress of the EUROPEAN ACADEMY OF PAEDIATRIC SOCIETIES

Neonatal shock and blood pressure:

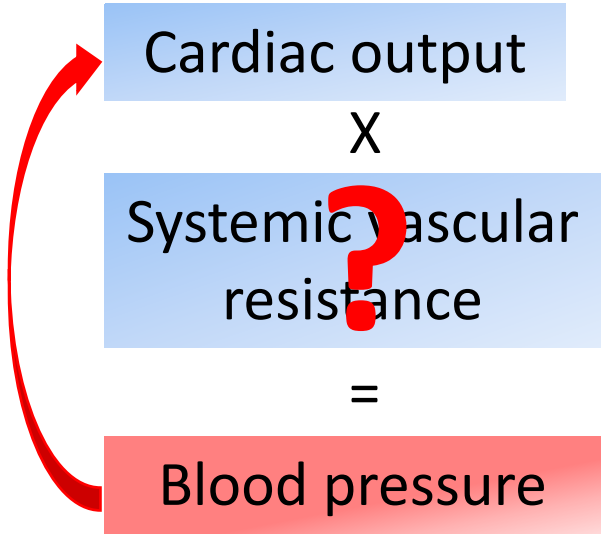
NIBP

- Easy to measure
- Non-invasively
- Automated measurement
- Repeated acquisition

Arterial blood pressure

- Continuous measurement
- More reliable than NIBP
- Arterial blood gas

Blood pressure reflects cardiac output (at a given vascular resistance)



If you look at **diastolic BP** and **systolic BP** it can tell you about the pathophysiology

Neonatal shock and blood pressure:

- Are there any normal values

- How do we treat it ?

STATE-OF-THE-ART

Treating hypotension in the preterm infant: when and with what: a critical and systematic review

Design: Systematic review of the literature in order to determine which preterm infants may benefit from treatment with interventions to elevate blood pressure (BP), and which interventions improve clinically important outcomes.

Results: Our review was not able to define a threshold BP that was significantly predictive of a poor outcome, nor whether any interventions for hypotensive infants improved outcomes, nor which interventions were more likely to be beneficial.

Conclusions: There is a distinct lack of prospective research of this issue, which pre
threshold that in

Dempsey & Barrington. Journal of Perinatology (2007) 27, 469–478

RECENT ADVANCES

Hypotension in the very low birthweight infant: the old, the new, and the uncertain

Volume expansion 10–20 ml/kg 0.9% saline

1st line inotrope Dopamine 5–20 µg/kg/min

2nd line inotrope Dobutamine 5–20 µg/kg/min

Life threatening hypotension

Noradrenaline 0.2–1 µg/kg/min

Rescue therapy Dexamethasone 250 µg/kg single dose

454

Is there a minimum Blood pressure at all ?

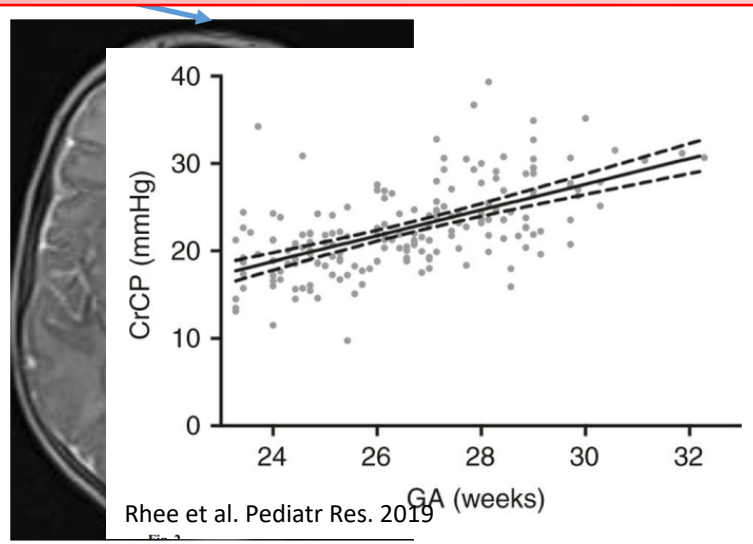
Is there a minimum blood pressure at all ?

Organs dependend on pressure

Systolic / mean

d pressure

Normal values are still lacking



Rhee et al. Pediatr Res. 2019

Assessment of Neonatal Shock

Multimodal approach

Clinical parameters

Blood pressure

Capillary refill

Urinary output

Level of conciousness

Laboratory test

pH, BE, Lactate

Central Venous Saturation

Hb, Creatinine, Liver function

Echocardiography

NPE

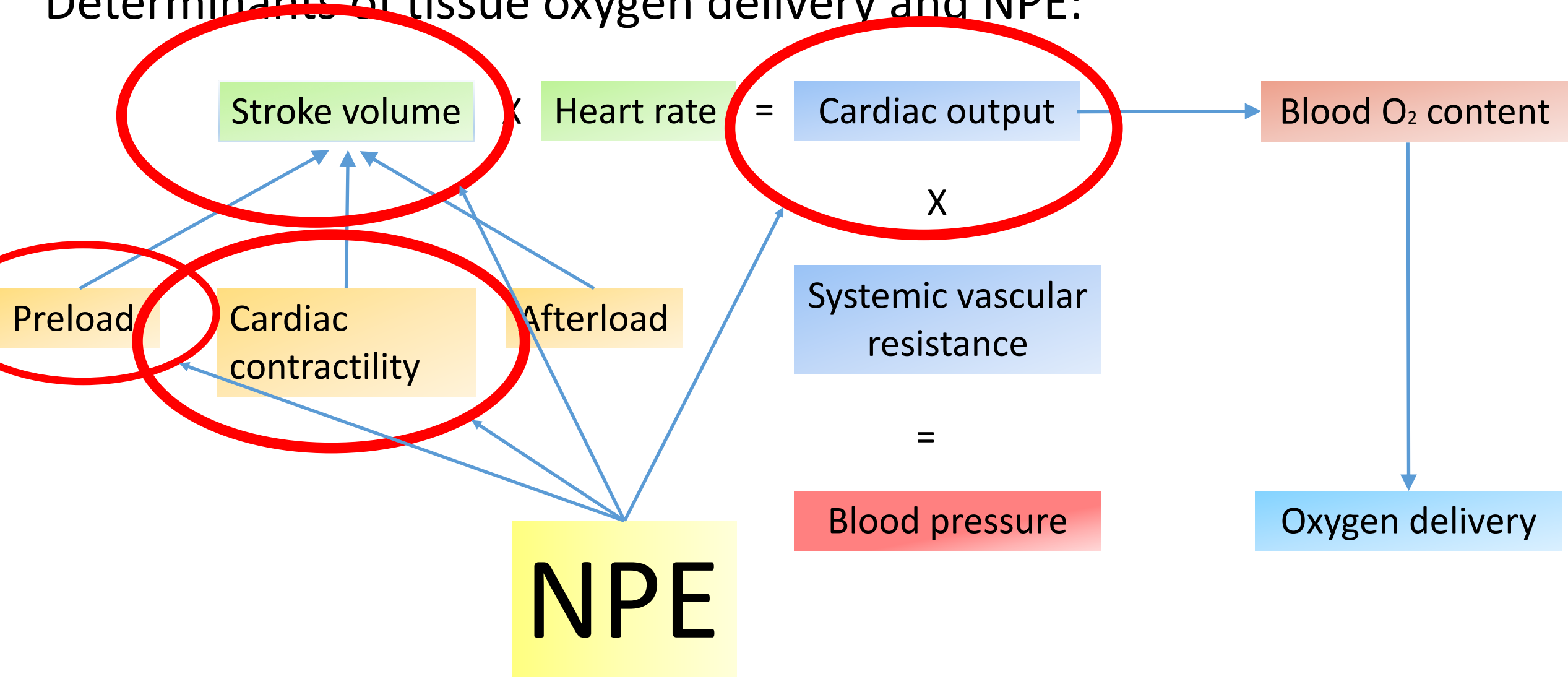
Understand the situation

EAPS The 10th Congress of the EUROPEAN ACADEMY OF PAEDIATRIC SOCIETIES
17-20 October, Vienna & Online

Assessment of shock clinical parameters:

	+	-
- **Blood pressure:**	- easy to get	- Pressure; not Flow
- **Diuresis:**	- easy to get, measure continuously	- Time delay
- **Capillary refill:**	- easy to get	- subjective, not always correlate with organ perfusion
- **Level of conciousness:**	- easy to get	- lots of confounders (sedation etc.)
- **BGA, Lacate:**	- reflects O2 supply, regional and whole body	- Confounders, regional/general perf.
- **Central venous SO2:**	- Reflects cardiac output	- Need for central line

Determinants of tissue oxygen delivery and NPE:



Preload, Assessment of volume status:

- Corrcetion of true hypovolemia is essential
- Excessive fluid administration is associated wíth morbidity and mortality
- No studies to assess predictive value of hemodynamic variables

Clinical evaluation:

- Heart rate, blood pressure, diuresis
- Arterial blood pressure variation,
- Response to fluid trial
- Liver compression

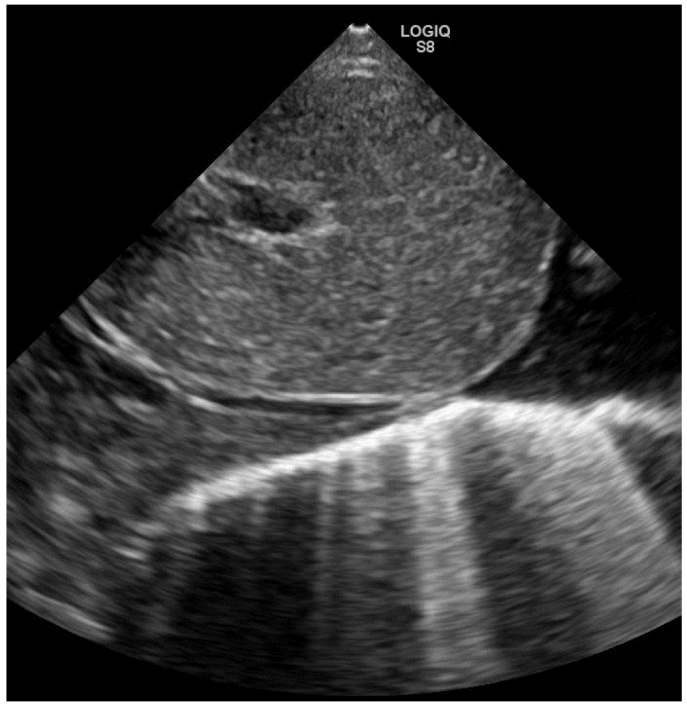
Echocardiography:

- IVC filling and collapsibility
- Left atrial filling (LA/AO)
- Left ventricular end-diastolic filling

Echocardiography: Assessment of volume status

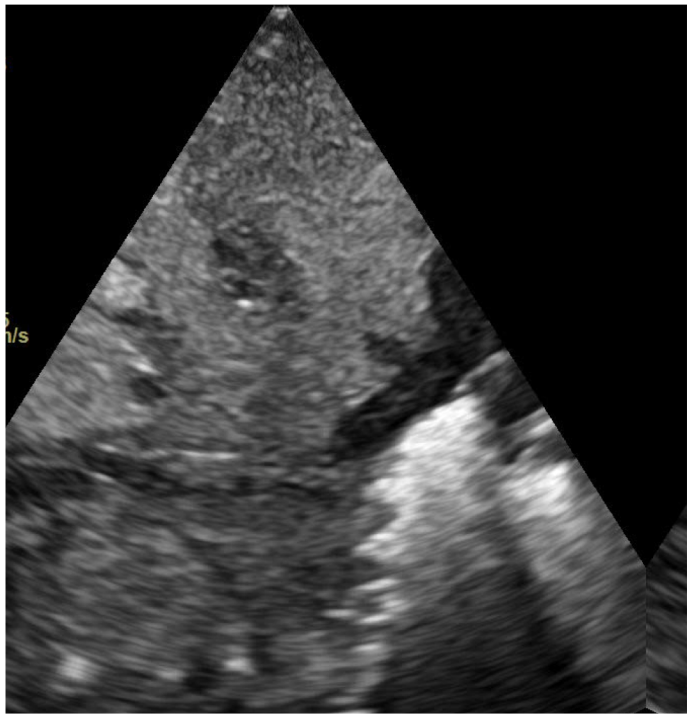
IVC Filling:

Low



regular

congested

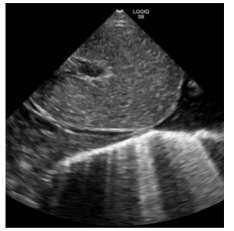


Assessment IVC filling:

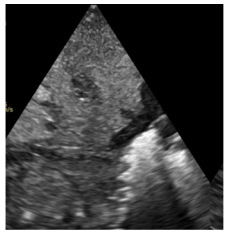
Transducer position:
- midline, just below the xiphoid process, and in the sagittal plane
- probe marker should be pointing towards the head, so that the heart appears just visible on the right of the screen

Normal Filling:
- pulsation with the cardiac cycle and respiratory motion

Under-filled:
- barely visible or collapse entirely on inspiration



Over-filled:
- large, and minimally pulsatile

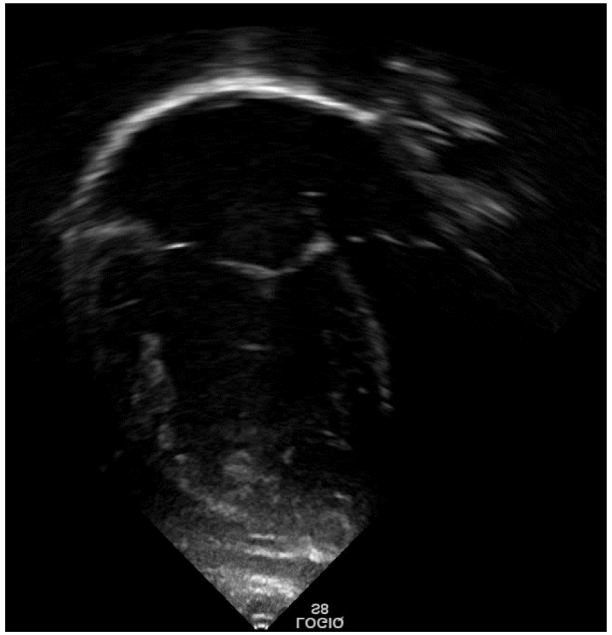


Assessment IVC filling: Confounders

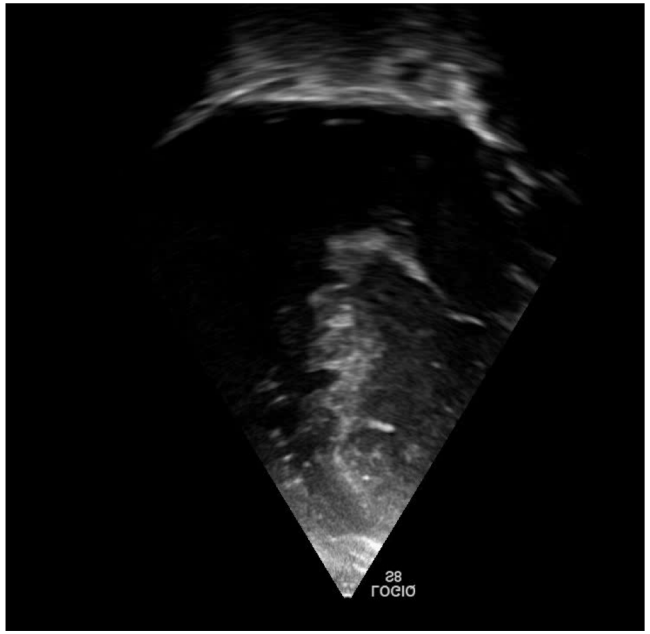
- Underfilling can be detected reliably

- Overfilling: Distinguishing between (true) volume overload and reduced venous return due to heart failure or increased intrathoracic pressure (e.g. high frequency oscillatory ventilation) is difficult

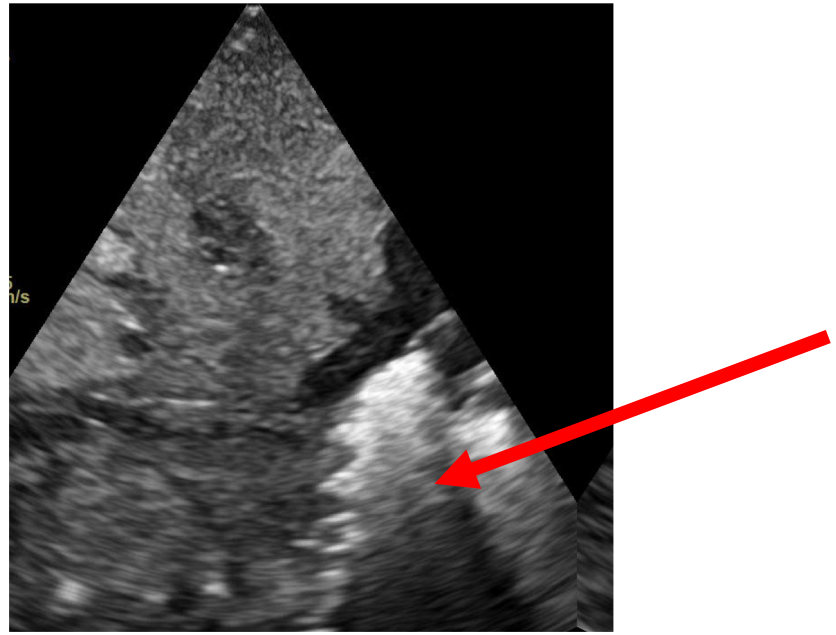
PH



Cardiac malformations (e.g. M. Ebstein)



HFO-Ventilation



EAPS 17-20 October, Vienna & Online
The 10th Congress of the EUROPEAN ACADEMY OF PAEDIATRIC SOCIETIES

Assessment of cardiac filling:

Transducer position: - 4 chamber view parasternal short axis ps long axis



Parameters:

Left ventricular filling	- M-Mode: LVED(d), - Eye balling
Left atrial filling	- LA / AO - Planimetrics, Volumetrics
Diastolic blood flow peripheral arteries (e.g, truncus coeliacus)	

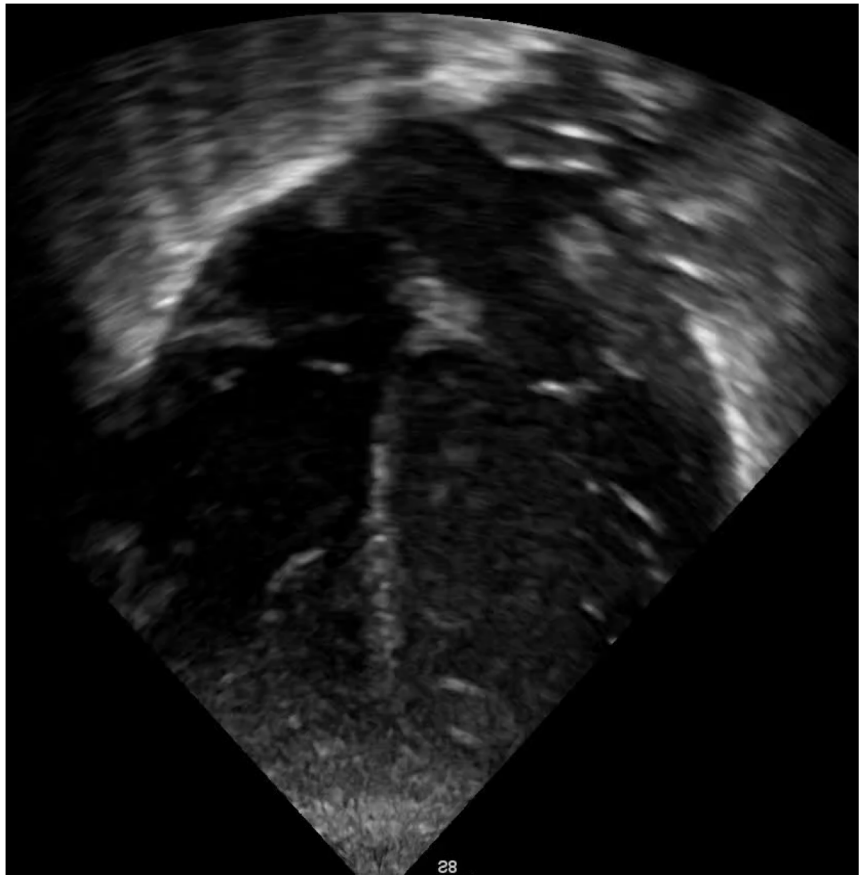
Confounders:

Left – right shunts (e.g. PDA), PH
Left – right shunts (e.g. PDA), PH
Diastolic run-off (e.g. PDA)

Assessment of left ventricular filling: Eyeballing 4 chamber view

Preterm 25 weeks GA, 460 g:

Underfilling

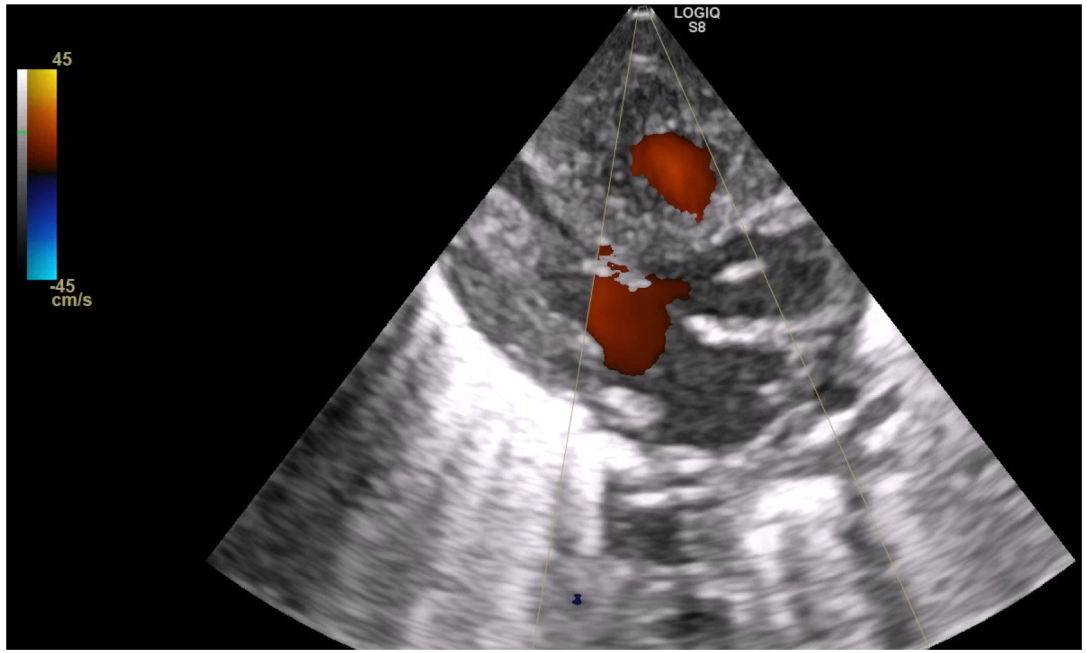


regular filling / overfilling

Assessment of left ventricular filling: Eyeballing parast long / short axis

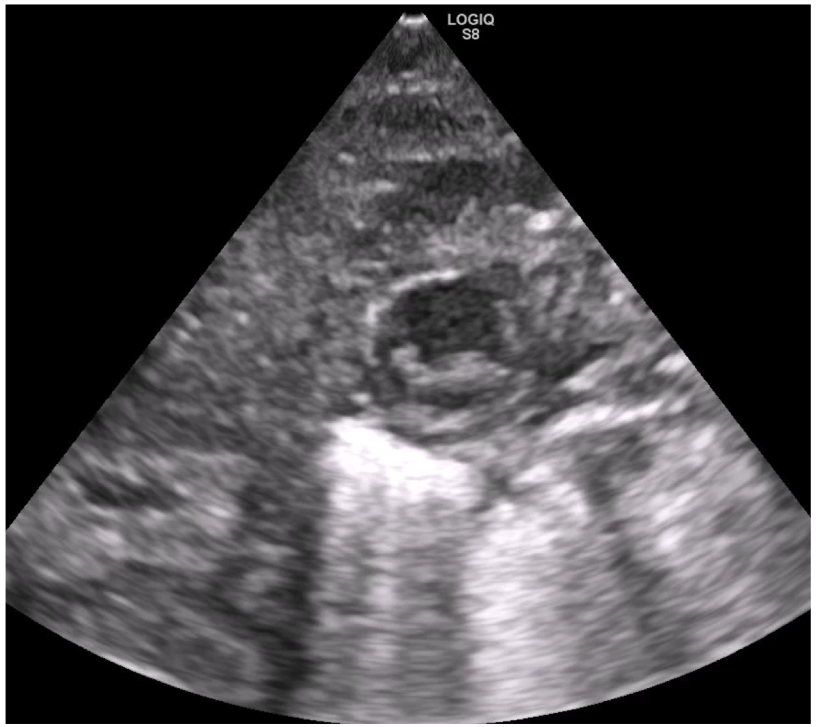
Example cardiac underfilling preterm 24 weeks GA, age 5 days

Parasternal long axis



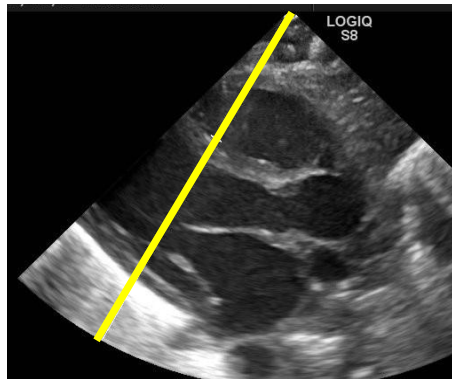
Underfilling

Parasternal short axis

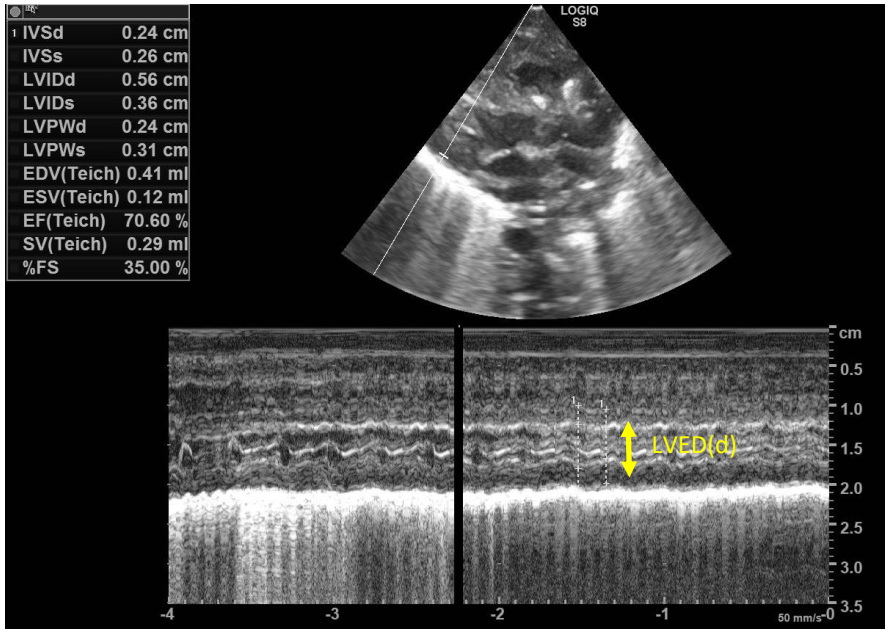


Assessment of left ventricular filling:

M-Mode, parasternal long axis view

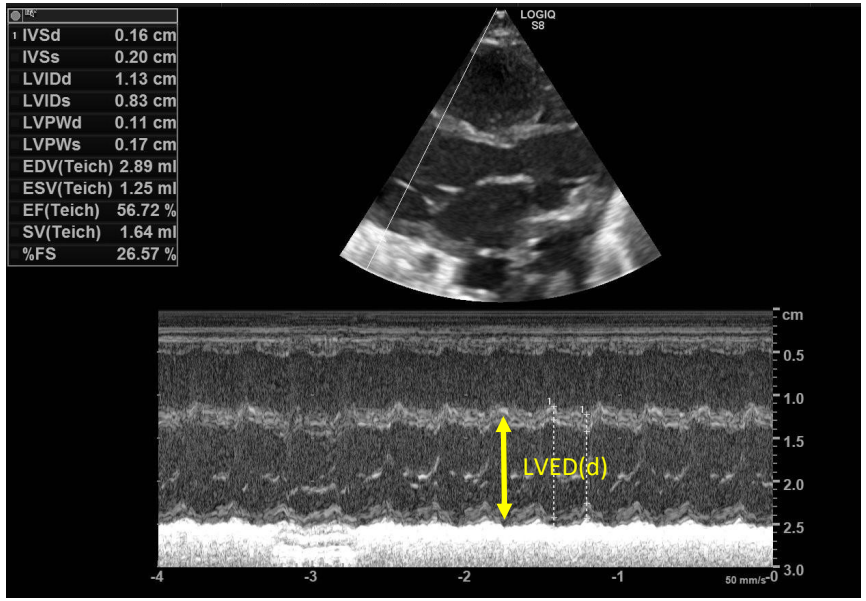


Under-filled LV



LVED(d) 5.6 mm

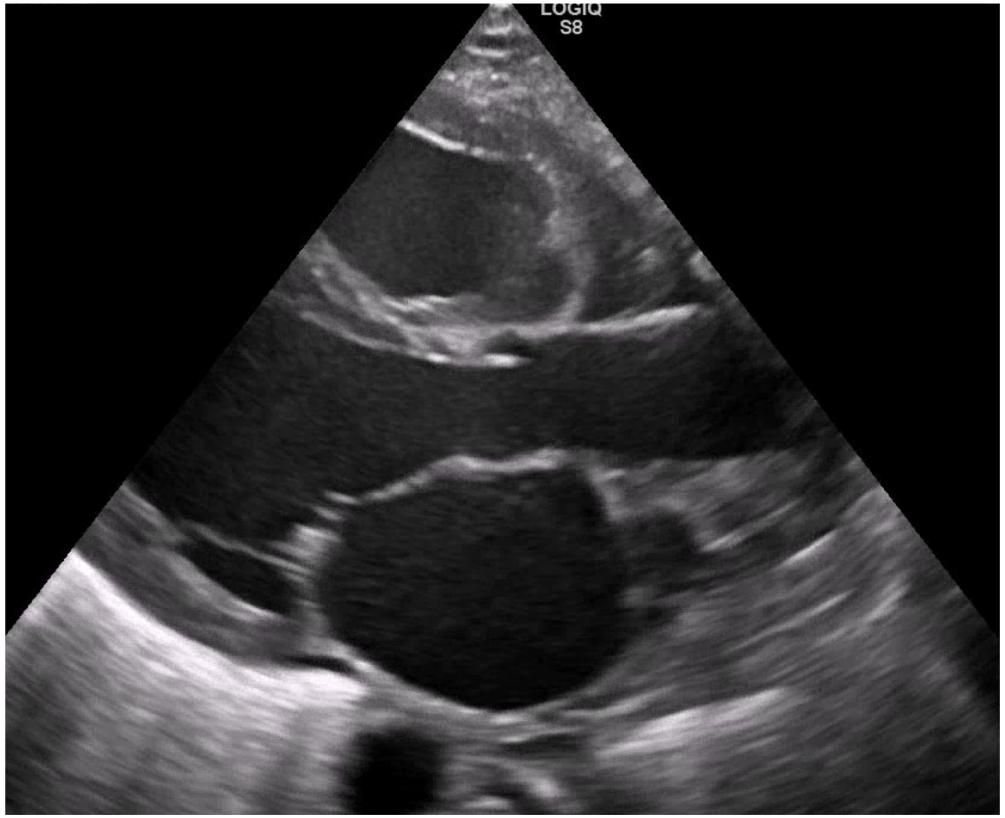
Regular filling



LVED(d) 11.3 mm

Assessment of left atrial filling:

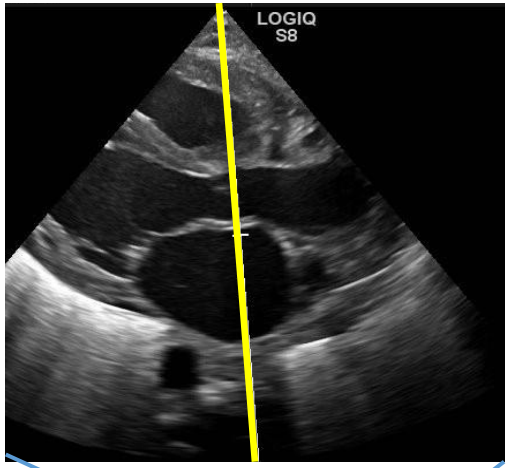
Parasternal long axis view: Eyeballing

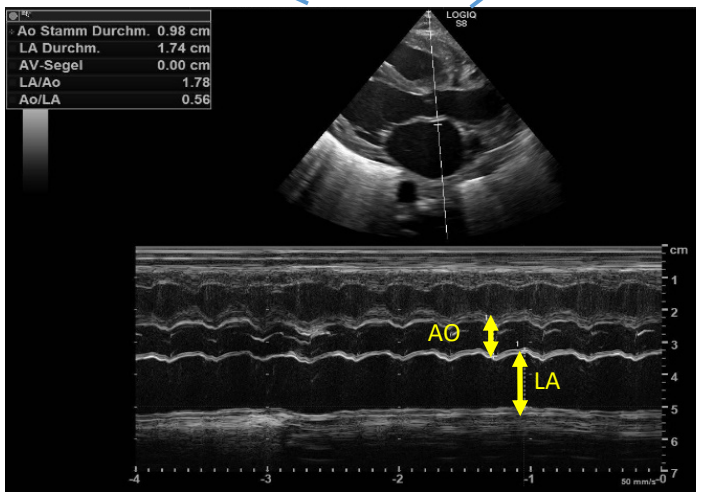


Preterm, GA 35 weeks, reduced LV-Function

Left atrium dilated

M-Mode





LA / AO = 1,8

Assessment of left atrial filling:

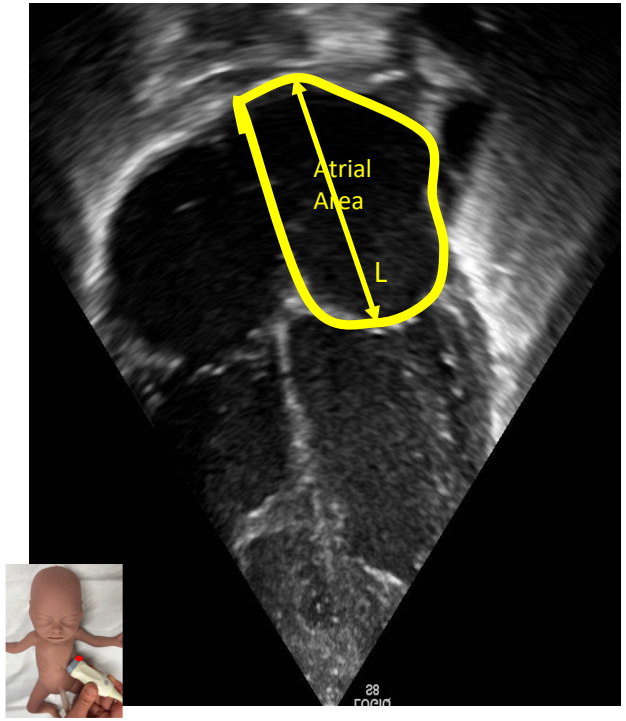
Bi-plane volume:

Measure and systole, (maximum size)

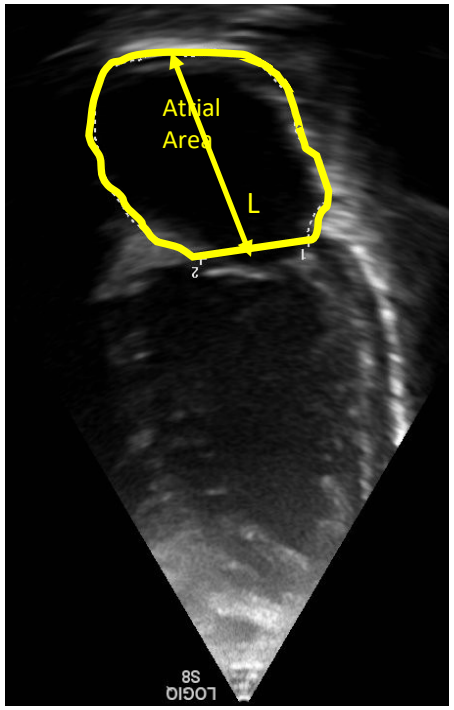
3 different formulas:

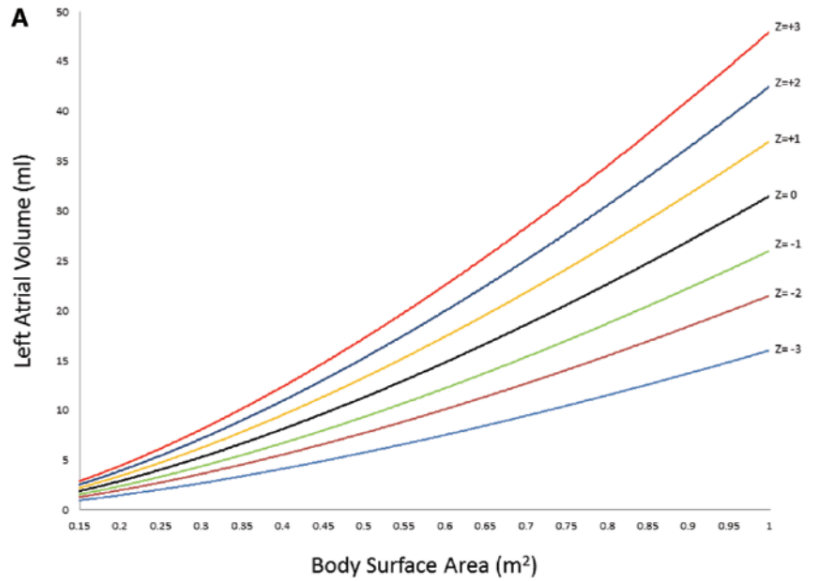
- Area lenght (using biplane atrial area and lenght)
- Simpson (disc method)
- ellipse method

4 chamber view



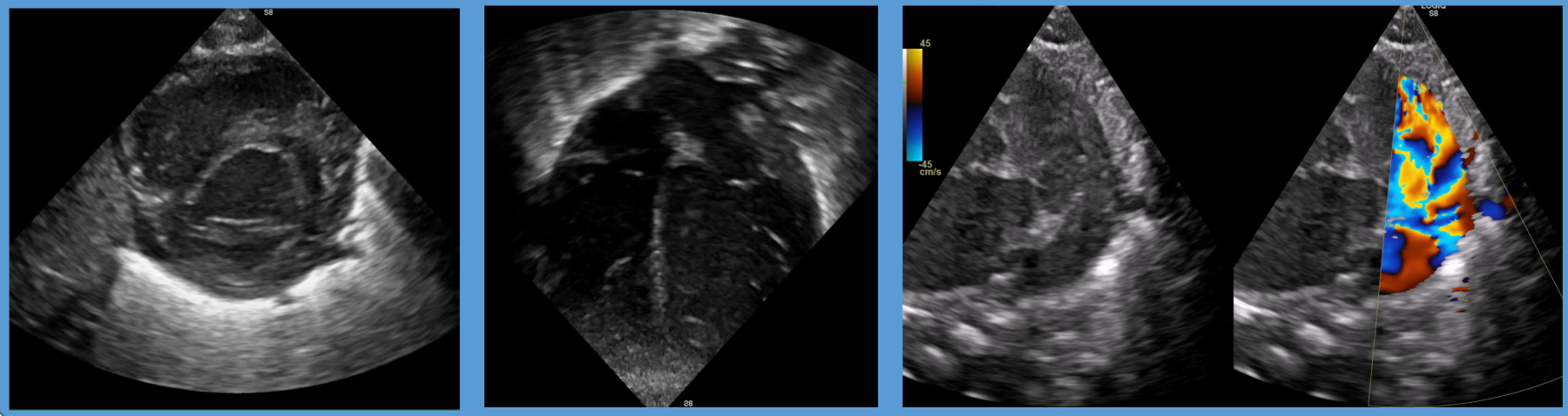
2 chamber view



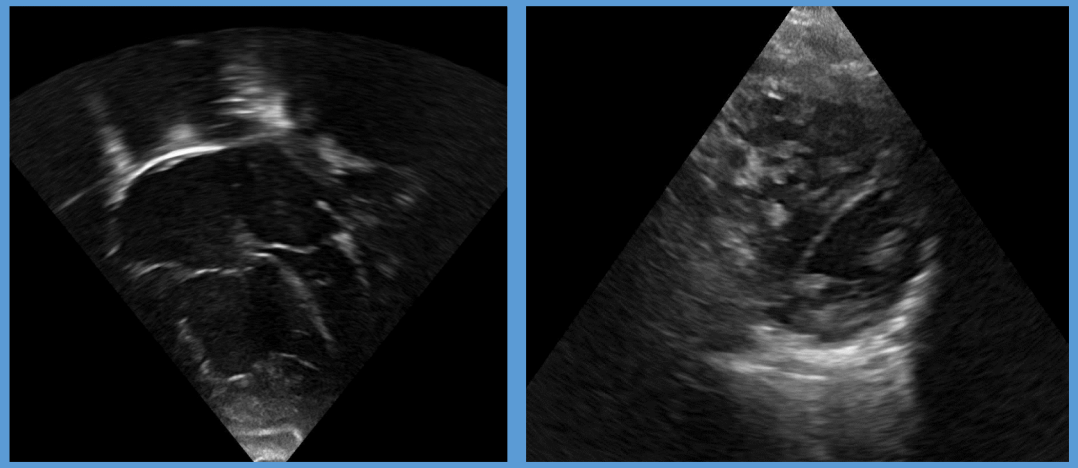


Bhatla et al. Circ Cardiovasc Imaging 2012

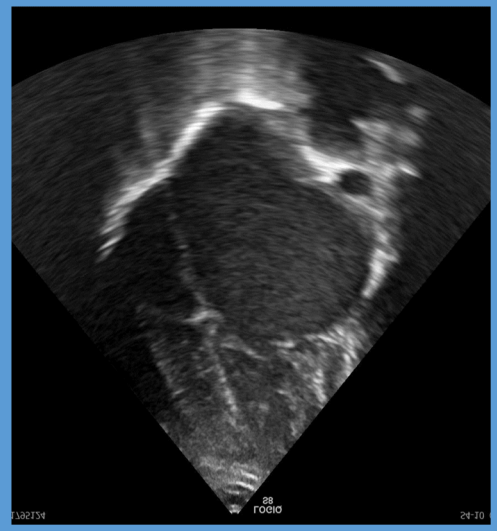
Assessment of cardiac filling - confounder:



PDA



PH



Mitral-Stenosis

Assessment of volume status:

- Assessment and therapy is essential in neonatal shock

- NPE: - can be helpful if you know the confounders and

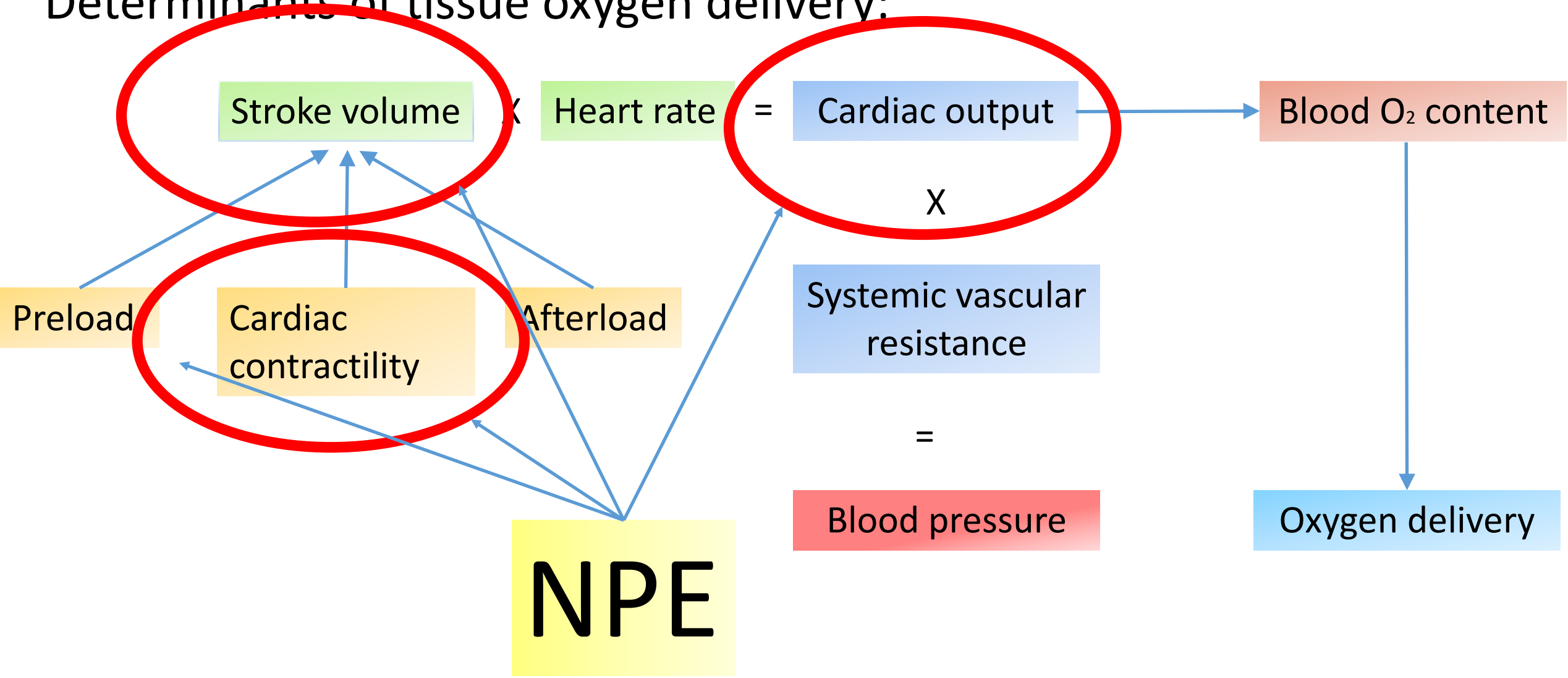
  use it in a combined multimodal approach (clinical background + NPE)

  - Underfilling can be assessed easy, reliable and fast.

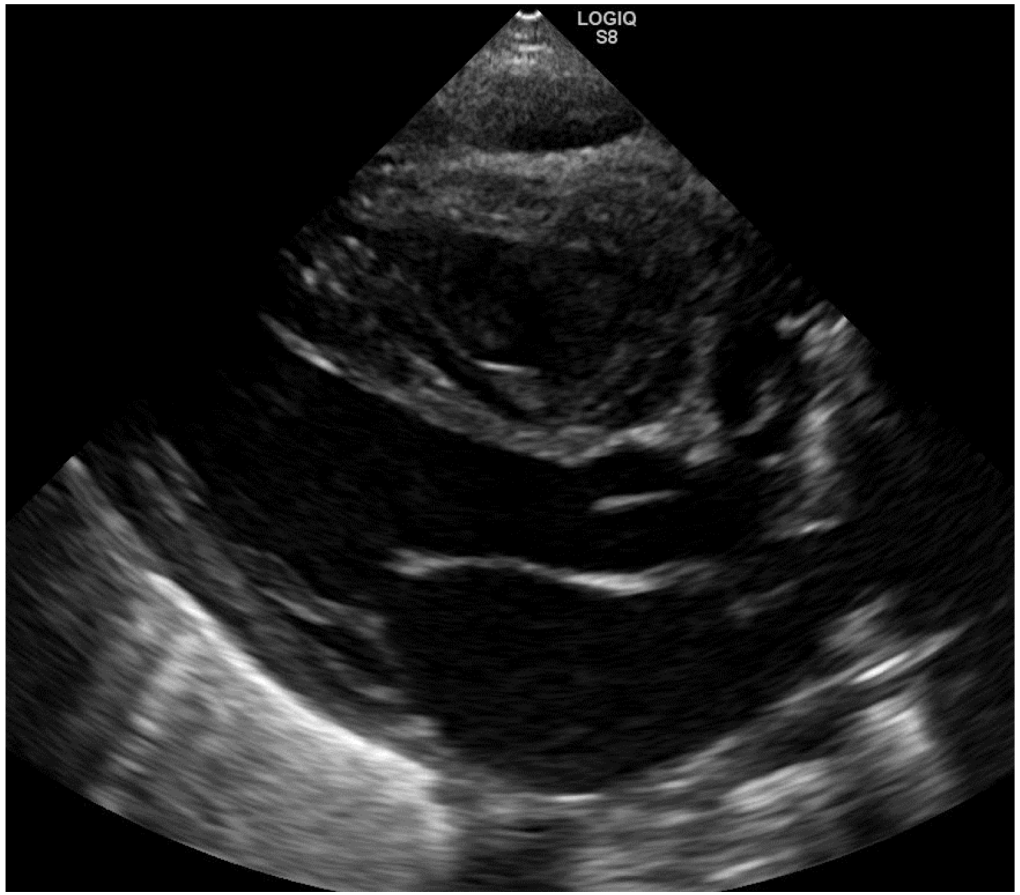
- NPE: Overall, NPE is not the best tool to assess intravascular volume in newborn infants

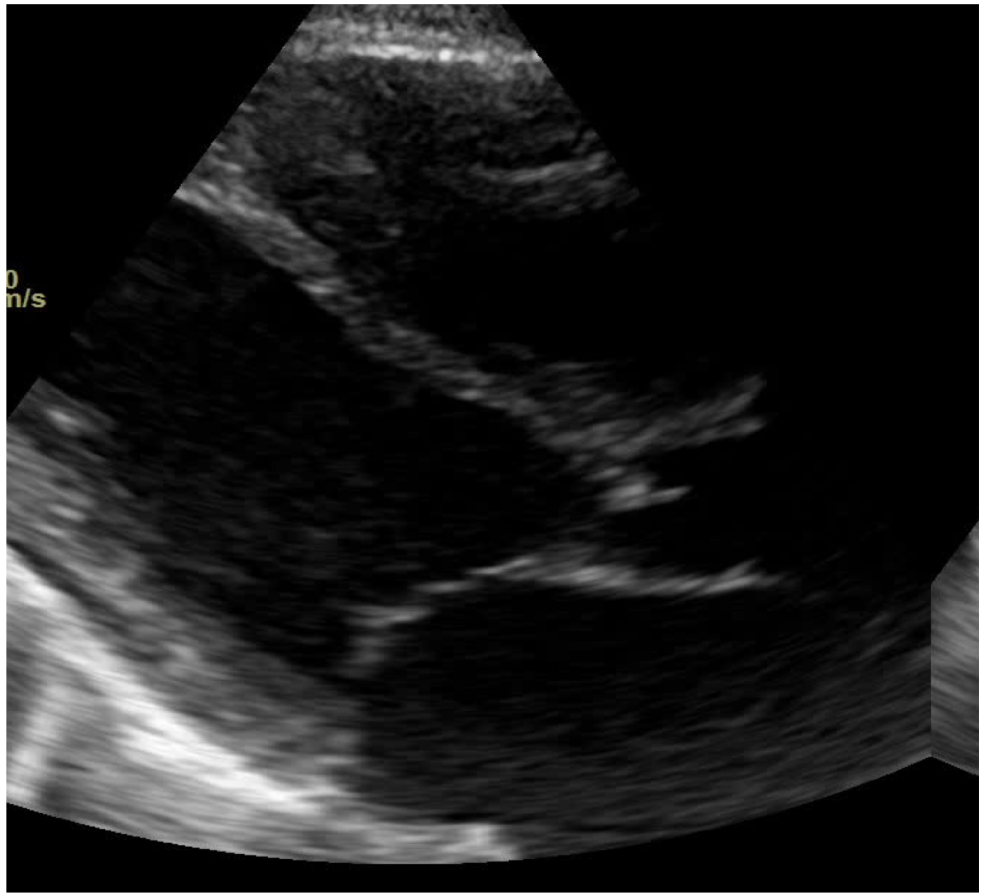
(Willem P. de Boode et al. Pediatric research 2018)

Determinants of tissue oxygen delivery:



Cardiac contractility: Eyeballing

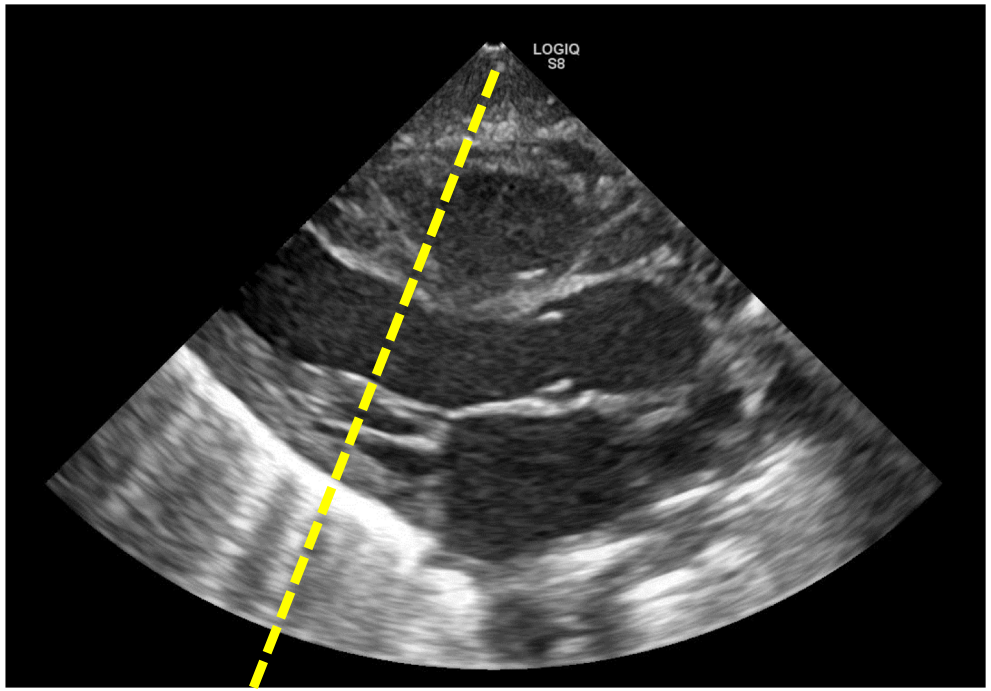


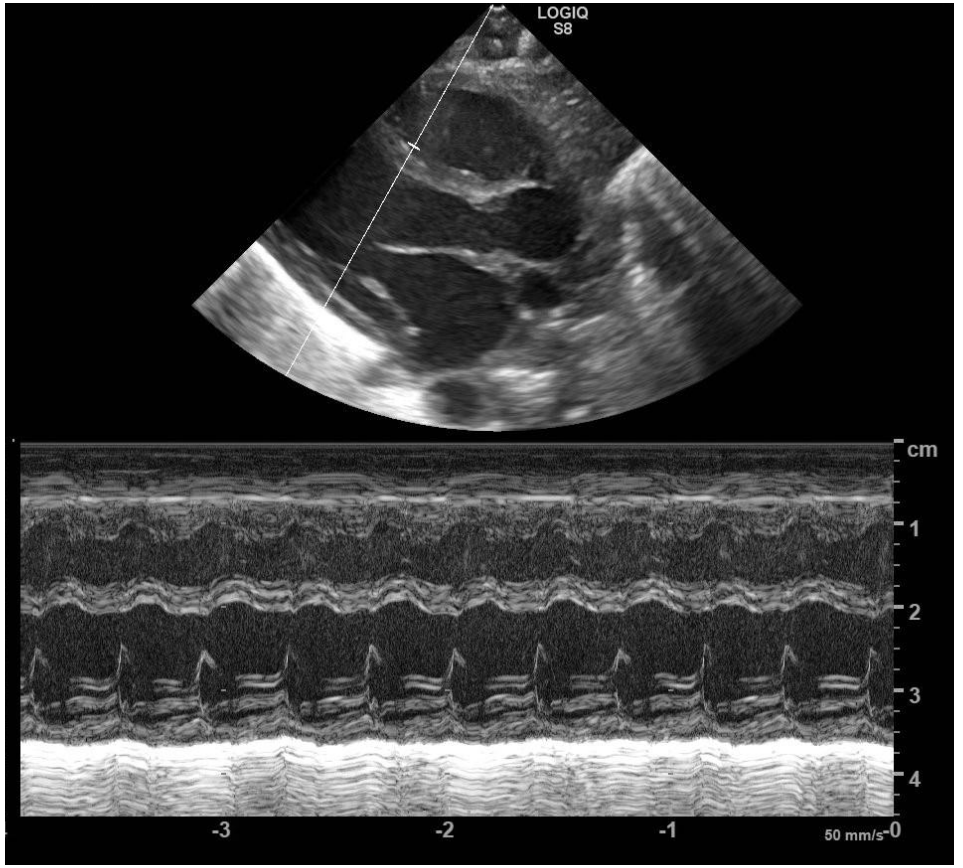


Cardiac contractility

M-Mode:

Parasternal long axis view

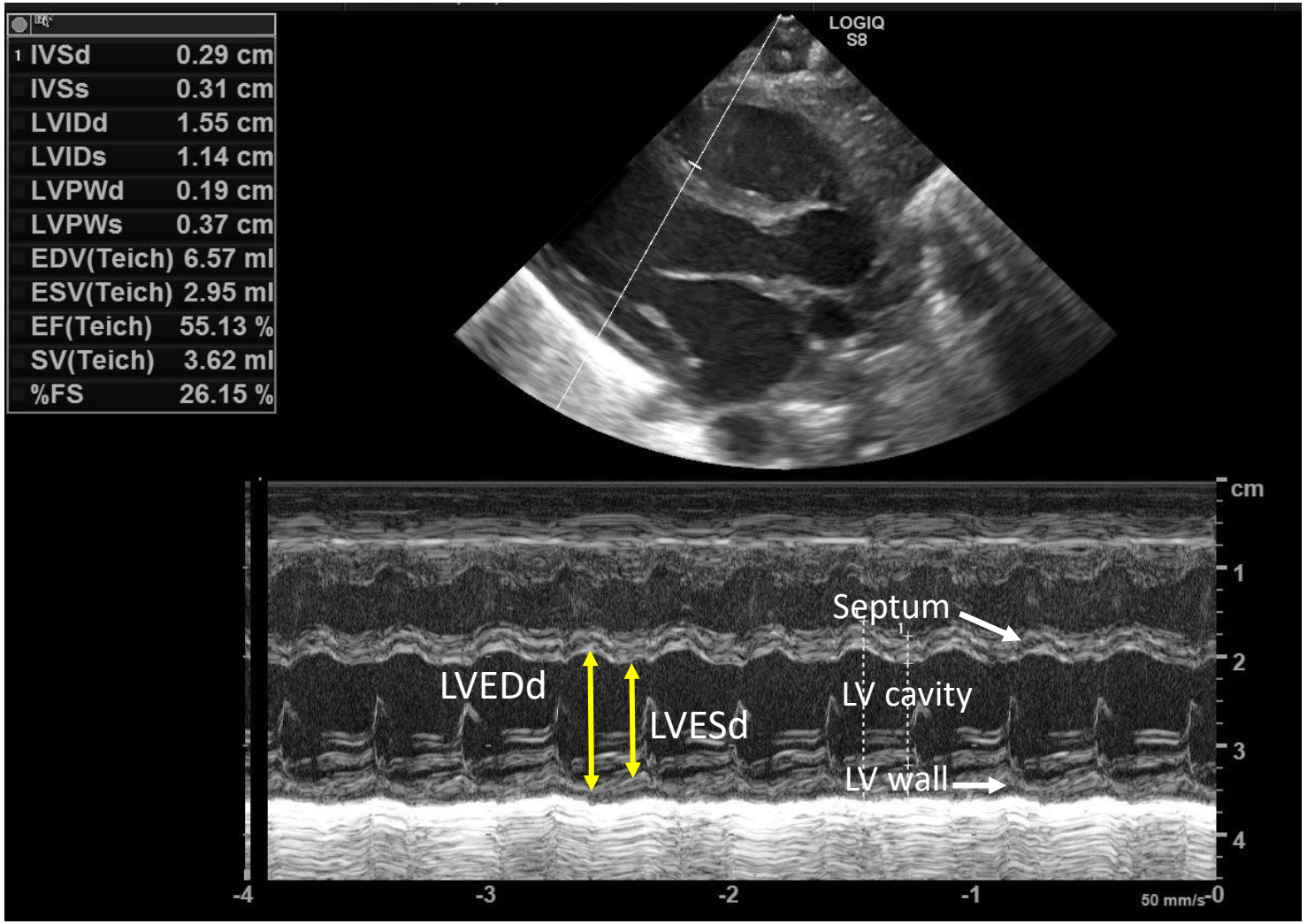




M-Mode:

$$FS\ (\%) = \frac{LVEDD - LVESD}{LVEDD}$$

Normal neonatal values are = 28 to 40%



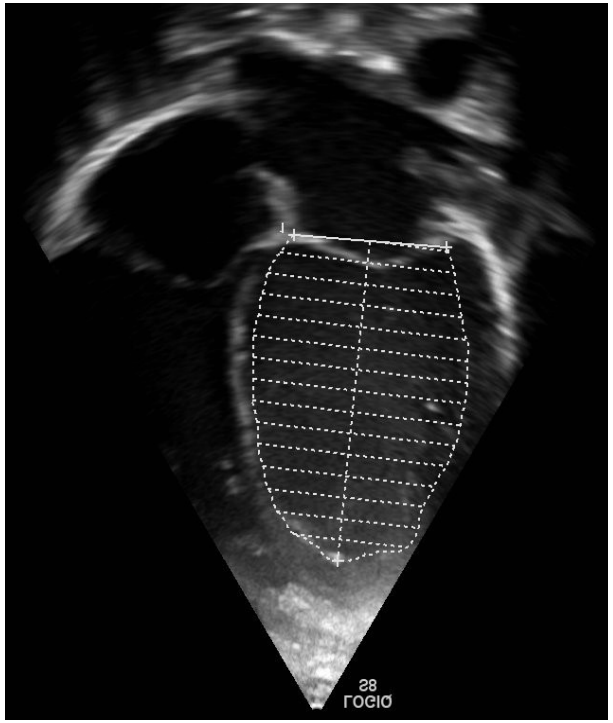
EF (Simpson): Bi-plane

Disc method using Area to calulate systolic and diastolic LV volume

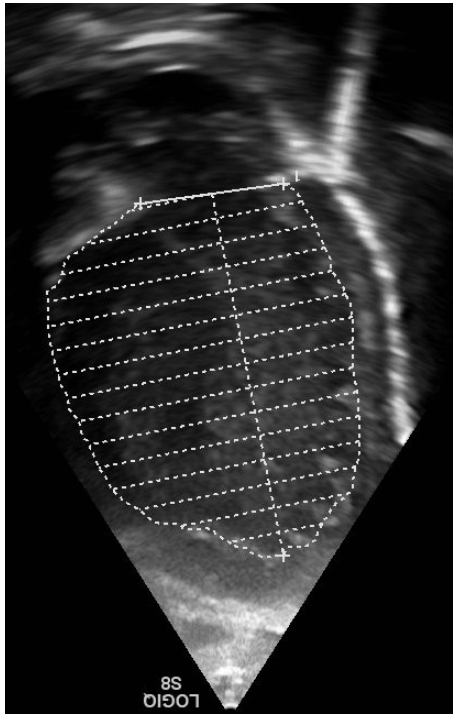
$$EF\ (\%) = \frac{LVEDV - LVESV}{LVEDV}$$

Normal neonatal values are = 55 – 65 %

4 chamber view



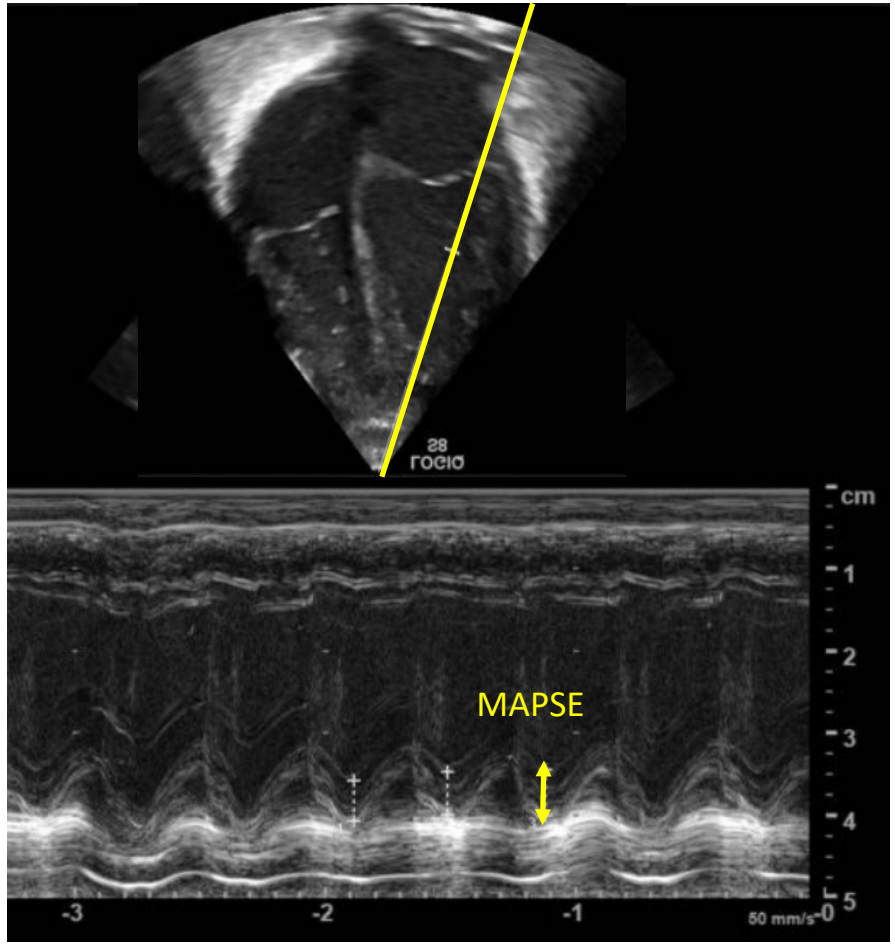
2 chamber view



Cardiac contractility - Longitudinal function

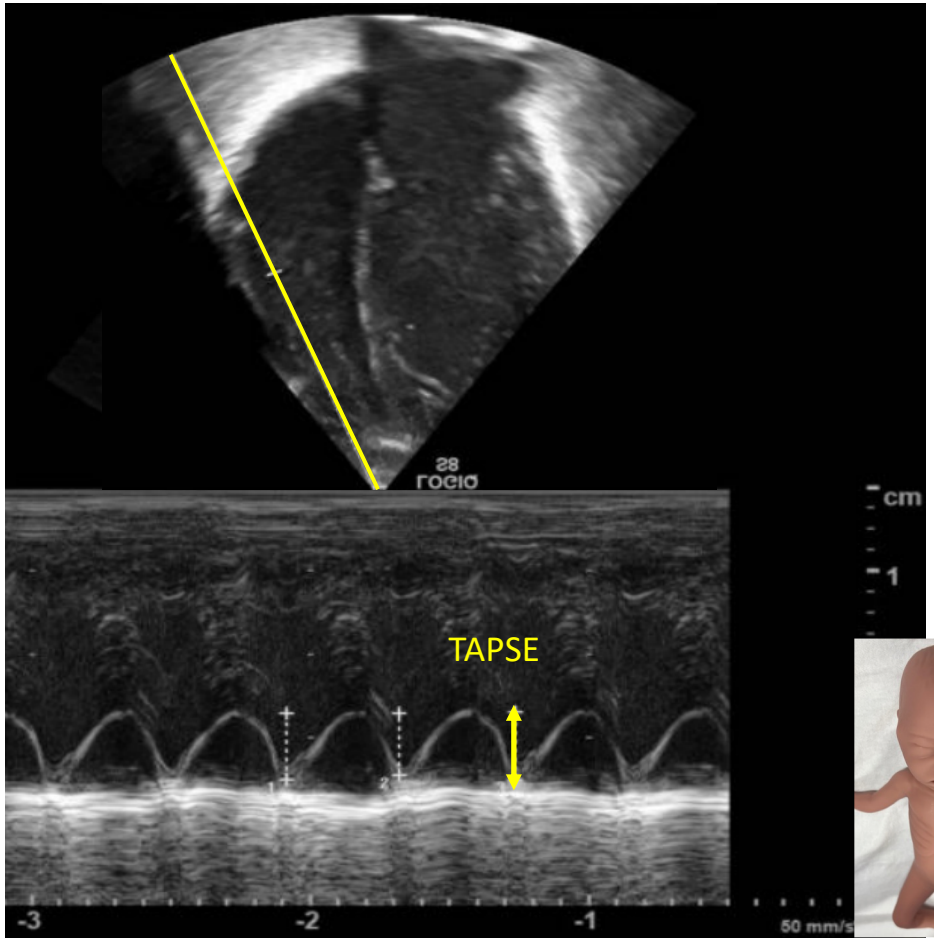
Left ventricle MAPSE

(Mitral annular plane systolic excursion)



Right ventricle TAPSE

(Tricuspidal annular plane syst. Exc.)



Left ventricular output: Velocity time integral (VTI)

VTI: - measurement 4 chamber view, PW-doppler LVOT/AV

- Area under the curve

- indicates how far the blood column is moving per heartbeat

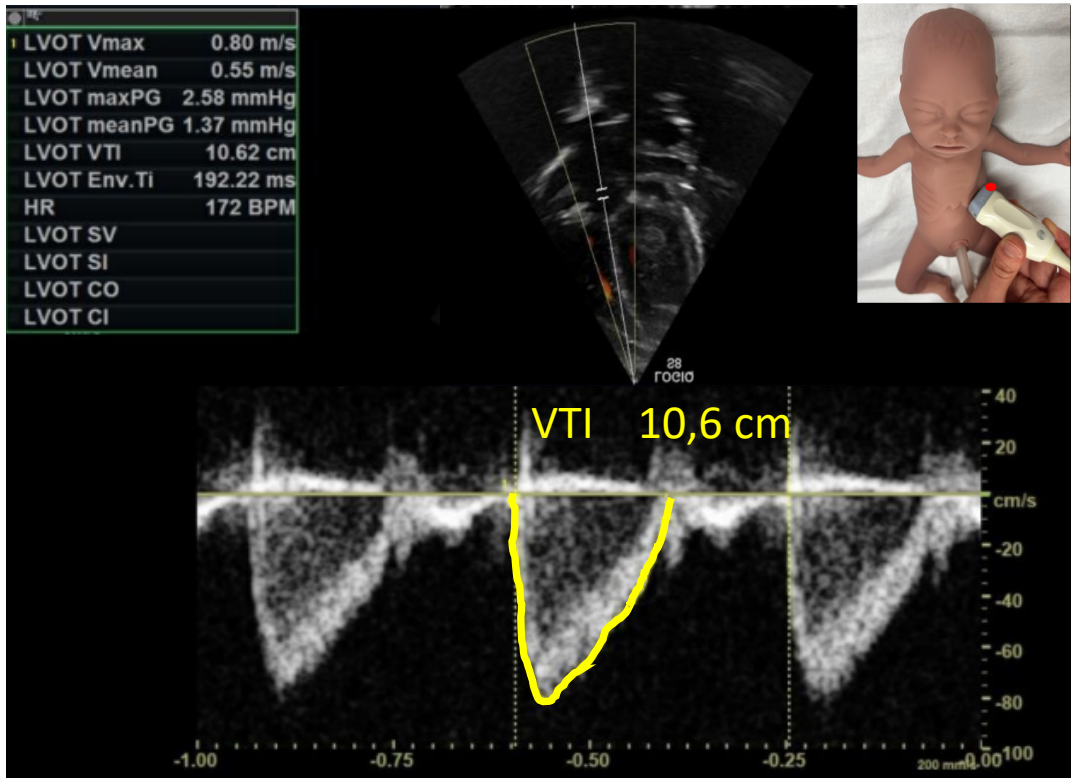
LV Stroke volume (ml) = AoCSA x VTI

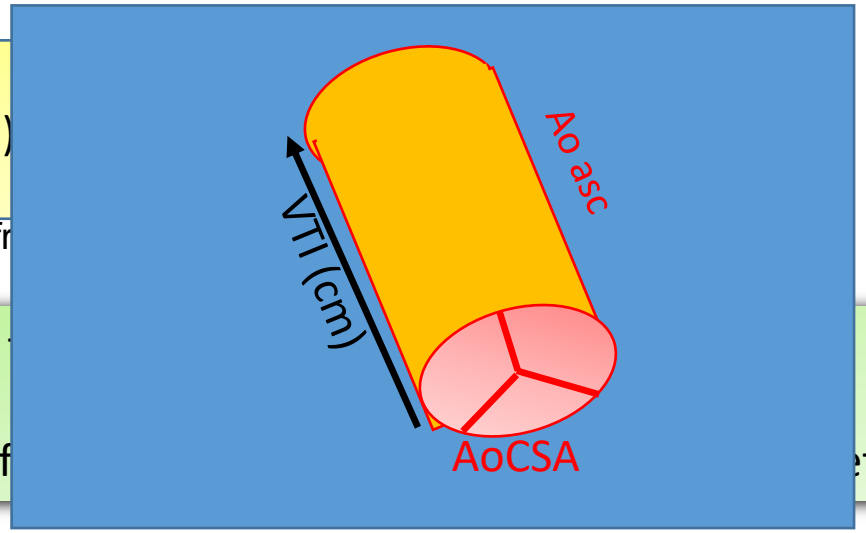
LV output (ml/min)= AoCSA x VTI x Heart Rate

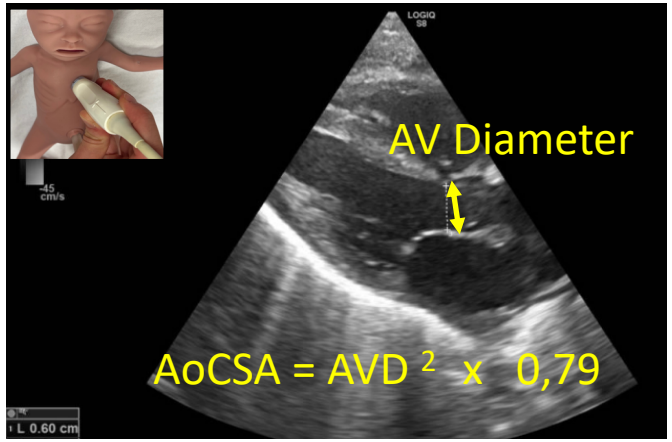
CAVE: Aortic diameter is squared for the Calculation of Stroke-Volume

CI (LV) (ml/kg/min)

Normal values range fr







- Stable parameter

- In the presence of eflect systemic perfusion

Myocardial Performance Index (MPI):

\+ Less load dependent.
Less affected by paradoxial septal wall movement

\- Technically difficult in tachycardia

$$MPI = \frac{\text{Isovolumetric contraction (1) + Isovolumetric relaxation (2)}}{\text{Ejection Time (b)}}$$

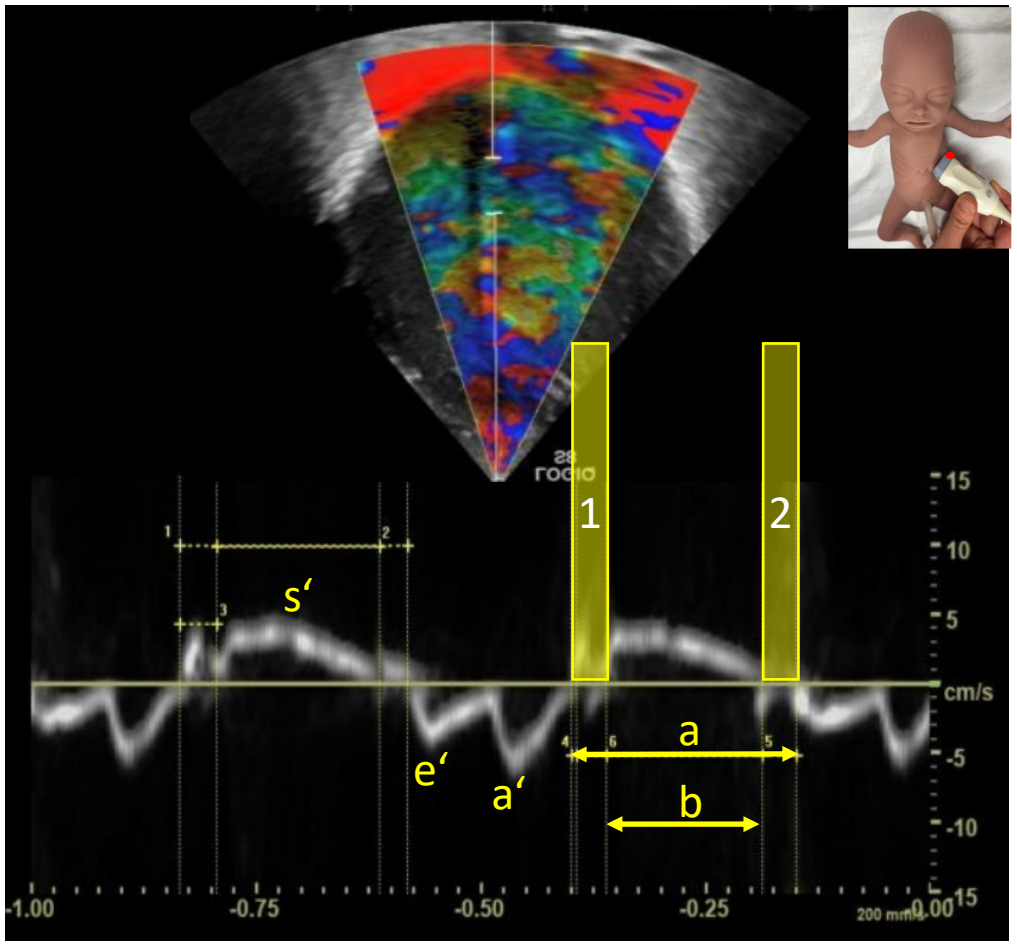
$$MPI = \frac{a - b}{b}$$

Normal values LV-MPI:

Term neonates	1st DOL: 0,37 (±0,1)	28th DOL: : 0,37 (±0,11)
Preterm	1st DOL: 0,35 (±0,09)	28th DOL: : 0,36 (±0,1)

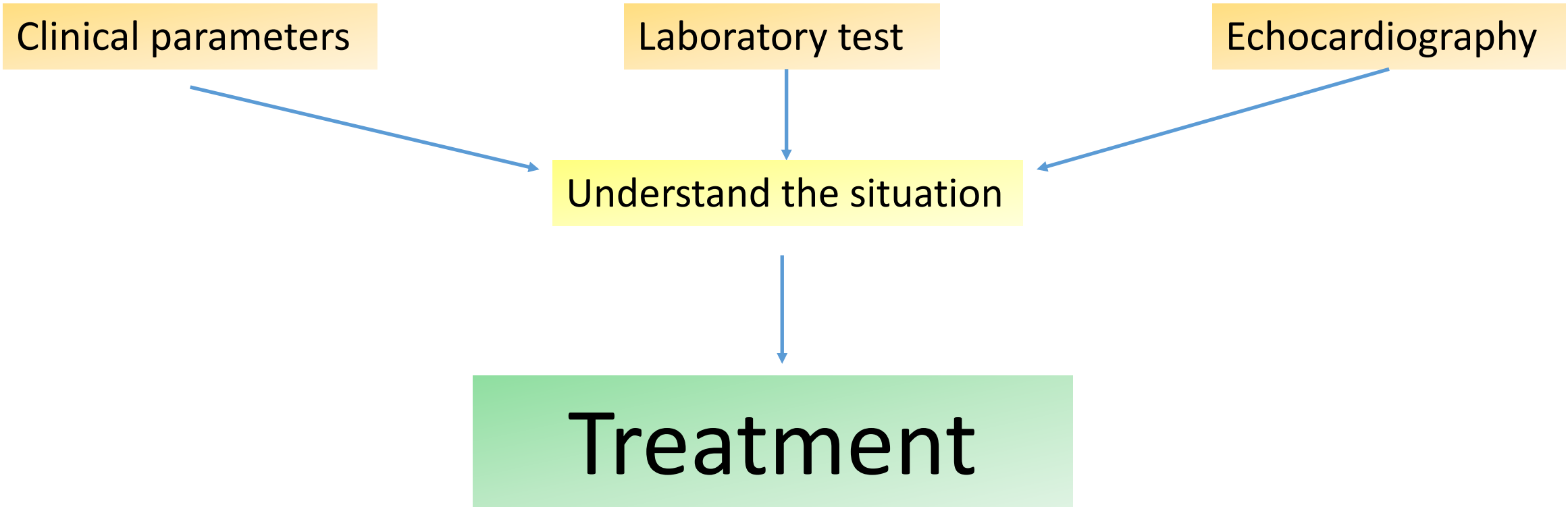
Bokiniec et al. 2016

Tissue doppler velocity and time intervals:



Assessment of Neonatal Shock

Multimodal approach

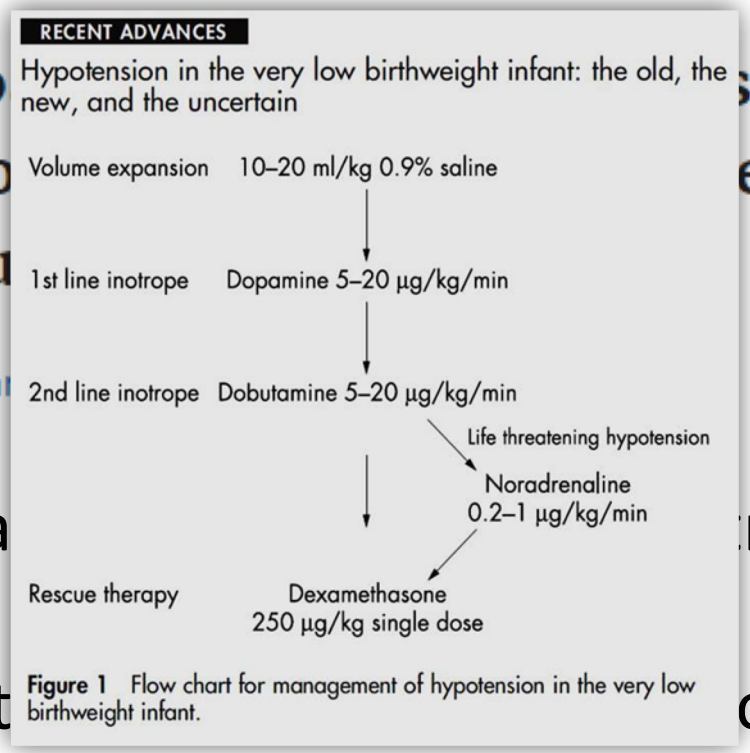


Managment of neonatal shock:

Review > Semin Fetal Neonatal Med. 2015 Aug;20(4):238-45. doi: 10.1016/j.siny.2015.03.005. Epub 2015 Mar 29.

Evidence–b siology–based approach to ent of neonatal cardiovascu

Shahab Noori [1], Istva



Given the lack of ra rolled trials …..

We must now use t of evidence and our present understanding of developmental physiology and pathophysiology …..

Pathophysiology-based approach to diagnosis and treatment of shock

Clinical triggers:
- Low blood pressure
- Low pH
- High Lactate
- Poor capillary refill
- Low urine output
- Tachycardia

Suspected low systemic blood flow state suspected

Categorise Pathophysiology

History Triggers:
- Blood loss
- Chorioamnionitis
- Perinatal asphyxia
- Hydrops fetalis
- Pulmonary hypoplasia

Low diastolic BP

NPE:

Contracility normal

Preload ↓ Afterload ↓

Hypovolemia
Blood loss
Cap. Leakage
Fluid loss
Tension pneumothx
HFOV

Warm Shock
Decreased SVR
Septic shock (warm)
SIRS
NEC
PDA

Low systolic BP

NPE:

Contractility ↓ Afterload ↑

Myocardial compromise
Asphyxia
PPHN
Cardiomyopathia

Cold shock
Increased SVR
Early sepsis (cold shock)
Transitional circulation
Hypothermia

Low Systolic/Diast BP

NPE

Contractility ↓

Progressive disease
Severe PPHN
Progressive Sepsis
Cardiogenic Shock
Pneumothorax

Individualized pathophysiology-based hemodynamic approach

Individualized pathophysiology-based hemodynamic approach

Low diastolic BP

NPE:

Contracility normal

Preload ↓ Afterload ↓

Hypovolemia

Volume expansion
Blood transfusion

Drainage pneumothx
HFVO MAP Reduction

Warm shock

<u>Decreased SVR</u>
Vasopressor (Norepinephrin, vasopressin)

Volume expansion
Inopressor (epinephrin)

Low systolic BP

NPE:

Contractility ↓ Afterload ↑

Cardiogenic s.

<u>Myocardial compromise</u>
Inopressor (epinephrin)

<u>Increased PVR</u>
PPHN - increase iNO

Cold shock

<u>Increased SVR</u>
Inodilatator (milrinone, dobutamine)
Inotrope (epinephrin)

<u>Increased PVR</u>
iNO
Sildenafil
MAP-reduction

Low Systolic/Diast BP

NPE

Contractility ↓

Progressive dis.

<u>Myocardial compromise</u>
Inotrop (epinephrin, milrinone, dobutamin)

<u>Decreased SVR</u>
Vasopressor (Norepinephrin, vasopressin)

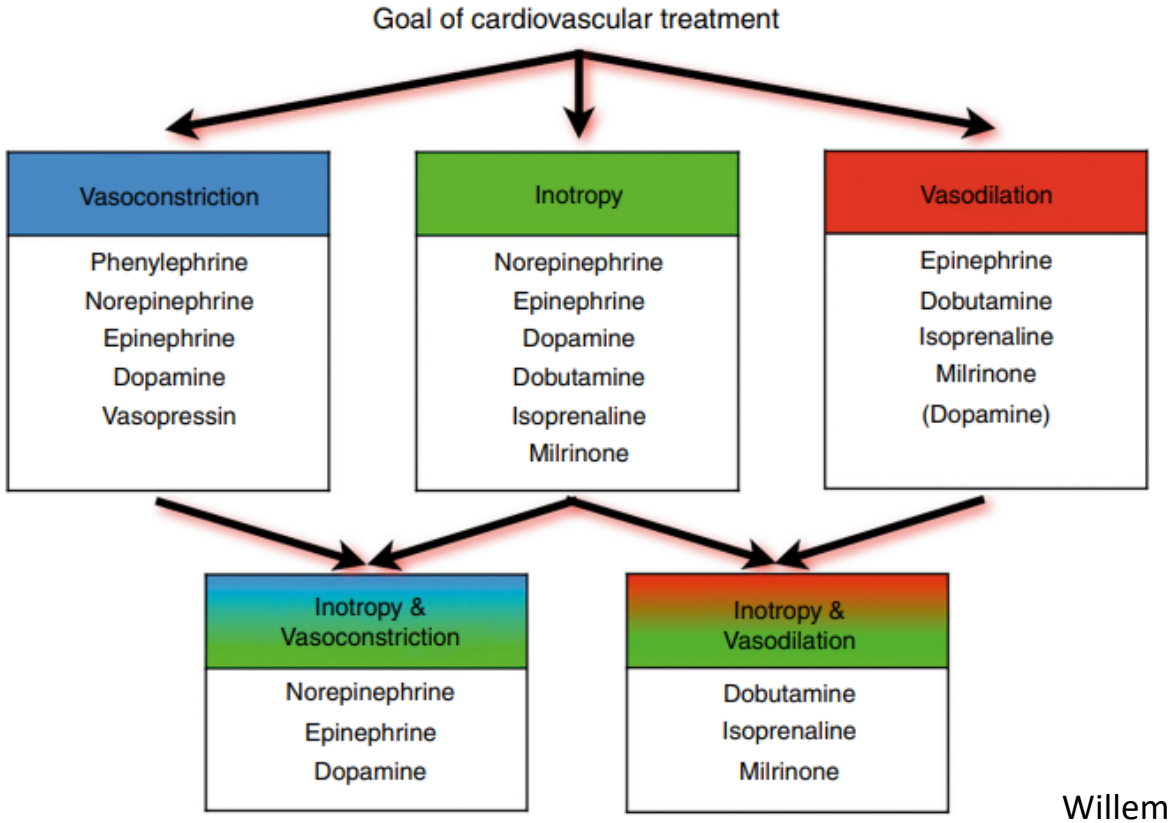
Volume expansion
Inopressor (epinephrin)

<u>Increased PVR</u>
iNO
Sildenafil
MAP-reduction

Managment of neonatal shock:

Volume expansion (isotonic fluids, blood transfusion,..)

Vasopressors | Inotropics | Inodilatators



Willem P. de Boode et al. , PediatricResearch 2018

EAPS 17-20 October, Vienna & Online
The 10th Congress of the EUROPEAN ACADEMY OF PAEDIATRIC SOCIETIES

Back to the intensive care unit

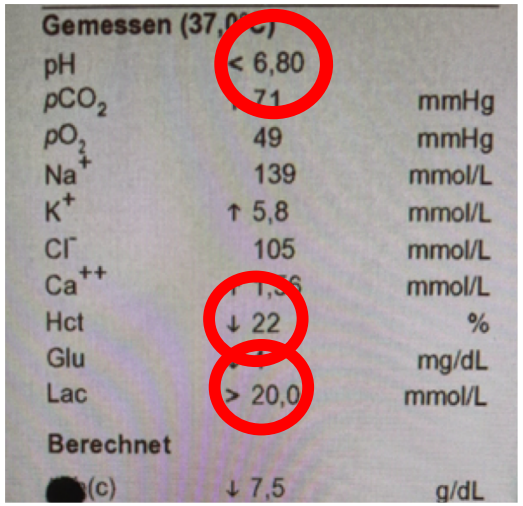
Neonatal shock:

Preterm 25+6 , DOL 1

Emergency delivery, vaginal bleeding

Ventilated:
22/6 cmH2O, 50 / min, 45%,

BP: 47 / **12** (18) mmHg



Low diastolic BP

NPE:
Contracility normal

Preload ↓

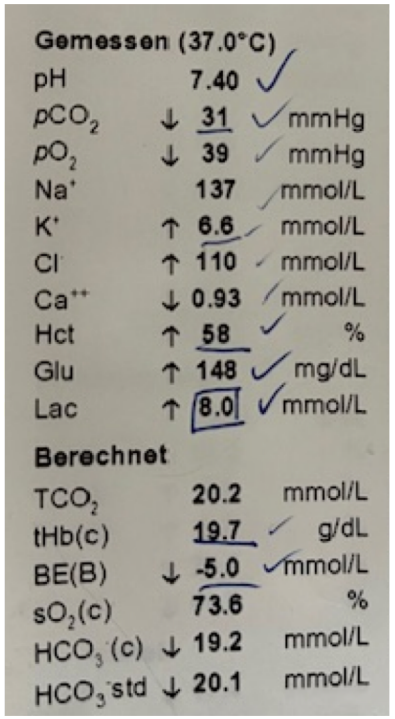
→ Blood transfusion
Volume expansion

Same patient (Preterm 25+6) DOL 5

SIRS, Severe respiratory falliure,

Ventilated: HFOV MAP 12, FiO2 70%, iNO 5 ppm

BP: **39 / 26 (31)** mmHg



Gemessen (37.0°C)			
pH		7.40	
pCO_2	↓	31	mmHg
pO_2	↓	39	mmHg
Na^+		137	mmol/L
K^+	↑	6.6	mmol/L
Cl^-	↑	110	mmol/L
Ca^{++}	↓	0.93	mmol/L
Hct	↑	58	%
Glu	↑	148	mg/dL
Lac	↑	8.0	mmol/L
Berechnet			
TCO_2		20.2	mmol/L
tHb(c)		19.7	g/dL
BE(B)	↓	-5.0	mmol/L
$sO_2(c)$		73.6	%
$HCO_3^-(c)$	↓	19.2	mmol/L
HCO_3^-std	↓	20.1	mmol/L

Low systolic BP

NPE:

RV- Afterload ↑

→ High PVR
Increase iNO
Decrease MAP

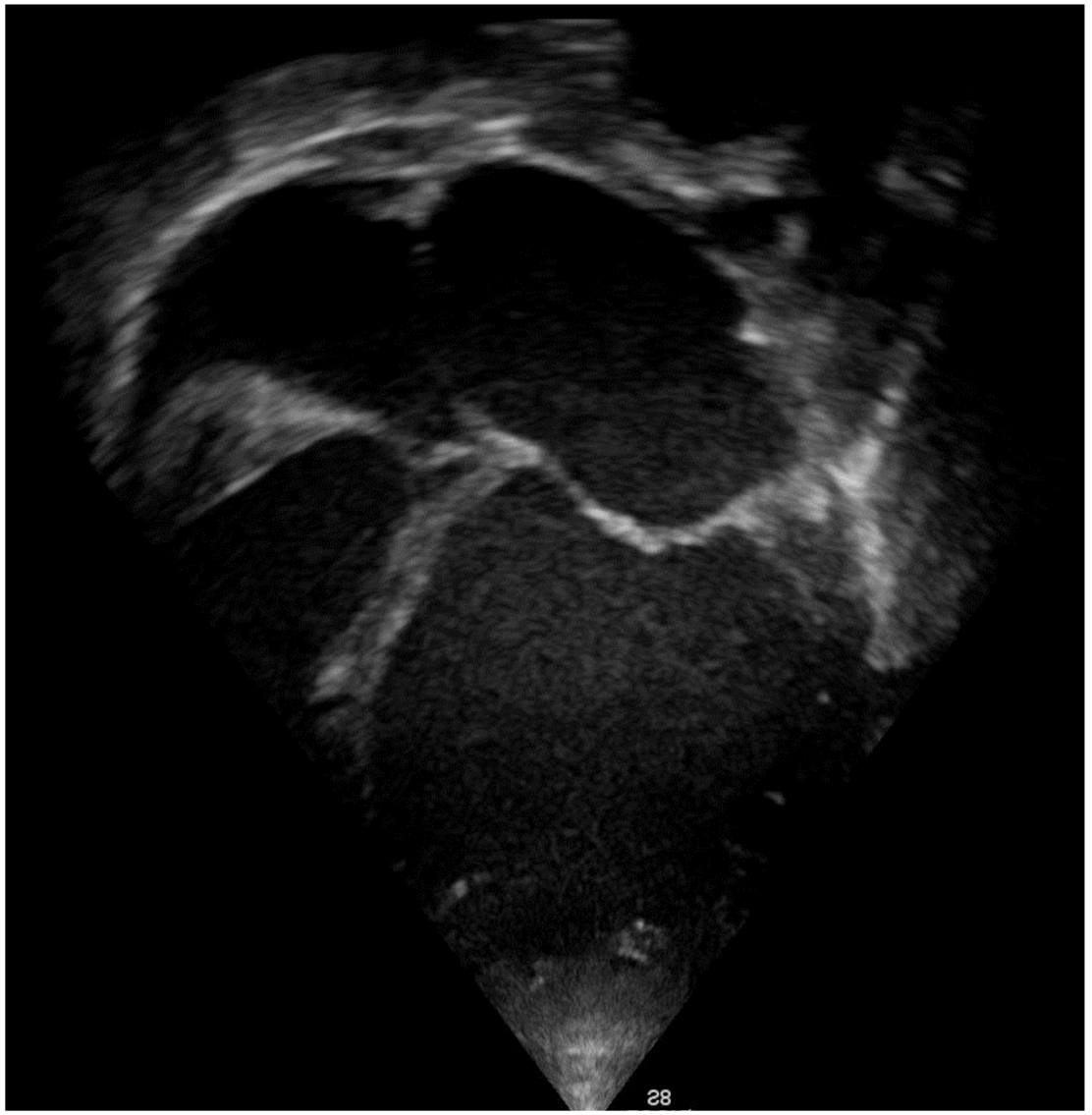
Neonatal shock:

Term Neonate, 2 weeks old

Tachypnea, pale, poor feeding, sweating

Lactate **4,5** mmol/L

BP: 50/27 (41) mmHg



Cardiogenic shock

Low systolic BP

NPE:

Contractility ↓

Consider:

<u>Increased SVR and acceptable BP</u>: Inodilatator (milrinone, dobutamine)
<u>Increased SVR low BP</u>: Inotrope (epinephrin), after stabilisation add Inodilatator

Cardiogenic shock:

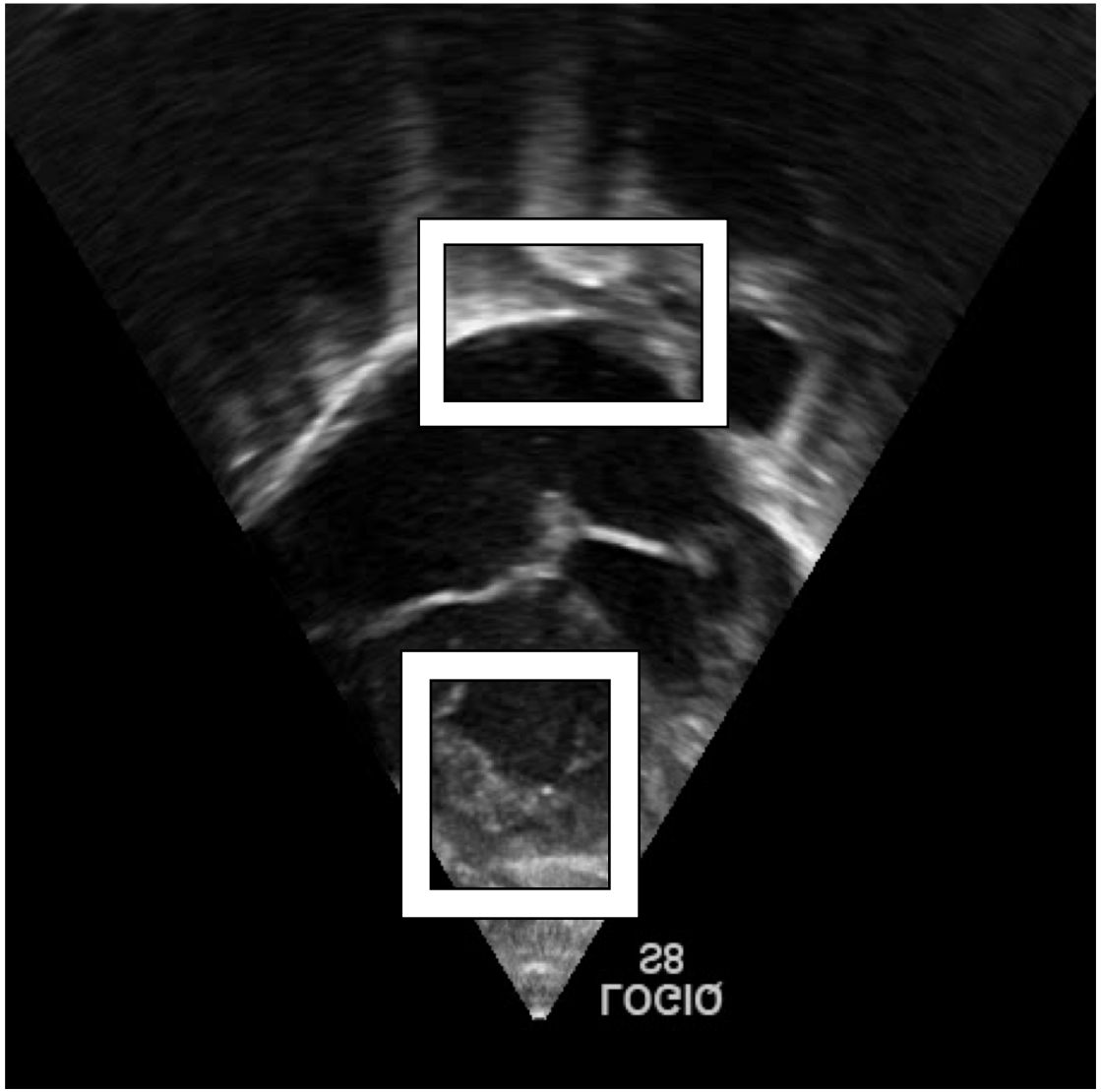
Neonate, 1 hour old:

Pale, SO2 85%, Heart rate 170/min

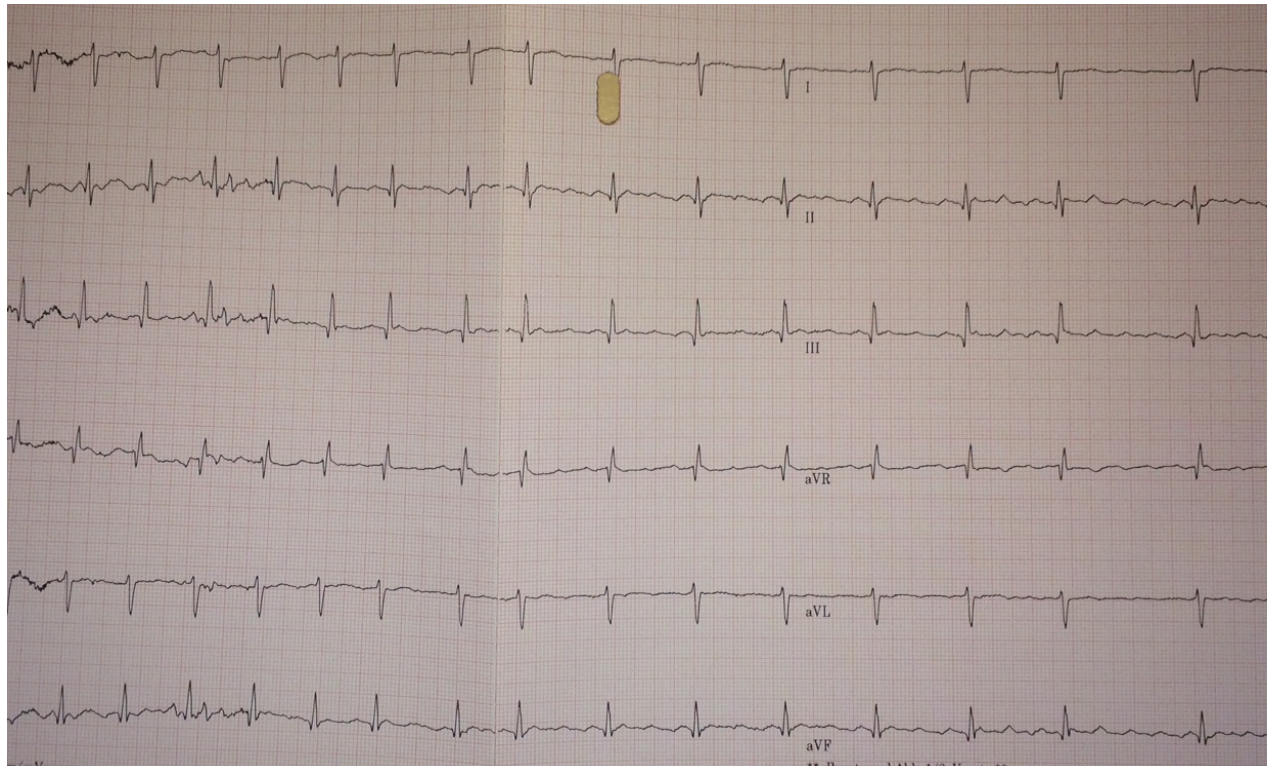
Admission from delivery room

Lactate 3,5 mmol/L

BP: 61/25 (40) mmHg

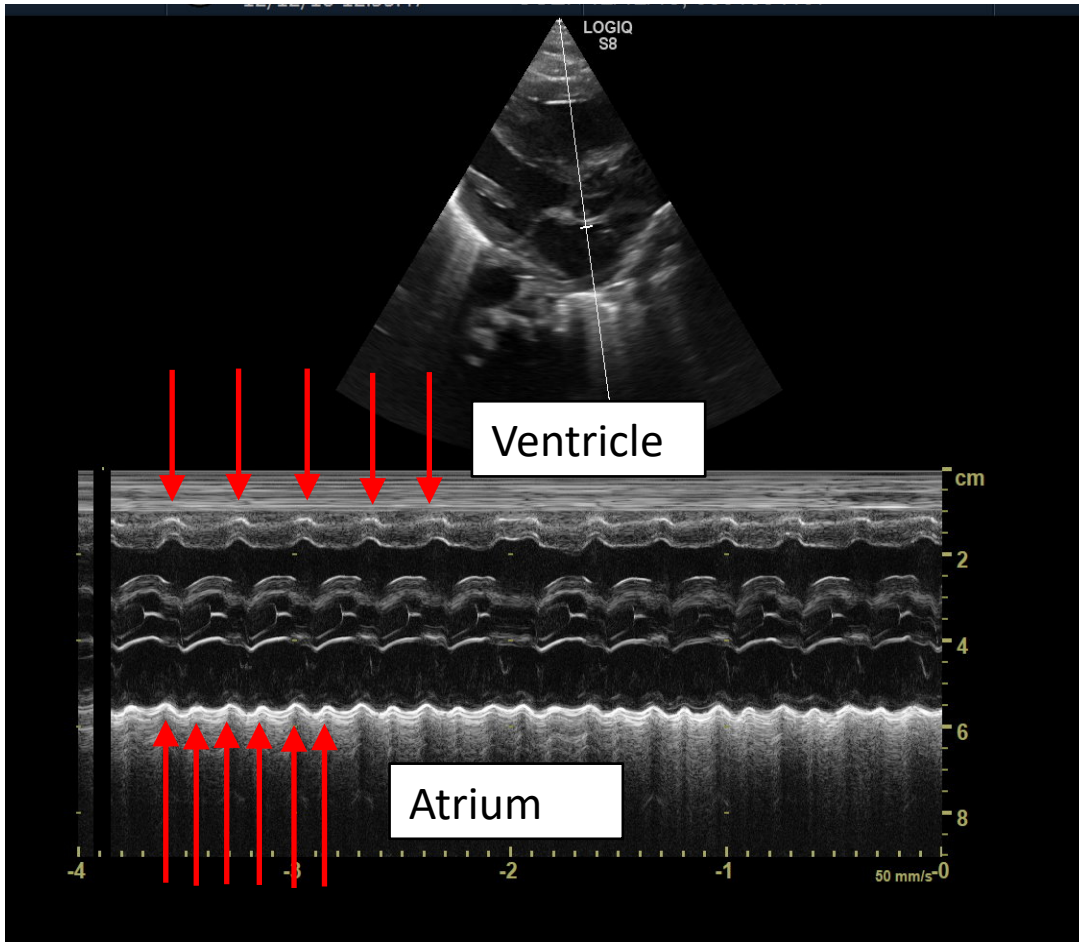


Cardiogenic shock



Management: DC-Cardioversion

Atrial flutter



Neonatal shock

Preterm 23+2 SSW, DOL 65

Sepsis,

Increased O2 demand on CPAP, O2 saturation drops

Laktate 2,8 mmol/l, pale, Capillary refill 3 sec.

BP 51/35 (38) mmHg

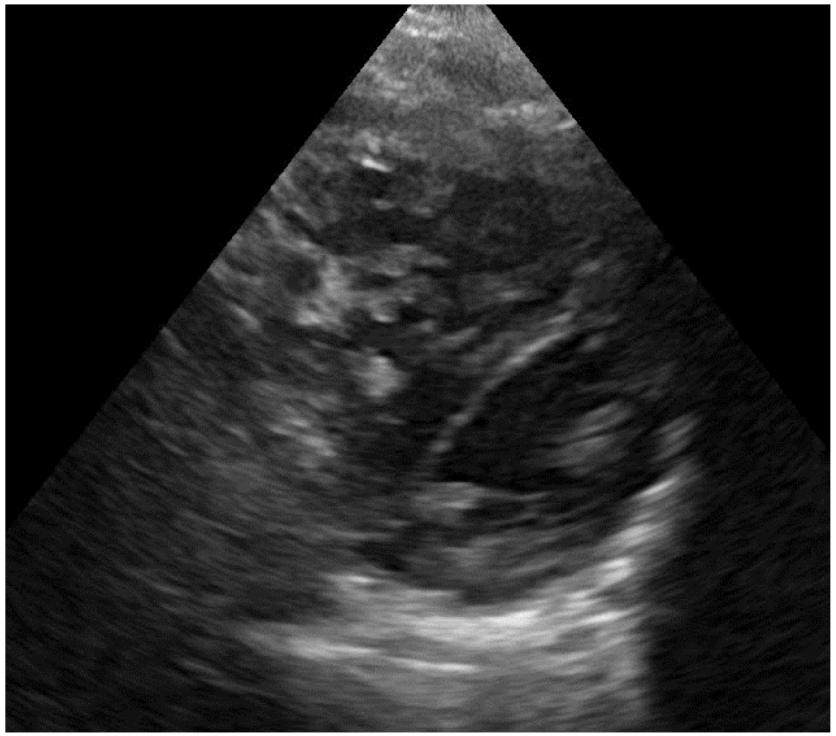
Low systolic BP

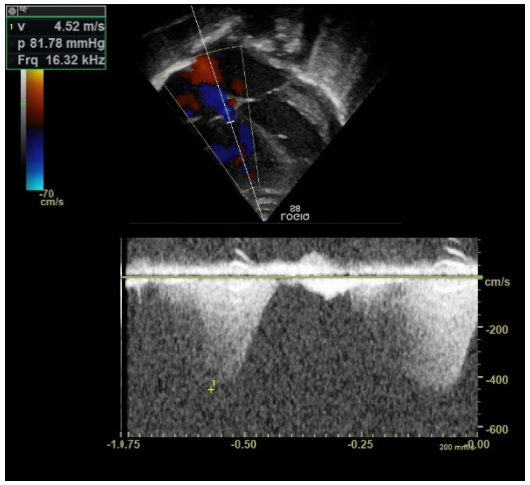
NPE:

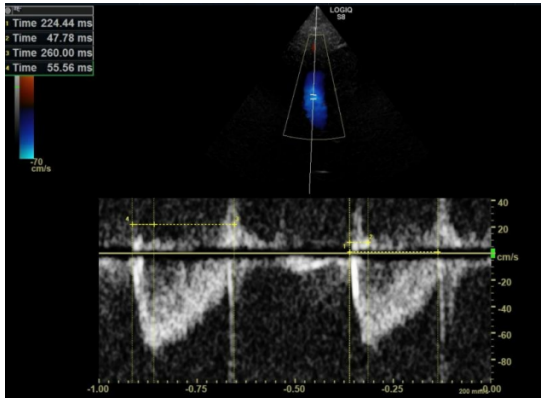
RV- Afterload ↑

Increased PVR

Consider
iNO
Sildenafil
MAP-reduction







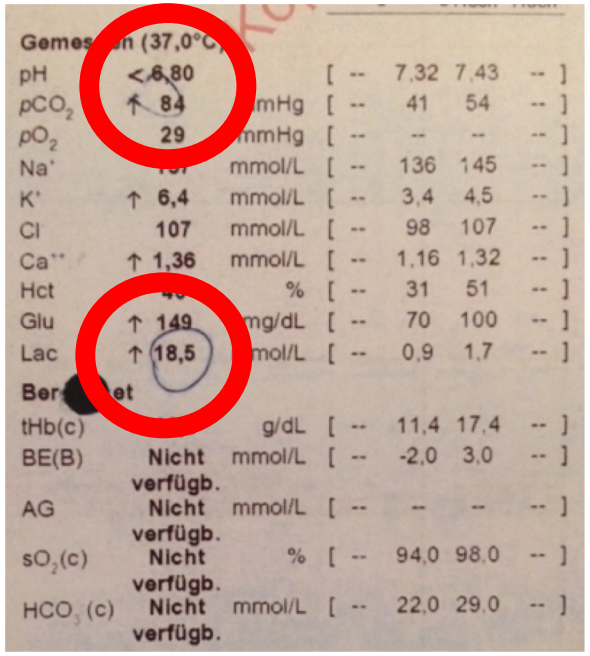
Thank you !

Any Questions ?

Therapy of neonatal shock: I have one more thing !!

Term neonate deteriates 2 hours after birth, tachypnea, shock

BP: not measurable
Capillary refill: 5-6 sec



Gemes[illegible]n (37,0°C)							
pH	< 6,80		[--	7,32	7,43	--]
pCO_2	↑ 84	mHg	[--	41	54	--]
pO_2	29	mmHg	[--	--	--	--]
Na^+	[illegible]	mmol/L	[--	136	145	--]
K^+	↑ 6,4	mmol/L	[--	3,4	4,5	--]
Cl^-	107	mmol/L	[--	98	107	--]
Ca^{++}	↑ 1,36	mmol/L	[--	1,16	1,32	--]
Hct	[illegible]	%	[--	31	51	--]
Glu	↑ 149	mg/dL	[--	70	100	--]
Lac	↑ 18,5	mol/L	[--	0,9	1,7	--]
Ber[illegible]et						
tHb(c)		g/dL	[--	11,4	17,4	--]
BE(B)	Nicht verfügb.	mmol/L	[--	-2,0	3,0	--]
AG	Nicht verfügb.	mmol/L	[--	--	--	--]
$sO_2(c)$	Nicht verfügb.	%	[--	94,0	98,0	--]
$HCO_3^-(c)$	Nicht verfügb.	mmol/L	[--	22,0	29,0	--]

EAPS 17-20 October, Vienna & Online
The 10th Congress of the EUROPEAN ACADEMY OF PAEDIATRIC SOCIETIES

Therapy of neonatal shock: I have one more thing !!

Congenital heart defects with critical reduction of the systemic perfusion

This case: Aortic valve atresia with closing ductus arteriosus

Treatment option: Re-opening the duct - Alprostadil

Take Home Message:

NPE in Neonatal shock

Just do it !

And know the strenghts and problems

Thank you !

Any Questions ?