Pemantauan Hemodinamik dan Obat Penunjang Kardiovaskular

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OBJEKTIF

- Mengetahui hemodinamik normal pada neonatus
- Mengetahui faktor yang berperan dalam stabilitas hemodinamik
- Mengetahui berbagai parameter untuk memonitor hemodinamik pada neonatus
- Mengetahui peran obat penunjang kardiovaskular dalam mempertahankan stabilitas hemodinamik

PENDAHULUAN

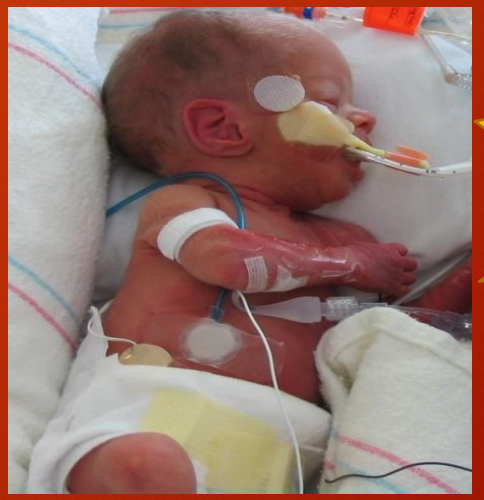
Sirkulasi yg baik → distribusi O2, nutrisi, & ekskresi baik → kelangsungan fungsi organ yang baik

Gangguan hemodinamik (Syok)

Tubuh → kompensasi dg redistribusi aliran darah ke organ vital

Pentingnya monitoring hemodinamik dini untuk mendeteksi instabilitas hemodinamik tahap awal → intervensi lebih cepat dilakukan → komplikasi & sekuele dapat dihindari atau diminimalisasi

Gangguan hemodinamik lebih lanjut → dekompensasi



Instabilitas hemodinamik → kegagalan fungsi organ

Apakah kita hanya menunggu & baru menyadari setelah gejala jelas?

Faktor yang mempengaruhi stabilitas **Hemodinamik**

- Usia gestasi saat lahir
- Asfiksia
- Infeksi perinatal
- Malformasi kongenital

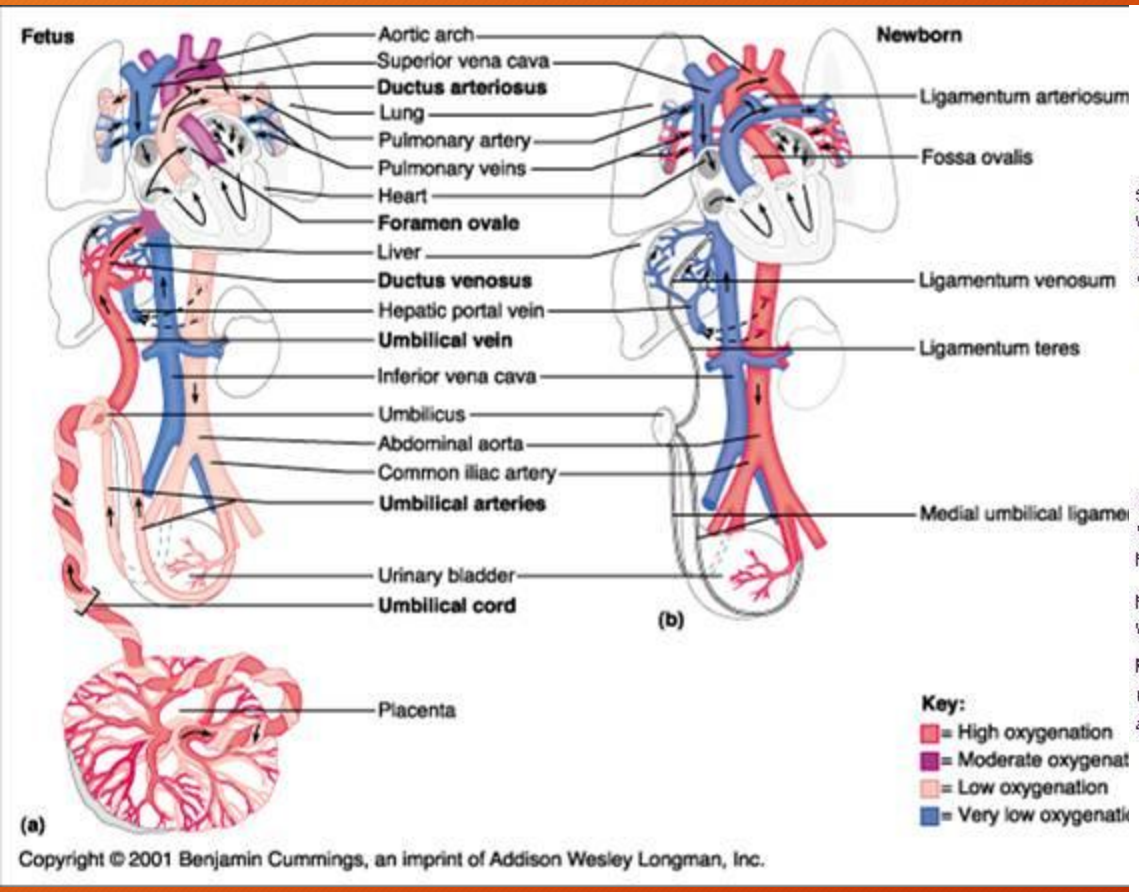
Faktor intrinsik

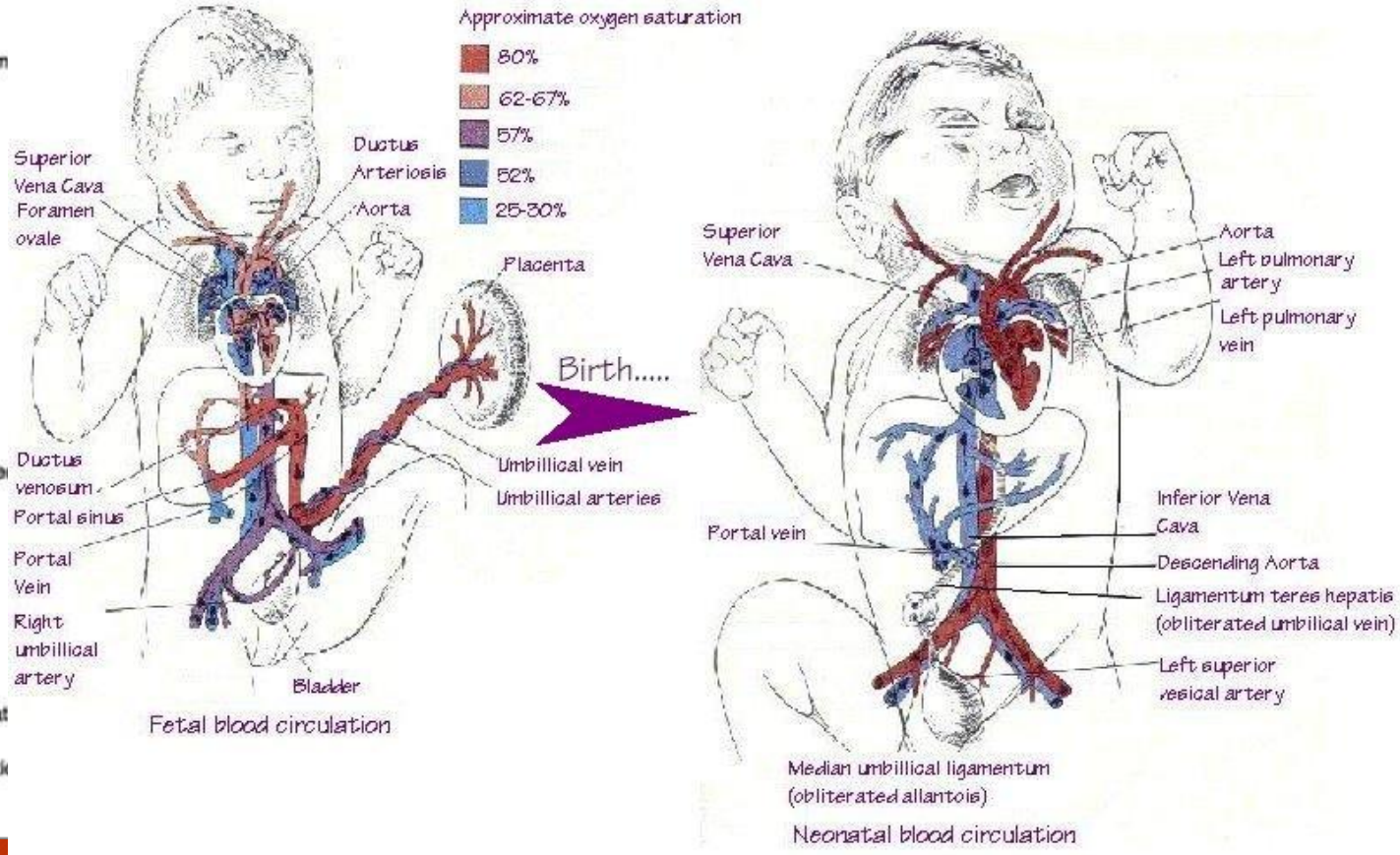
Faktor ekstrinsik

- Komplikasi / penyulit pada kehamilan
- Komplikasi / penyulit pada persalinan
- Obat yang dikonsumsi ibu
- *Timing of cord clamping*

Pendahuluan

Masa transisi sirkulasi fetus – neonatus

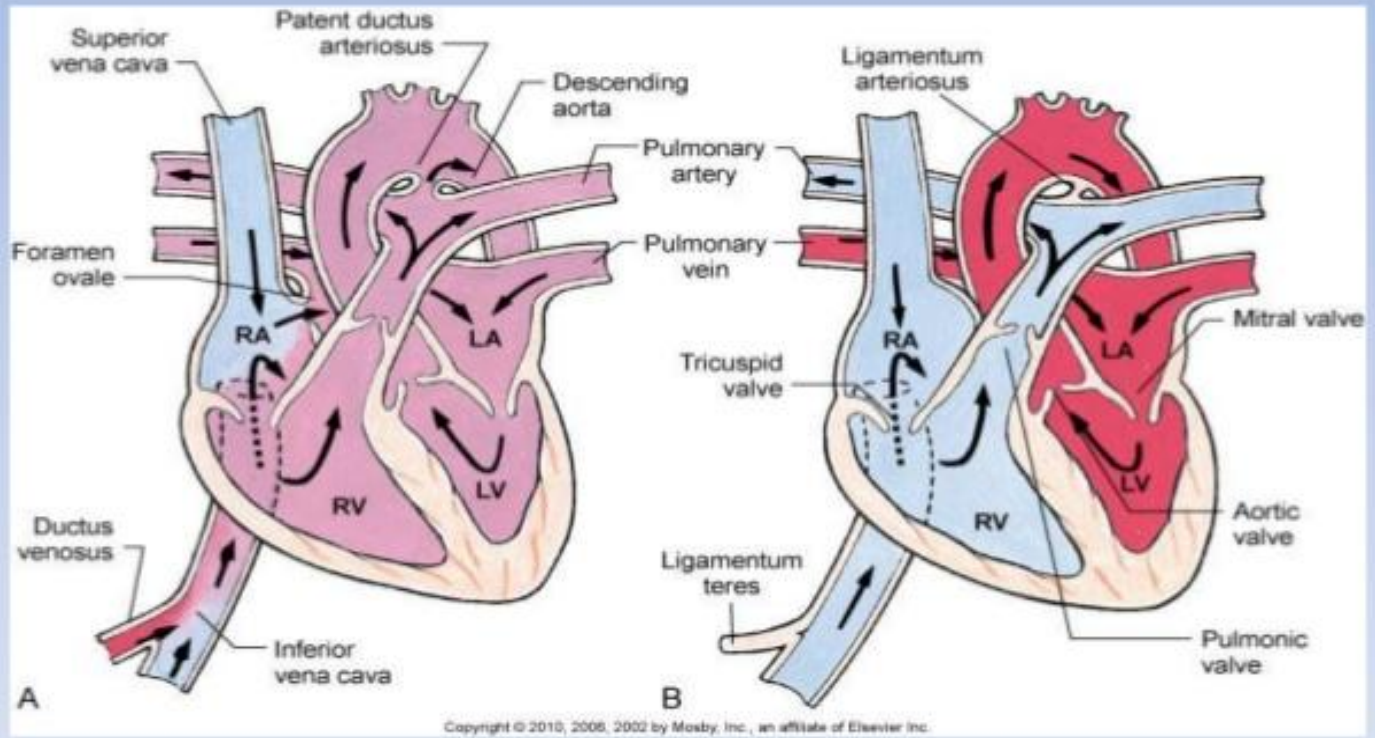




Events that occur at birth

Pendahuluan

The Normal Fetal and Newborn Heart



Fetal

Newborn

Faktor yang berperan dalam stabilitas hemodinamik

Figure 9- ressure

Resistensi vaskular

$$BP = SVR \times CO$$

Vasodilatasi
→ SVR ↓

Anaphylactic Shock
Neurogenic Shock
Septic Shock
Vasodilator Drug Induced Shock

$$HR \times SV$$

Denyut jantung

Tachycardia Extremes
Bradycardia Extremes

Kontraksi

$$EDV - ESV$$

V preload

V preload↓
→ EDV ↓

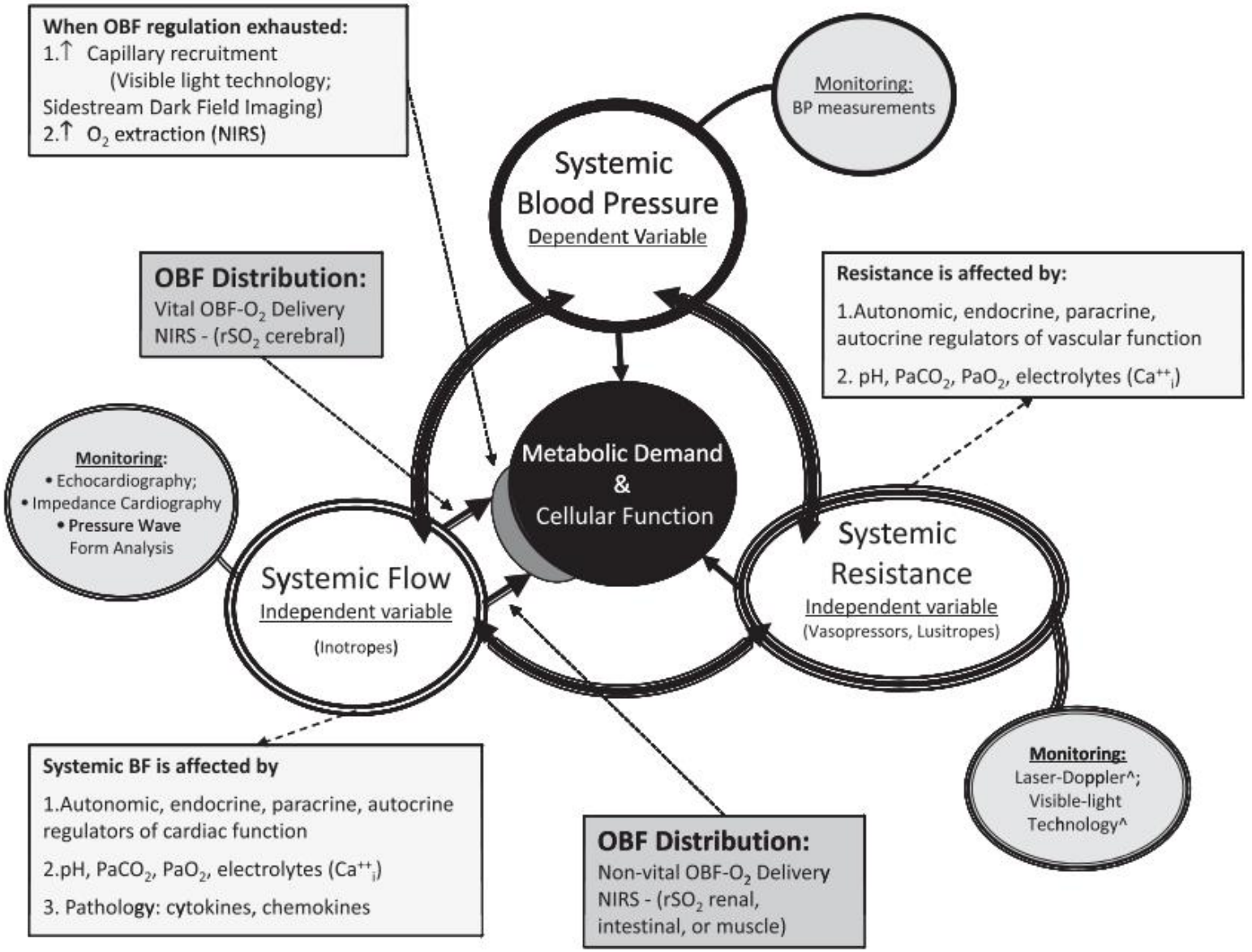
Hypovolemic Shock

Kontraksi ↓
→ ESV ↑

Cardiogenic Shock

BP = blood pressure, SVR = systemic vascular resistance, HR = heart rate, SV = stroke volume, EDV = end diastolic volume (i.e. preload), ESV = end systolic volume (i.e. contractility)

Boville B et al. Pediatric Cardiopulmonary Care. US: Lifesciences Corp; 2011



Interaction among and monitoring of blood pressure (BP), blood flow, blood flow distribution and vascular resistance

Journal of Perinatology (2010) 30, S38–S45; doi:10.1038/jp.2010.101

Fase Shok	Patofisiologi	Gejala & Tanda
Syok terkompensasi	Tahap ini: mekanisme kompensasi tubuh → mempertahankan fungsi organ • Redistribusi aliran darah ke organ vital (otak, ginjal, & adrenal) dg mekanisme regulasi vasomotor (vasodilatasi di organ vital & vasokonstriksi di organ lain) • ↑kontraktilitas miokard • Ginjal → ↓ekskresi air & garam	Tahap ini: gejala dapat tidak nyata • denyut nadi ↑ • Tekanan nadi menyempit • Akral mulai dingin • CRT ≥3s • Tekanan darah N • Diuresis ↓
Syok dekompensasi	• Mekanisme kompensasi gagal → sirkulasi & O_2 ke organ vital↓ • Metabolisme anaerob ↑ → produksi laktat ↑ → asidosis metabolik • Asidosis → mengganggu kontraktilitas miokard • Asidosis → mengganggu pompa elektrolit → kebocoran cairan ke extraseluler • Mediator inflamasi ↑ → ↓ perfusi jaringan	• Denyut nadi ↑↑ • Akral dingin • CRT > 3s • Tekanan darah ↓ • Diuresis ↓↓ / (-)

SYOK IREVERSIBEL

Choudhory P, et al. Principles of pediatric and neonatal emergencies. 3rd ed. Delhi: JP Medical; 2011

Monitoring Hemodinamik

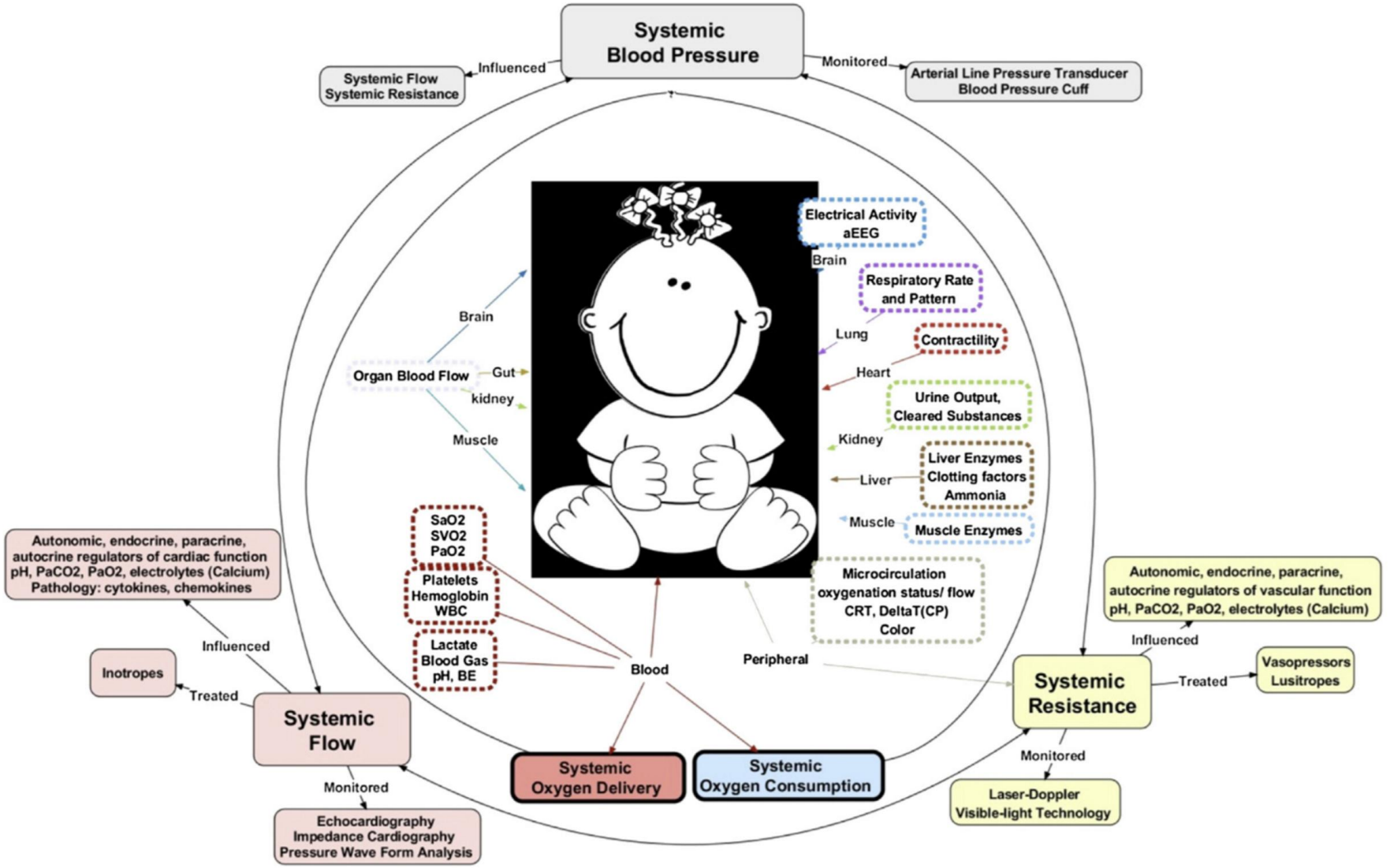


Figure 2. Tools for a global (outside circles) and regional (inside the circles) assessment of developmental hemodynamics. Global monitoring of the relationships between systemic flow, blood pressure and resistance and arterial/venous oxygen content provides information on systemic oxygen delivery and consumption. Regionally monitored parameters provide direct or indirect information on specific organ blood flows, function and vital or non-vital blood flow regulatory assignments. aEEG, amplitude-integrated electroencephalography; CRT, capillary refill time; BE, base excess; SaO_2, arterial oxygen saturation; SVO_2, venous oxygen saturation; $PaCO_2$, arterial partial pressure of carbon dioxide; PaO_2, arterial partial pressure of oxygen; WBC, white blood cells. Modified with permission from Soleymani et al. [1].

Azhibekov T et al. Transitional cardiovascular physiology and comprehensive hemodynamic monitoring in the neonate: relevance to research and clinical care. Seminars in Fetal & Neonatal Medicine 2014; 19: 45-53

Conventional assessment

Blood pressure, heart rate, O_2 saturation

Indirect clinical and laboratory signs of CV function (Uv, CRT, ΔT_{C-P}, BD, serum lactate)

Systemic (rapidly dispersed periodic variable) and organ blood flow using

Echocardiography (LVO, RVO, SVC flow, PAP, EF, SF, MPI, VTI, PI, RI, etc.)

Dilution techniques, direct and modified Fick methods, arterial ultrasound probes, arterial pressure waveform analysis, impedance cardiography, electrical cardiometry

NIRS, laser or side-stream dark field-imaging technology[a]

O_2 delivery and consumption in tissues

Continuous wave differential or spatially resolved NIRS for CBF (ΔCBV, absolute CBF, CFOE, TOI[b])

Spatially resolved NIRS for cerebral, renal, intestinal and muscle oxygenation ($[rSO_2]$[b])

Visible light technology (mucosal or skin tissue capillary HgB saturation; StO_2)

Functional assessment (aEEG for brain activity)

Data collection requires use of real-time data acquisition systems

Assessment of cardiovascular function, organ perfusion, O_2 delivery and brain function in neonates at the bedside

Journal of Perinatology (2010) 30, S38–S45; doi:10.1038/jp.2010.101

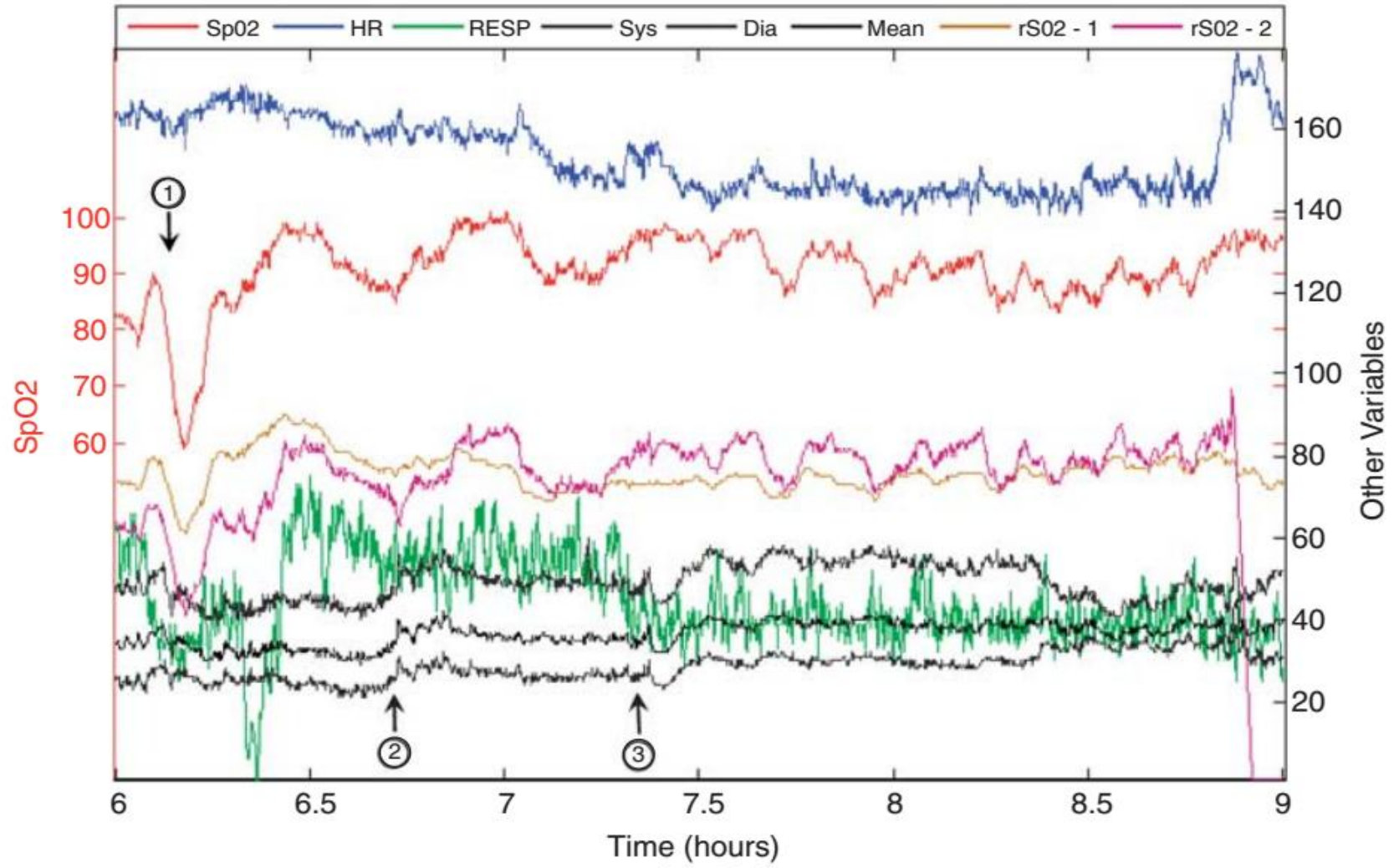


Figure 2 Selected hemodynamic parameters recorded by the monitoring tower in a 26 weeks' gestation extremely preterm neonate during the first day after delivery. Hemodynamic parameters continuously monitored by the hemodynamic monitoring tower in the 26 weeks' gestation, <1-day-old extremely preterm neonate included arterial oxygen saturation (SpO_2), heart rate (HR), respiratory rate (RESP), systolic (Sys), diastolic (Dia) and mean (Mean) BP and cerebral (rSO_2-1) and renal (rSO_2-2) mixed venous tissue oxygen saturation using near infrared spectroscopy. These parameters are depicted on the y −axis, whereas age after delivery in hours is shown on the x axis.

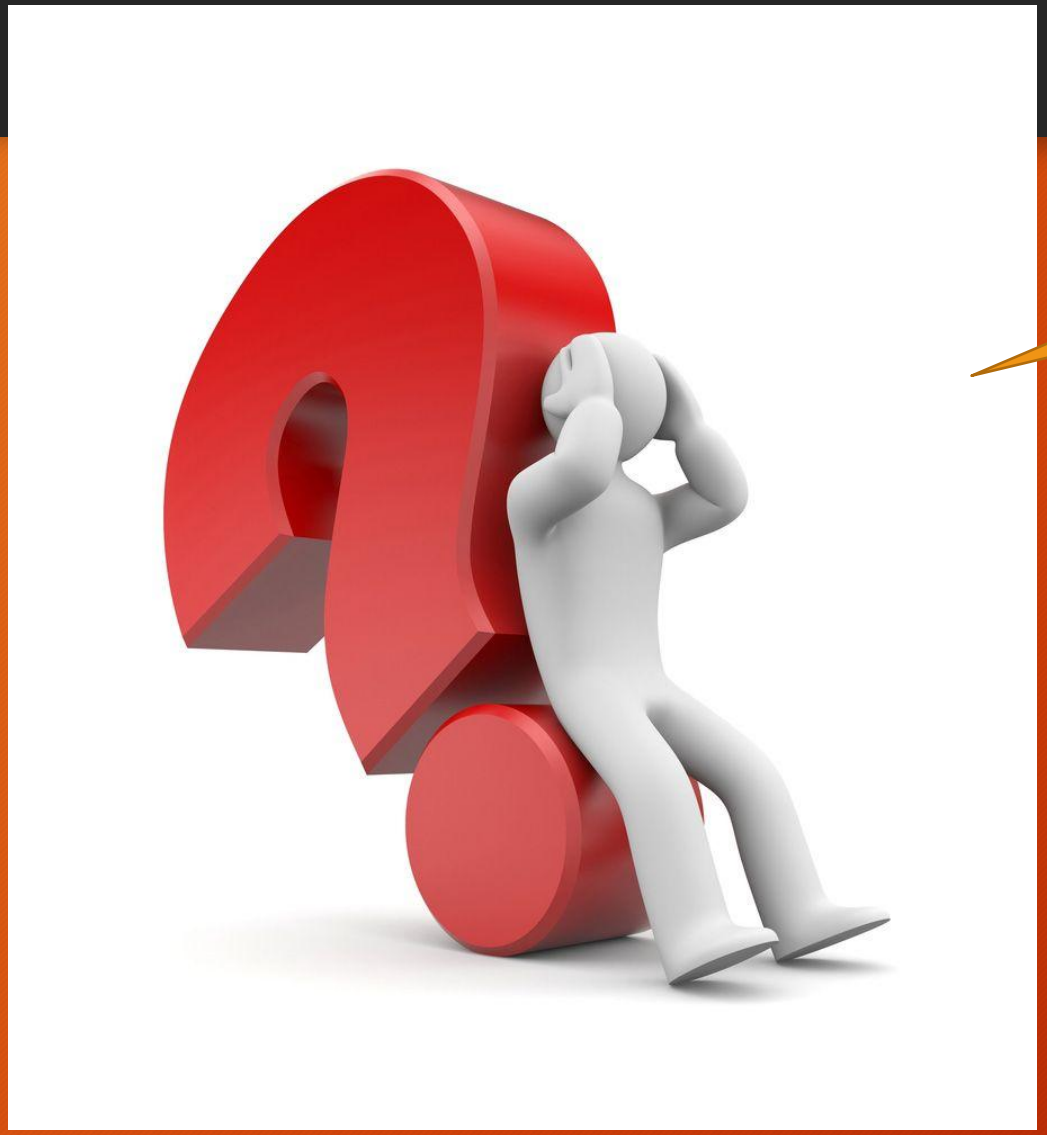
Journal of Perinatology (2010) 30, S38–S45; doi:10.1038/jp.2010.101

Table 1
The most frequently monitored systemic and regional hemodynamic parameters in neonates.[a]

	Parameter	Technology/method	Purpose and acquisition (C, I or C/I)
Systemic perfusion (BP and CO)	Heart rate	Electrocardiography (electrodes)	In conjunction with stroke volume give flow status (C)
	BP	Arterial line/cuff (oscillometry; Doppler ultrasound)	Perfusion pressure (C/I)
	Stroke volume/ CO	Echocardiography	Systemic, pulmonary (CO) and organ blood flow, cardiac function (I)
		Impedance electrical cardiometry	Systemic blood flow (CO) (C)
Systemic oxygenation	SpO_2	Pulse oximetry	Oxygenation on the arterial side (C)
CO_2 status	TCOM	CO_2 diffusion through skin	Effect on cerebral vasculature (changes in CBF) (C)
Regional perfusion	Regional O_2 saturation	Near-infra-red spectroscopy	Tissue oxygenation and (indirectly) organ perfusion (C)
Peripheral perfusion	Microcirculation (oxygenation; blood flow velocity; capillary recruitment)	Visible light technology	Peripheral perfusion (C)
		Laser Doppler flowmetry	Peripheral perfusion (C)
		OPS and SDF	Peripheral perfusion (C)
Indirect assessment of perfusion	Capillary refill time	Visual	Indirect clinical sign of systemic perfusion (I)
	Delta T (C—P)	Temperature	Indirect clinical sign of systemic perfusion (I)
	Color	Visual	Peripheral perfusion (I)
Organ function	Brain electrical activity	aEEG	Assessment of brain activity (C)
	Urine output	Urinary catheter	Assessment of renal function (I)

BP, blood pressure; CO, cardiac output; SpO_2, arterial oxygen saturation; aEEG, amplitude-integrated electroencephalography; TCOM, transcutaneous CO_2 monitoring; CBF, cerebral blood flow; OPS, orthogonal polarization spectral (imaging); SDF, side-stream dark-field imaging; delta T (C—P), difference between core and peripheral temperatures.
Methods used to monitor different components of systemic perfusion (BP and CO) and oxygenation, carbon dioxide production and elimination, regional (organ) and peripheral (microcirculation) perfusion and organ function (aEEG) along with the indirect methods used in the clinical practice to assess perfusion and organ function. Acquisition may be continuous (C), intermittent (I) or both (C/I).
[a] Modified with permission from Soleymani et al. [1].

Azhibekov T et al. Transitional cardiovascular physiology and comprehensive hemodynamic monitoring in the neonate: relevance to research and clinical care. Seminars in Fetal & Neonatal Medicine 2014; 19: 45-53



Begitu banyak parameter. Parameter mana yang paling baik??

Parameter	
Denyut nadi	Terdapat korelasi tidak signifikan antara denyut nadi & aliran darah sistemik (*SBF-Systemic Blood Flow*) – *Kluckow M et al. Low superior vena cava flow and intraventricular hemorrhage in preterm infants. Arch Dis Child Fetal Neonatal Ed 2000;82:F188-94*
Warna	Terdapat ketidaksepakatan antar observer ketika diperlihatkan video mengenai kapan bayi berwarna pink, dimana nilai SpO2 divariasikan – *O Donnel CP et al. Clinical Assessment of infant colour at delivery. Arch Dis Child Fetal Neonatal Ed 2007;92:F465-7*
CRT	Terdapat keseragaman nilai CRT antarobserver ketika melakukan CRT di area dada, namun tidak seragam ketika dilakukan di dahi, lengan, tumit. – *Raichur DV et al. Capillary refill time in neonates: beside assessment. Indian J Pediatr. 2001 Jul; 68(7):613-5* CRT > 3s memiliki 55% sensitivitas & 80% spesifisitas utk memprediksi SBF rendah – *Osborn DA et al. Clinical detection of upper body blood flow in very premature infants using blood pressure, capillary refill time and central-peripheral temperature difference. Arch Dis Fetal Neonatal Ed 2004;89:F168-73*

Parameter	
Diuresis	Terdapat korelasi tidak signifikan antara diuresis, CRT, dan SBF rendah – *Miletin J et al. Bedside detection of low systemic flow in the very low birth weight infant on day 1 of life. Eur J Pediatr 2009; 168:809-13*
Serum laktat	Serum laktat kadar ≥2.8 mmol/L mempunyai 100% sensitivitas & 60% spesifitas utk mendeteksi SBF rendah - *Miletin J et al. Bedside detection of low systemic flow in the very low birth weight infant on day 1 of life. Eur J Pediatr 2009; 168:809-13*
Perbedaan antara temperature sentral & perifer (CPTd)	Tidak ada korelasi antara CPTd dengan SBF - *Osborn DA et al. Clinical detection of upper body blood flow in very premature infants using blood pressure, capillary refill time and central-peripheral temperature difference. Arch Dis Fetal Neonatal Ed 2004;89:F168-73*
Tekanan darah (TD)	Terdapat korelasi buruk antara tekanan darah dengan SBF pada bayi premature < 30 minggu - *Osborn DA et al. Clinical detection of upper body blood flow in very premature infants using blood pressure, capillary refill time and central-peripheral temperature difference. Arch Dis Fetal Neonatal Ed 2004;89:F168-73*

Combination of different clinical hemodynamic variables

Table 1
Overview of predictive values of different clinical hemodynamic variables.

Variable	Cut-off value	Population	Definition low SBF	Se	Sp	PPV	NPV	Reference
Blood pressure								
MBP	<30 mm Hg	Preterms, PDA included	LVO <150 ml kg^{-1} min^{-1}	42%	79%	44%	78%	Kluckow, 1996 [34]
MBP	<30 mm Hg	Preterms, PDA excluded	LVO <150 ml kg^{-1} min^{-1}	38%	93%	75%	73%	Kluckow, 1996 [34]
MBP	< 30 mm Hg	Preterms	LVO <10th percentile	57%	50%	24%	81%	Pladys, 1999 [9]
MBP	< 30 mm Hg	Preterms	LVO <150 ml kg^{-1} min^{-1}	100%	54%	18%	100%	Pladys, 1999 [9]
MBP	< 30 mm Hg	Preterms, PNA 5 h	SVC flow <30 ml kg^{-1} min^{-1}	80%	60%	17%	97%	Kluckow, 2000 [8]
MBP	< 30 mm Hg	Preterms, PNA <12 h	SVC flow <41 ml kg^{-1} min^{-1}	59%	77%	36%	90%	Osborn, 2004 [6]
MBP	< GA (wks)	Preterms, PNA <12 h	SVC flow <41 ml kg^{-1} min^{-1}	30%	88%	71%	85%	Osborn, 2004 [6]
Urine output/renal function								
Rise in blood potassium	>0.12 mmol/h	Preterms, PNA <12 h	SVC flow	35%	NA	93%	NA	Kluckow, 2001 [17]
Capillary refill time (CRT)								
CRT	≥3 s	Preterms, PNA <12 h	SVC flow <41 ml kg^{-1} min^{-1}	55%	80%	33%	91%	Osborn, 2004 [6]
CRT	≥4 s	Preterms, PNA <12 h	SVC flow <41 ml kg^{-1} min^{-1}	29%	96%	55%	88%	Osborn, 2004 [6]
Blood lactate concentration								
Lactate	>2.8 mmol/l	VLBW, PNA <12 h	SVC flow <40 ml kg^{-1} min^{-1}	100%	60%	NA	NA	Miletin, 2009 [28]
Central–peripheral temperature difference (CPTd)								
CPTd	≥2 °C	Preterms, PNA <12 h	SVC flow <41 ml kg^{-1} min^{-1}	40%	69%	23%	83%	Osborn, 2004 [6]
Combination of clinical variables								
MBP and/or CRT	MBP<30 mm Hg CRT≥3 sec	Preterms, PNA <12 h	SVC flow<41 ml kg^{-1} min^{-1}	78%	63%	31%	NA	Osborn, 2004 [6]
CRT and lactate	CRT≥4 sec Lact >4 mmol/l	VLBW, PNA <12 h	SVC flow <40 ml kg^{-1} min^{-1}	50%	97%	80%	88%	Miletin, 2009 [28]

CRT: capillary refill time; CPTd: central–peripheral temperature difference; MBP: mean blood pressure; NA: not available; LVO: left ventricular output; NPV: negative predictive value; PDA: persistent ductus arteriosus; PNA: postnatal age; PPV positive predictive value; SBF: systemic blood flow; Se: sensitivity; Sp: specificity; SVC: superior vena cava; VLBW: very low birth weight.

de Boode. Clinical monitoring of systemic hemodynamics in critically ill newborns. Early Human Development 86 (2010) 137–141

METODE MONITORING HEMODINAMIK

Klinis / Lab

Klinis

- Tanda & gejala →
  - Warna, CRT, CPTd, status mental
- Tanda2 vital
- Diuresis

Lab

- AGD
- Serum laktat

Non klinis / Lab

Noninvasive

- Capnografi
- Parameter ventilator
- Transcutaneous O2
- Pulse oximetry
- ECG
- Echocardiography
- NIRS (Near Infrared Spectroscopy)
- USCOM (Ultrasound Cardiac Output Monitor)

Invasive

- Arterial cannulation
- Central venous cannulation
- Pulmonary artery cathether
- PCCO (Pulse index Contour Continuous Cardiac Output)

Advanced

- fMRI
- fECHO
- IEC (impedance electrical cardiometry)

Obat Penunjang Kardiovaskular

Table 5: Recommended management of neonatal shock

A. Early detection	Early recognition and intervention are crucial for favourable outcomes.
B. Aggressive fluid therapy	Mortality is significantly reduced if hemodynamic function is optimized early.[16] There is no advantage in using crystalloids instead of colloids in septic shock.[17] Intraventricular haemorrhage and infection transmission is lower with crystalloids. The incidence of pulmonary edema is less with 5% albumin.[18] Bolus resuscitation as a life-saving intervention in shock without hypotension is challenged. Infants who do not diurese after adequate fluids may need diuretics to prevent fluid overload.[14]
C. Antibiotics	Blood cultures, biochemical markers for sepsis, blood glucose and ionized calcium should be taken before initiating antibiotics for suspected sepsis.[19] Ampicillin plus gentamycin is more effective than cefotaxime plus gentamycin.[20] Cefotaxime is preferred for meningitis.
D. Respiratory support	Respiratory failure accompanying shock requires elective ventilation. Anoxia and over-distension of alveoli- a potent IL-6 inducer should be avoided
E. Metabolic support	There is no consensus on ideal blood sugar but it should not be lower than 30 mg/dL.[21] Level of 175 mg/dL or more has a 2.5X increased mortality; same in ELBW babies with level above 150 mg/dL. Insulin should be used only when sugar level exceeds 180mg/dL in refractory shock and unfavourable response newborn.[22] There is no evidence to support bicarbonate therapy in acidemia of septic shock. Hypocalcemia is a reversible cause of cardiac dysfunction; it should be normalized. Corticosteroids often used in septic shock when volume expansion and inotropes are unable to raise BP, appear to increase mortality in a subset of patients.[23] Consequently, corticosteroids are recommended for refractory shock when adrenal insufficiency is suspected.[24]
F. Nutrition	In infants with poor muscle mass and energy reserves, metabolic requirements increase due to hypercatabolic state in sepsis. Appropriate enteral feeding to reduce bacterial translocation from gut mucosa and preserve gut mucosal function is advocated.
G. Cardiovascular support	Inotropes like dopamine, dobutamine, epinephrine and norepinephrine are indicated via iv or io route before central access is achieved when myocardial contractility remains poor despite adequate volume replacement. Delay increases mortality 20-fold.[25] Epinephrine and norepinephrine raise mean arterial pressure but epinephrine causes adverse hyperglycemia requiring insulin, increased plasma lactate and inadequate gastric mucosa perfusion.[26] Dopamine is the first line drug although dobutamine raises systemic blood flow more effectively.[27] It reduces TSH release making hypothyroidism diagnosis difficult. The best vasoactive drug schedule for premature transition shock is low dose dopamine and dobutamine, Epinephrine is a potent inotrope and chronotrope, and a systemic and pulmonary vasodilator. Norepinephrine is indicated for "warm" shock in neonates.

Sinniah et al. Shock in the neonate. IeJSME 2013; 7(12): 17-28

Dopamin

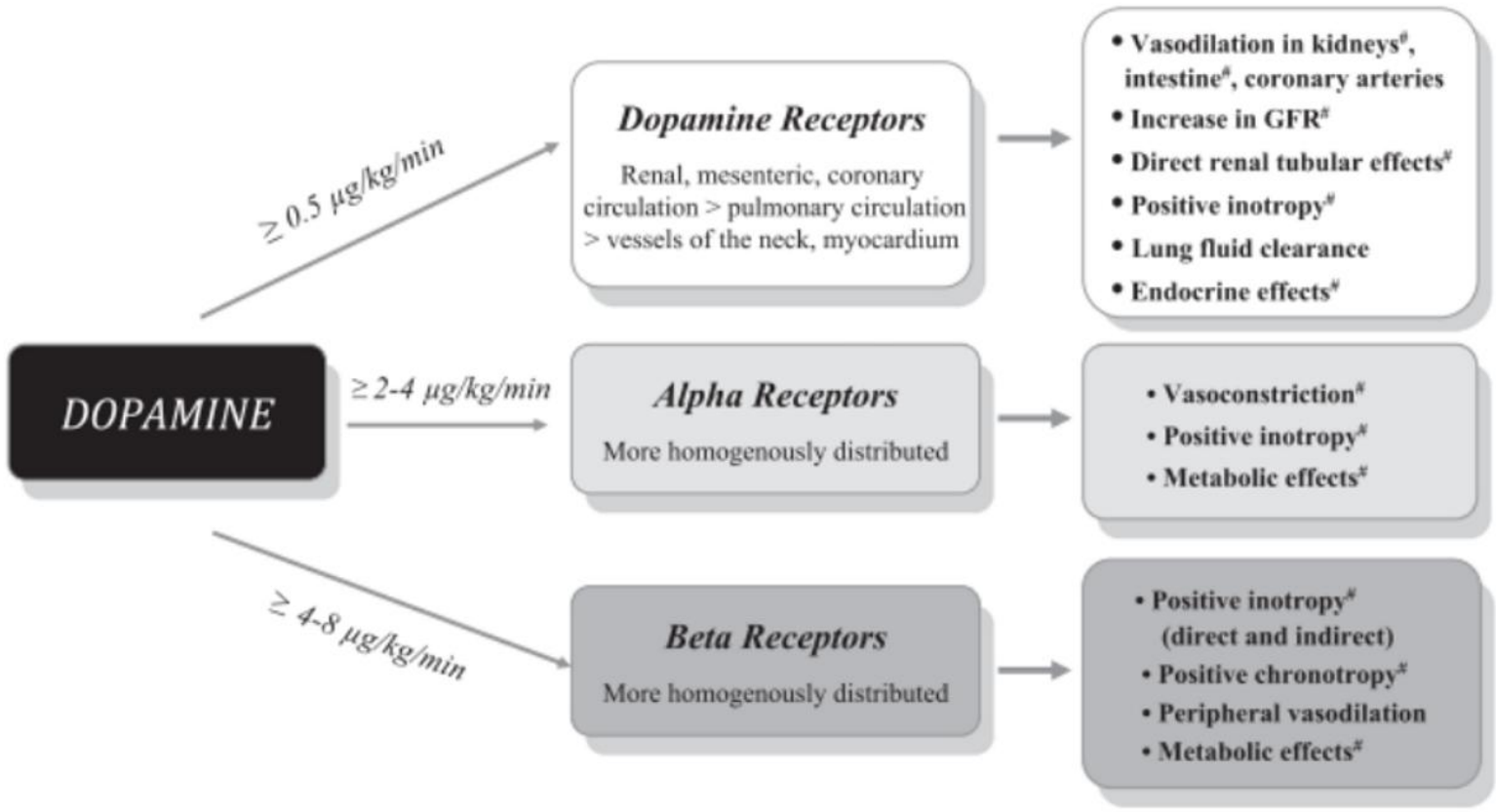


Fig. 3. Dose-dependent cardiovascular, renal, and endocrine effects of dopamine in neonates. Receptor-specific hemodynamic, renal, pulmonary, and endocrine actions of dopamine are shown in the absence of adrenoreceptor downregulation (#denotes the effects demonstrated in preterm neonates). (*Modified from* Seri I. Management of hypotension and low systemic blood flow in the very low birth weight neonate during the first postnatal week. J Perinatol 2006;26:S8–13; with permission.)

Noori S et al. Neonatal blood pressure support: the use of inotropes, lusitropes, and other vasopressor agents. J Clin Perinatol 2012; 39: 221-238

Dopamin

- reseptor **dopamin**
- reseptor **α** dan **β-adrenergik**
- reseptor **serotonin**
- aktivitas ***dose dependent***

Dopamin

Dosis Rendah

- **Dosis ginjal**
- 0,5-2μg/kgBB/mnt
- Stimulasi reseptor **dopamin perifer**
- **Meningkatkan aliran darah ginjal, mesenterika** dan **koroner**

Dosis Sedang

- **Dosis kardiotonik**
- 2-10μg/kgBB/mnt
- Stimulasi reseptor **ß-adrenergik**
- Meningkatkan **denyut jantung** dan ***cardiac output***
- **Vasodilator perifer**

Dosis Tinggi

- **Dosis presor**
- Aktivasi reseptor a **adrenergik** dan **serotonin**
- Peningkatan **resistensi vaskular** sistemik

Dobutamin

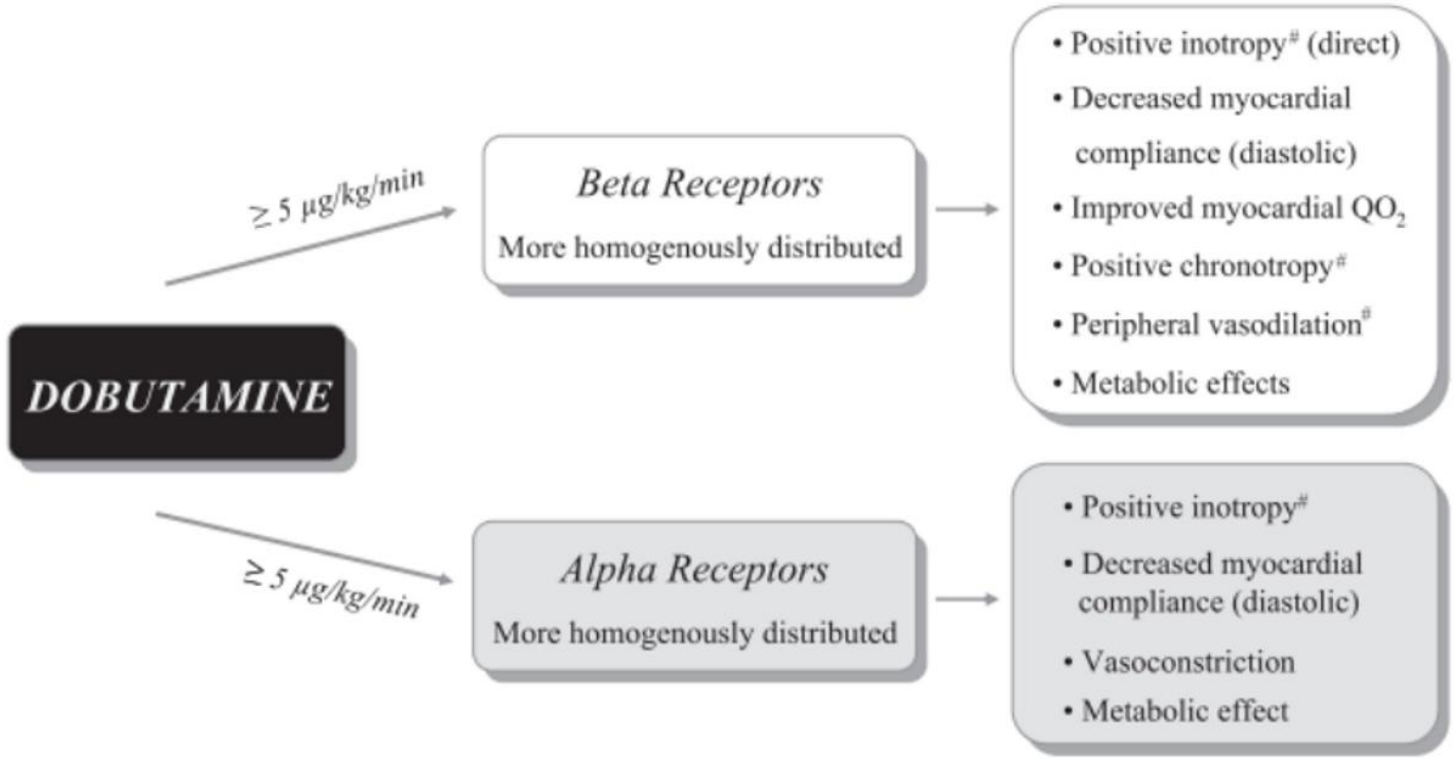


Fig. 4. Dose-dependent cardiovascular effects of dobutamine in neonates. Receptor-specific cardiovascular actions of dobutamine are shown presuming absence of adrenoreceptor downregulation (#denotes the effects demonstrated in neonates). (*Modified from* Seri I. Management of hypotension and low systemic blood flow in the very low birth weight neonate during the first postnatal week. J Perinatol 2006;26:S8–13; with permission.)

Noori S et al. Neonatal blood pressure support: the use of inotropes, lusitropes, and other vasopressor agents. J Clin Perinatol 2012; 39: 221-238

Dobutamin

Martin & Fanaroff

- dosis : 5–20 mcg/kgBB/menit IV
- **meningkatkan *output* jantung** (reseptor β-1, kardioselektif)

Digunakan sebagai terapi awal inotropik

- untuk kelainan **disfungsi miokardium**

Dopamin/Dobutamin Infus

- Berikan terapi cairan awal
- Monitor tekanan darah dan frekuensi jantung
- Gunakan *infusion pump*
- Infus melalui kateter vena umbilikal/PICC
  - ◊ Bila jalur vena sentral tidak ada, gunakan jalur perifer terpisah

▲Jangan menggunakan arteri umbilikal atau arteri lain

▲ Jangan melakukan *flush* dari jalur dopamin

Racikan Dopamin/Dobutamin

Dopamin/Dobutamin	Dose equivalent
15mg/kgBb dalam 50 ml D5%	1 ml/jam : 5 mikrogram/kgBb/min
30mg/kgBb dalam 50 ml D5%	1 ml/jam : 10 mikrogram/kgBb/min
Dose range	5-20 mikrogram/kgBb/min

Persiapan Dopamin/Dobutamin

	Dopamin/Dobutamin
Membuat larutan*	30 mg/kg dalam 50 ml
Dosis equivalen	1 ml/jam = 10 mikrogram/kg/mnt
Dosis interval	5-20 mikrogram/kg/mnt

***kondisi khusus jika bayi BBLASR larutan bisa dibuat dalam 25 ml**

Tabel Contoh lain
(lebih jarang dipakai)

Ordered Dose (mcg/kg/min) using a dopamine solution containing 800 mcg per ml of IV fluid								
Weight in kg	5 mcg/kg/min	7.5 mcg/kg/min	10 mcg/kg/min	12.5 mcg/kg/min	15 mcg/kg/min	17.5 mcg/kg/min	20 mcg/kg/min	25 mcg/kg/min
0.5 kg	0.2 ml/hr	0.3 ml/hr	0.4 ml/hr	0.5 ml/hr	0.6 ml/hr	0.7 ml/hr	0.8 ml/hr	0.9 ml/hr
1 kg	0.4 ml/hr	0.6 ml/hr	0.8 ml/hr	0.95 ml/hr	1.1 ml/hr	1.3 ml/hr	1.5 ml/hr	1.9 ml/hr
1.5 kg	0.6 ml/hr	0.8 ml/hr	1.1 ml/hr	1.4 ml/hr	1.7 ml/hr	2 ml/hr	2.3 ml/hr	2.8 ml/hr
2 kg	0.8 ml/hr	1.1 ml/hr	1.5 ml/hr	1.9 ml/hr	2.3 ml/h	2.6 ml/hr	3 ml/hr	3.8 ml/hr
2.5 kg	0.95 ml/hr	1.4 ml/hr	1.9 ml/hr	2.3 ml/hr	2.8 ml/hr	3.3 ml/hr	3.8 ml/hr	4.7 ml/hr
3 kg	1.1 ml/hr	1.7 ml/hr	2.3 ml/hr	2.8 ml/hr	3.4 ml/hr	3.9 ml/hr	4.5 ml/hr	5.6 ml/hr
3.5 kg	1.3 ml/hr	2 ml/hr	2.6 ml/hr	3.3 ml/hr	3.9 ml/hr	4.6 ml/h	5.3 ml/hr	6.6 ml/hr
4 kg	1.5 ml/hr	2.3 ml/h	3 ml/hr	3.8 ml/hr	4.5 ml/h	5.3 ml/hr	6 ml/hr	7.5 ml/hr
4.5 kg	1.7 ml/hr	2.5 ml/hr	3.4 ml/hr	4.2 ml/h	5.1 ml/hr	5.9 ml/hr	6.8 ml/hr	8.4 ml/hr
5 kg	1.9 ml/hr	2.8 ml/hr	3.8 ml/hr	4.7 ml/hr	5.6 ml/hr	6.6 ml/hr	7.5 ml/hr	9.4 ml/hr

Epinefrin

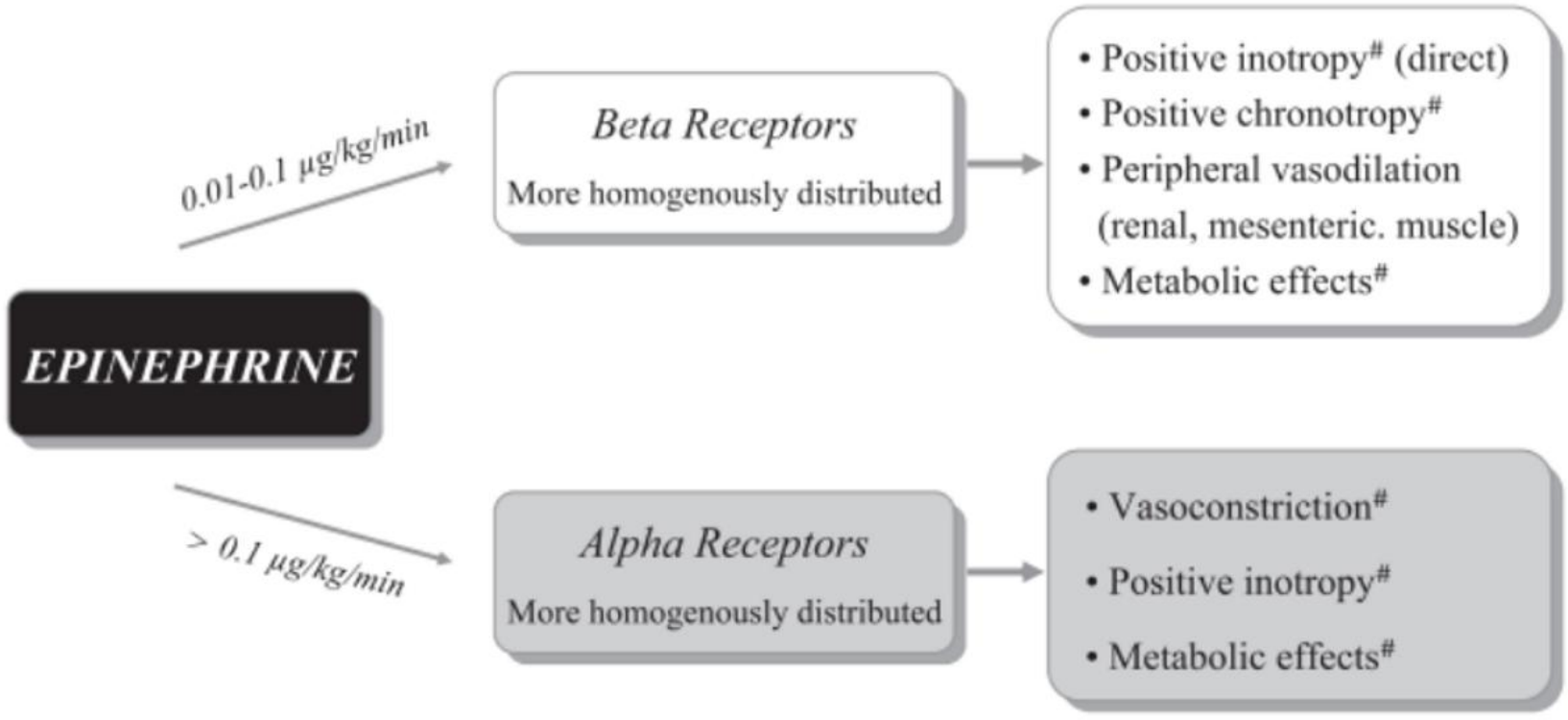


Fig. 5. Dose-dependent cardiovascular effects of epinephrine in neonates. Receptor-specific cardiovascular actions of epinephrine are shown presuming absence of adrenoreceptor downregulation (#denotes the effects demonstrated in neonates). (*Modified from* Seri I. Management of hypotension and low systemic blood flow in the very low birth weight neonate during the first postnatal week. J Perinatol 2006;26:S8–13; with permission.)

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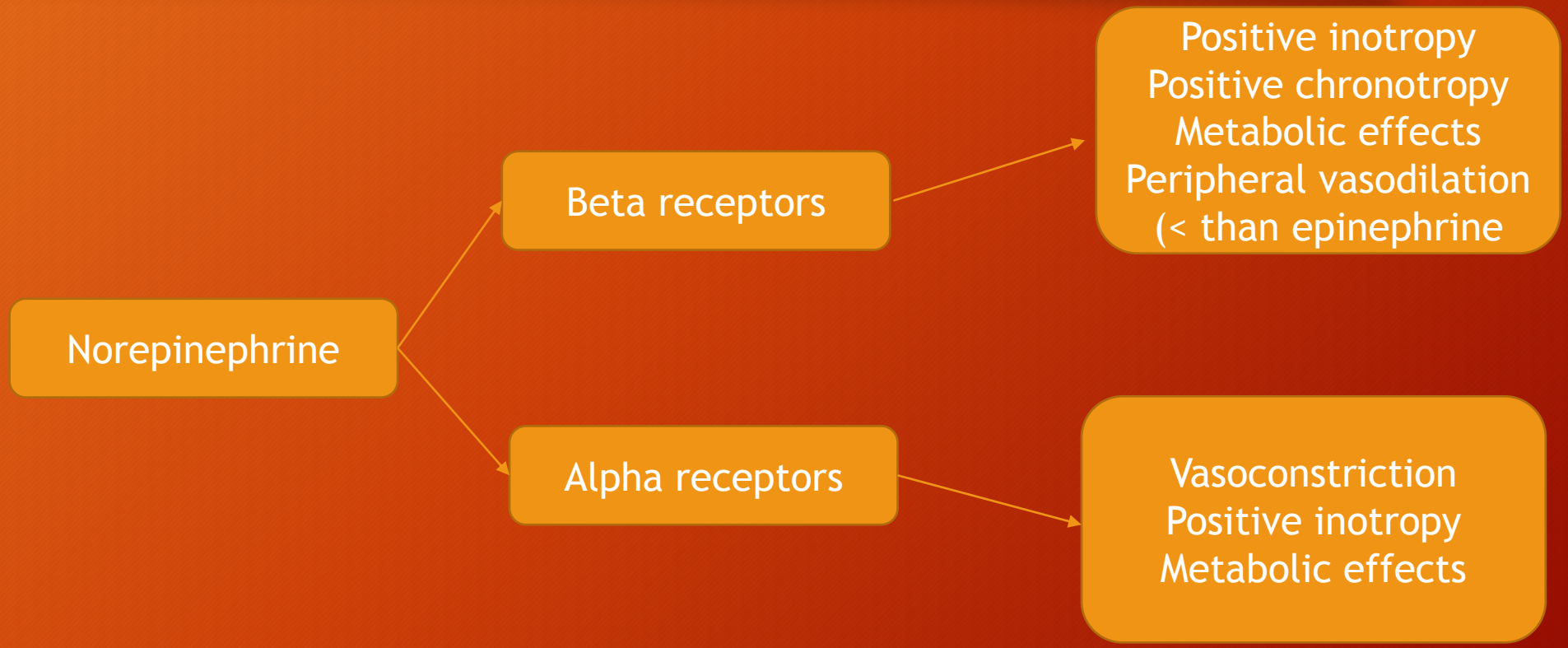
Efinefrin

- **Katekolamin endogen**
  - **Dosis rendah** bekerja pada **β1-adrenergik**
  - inotropik, kronotropik dan vasodilator
    - **menstimulasi jantung**
  - **Dosis tinggi** bekerja pada **α-adrenergik**
    - meningkatkan **resistensi perifer**

Martin dan Fanaroff

- infus drip 0,05–1mg/kgBB/menit IV

Norepinefrin



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Norefinefrin

- Lebih menyebabkan **vasokonstriksi** dibandingkan adrenalin
- Penggunaan noradrenalin **terbatas** karena efek vasokonstriksinya yang **dominan**
  - kemungkinan **iskemik** dan peningkatan ***afterload***

Kortikosteroid

- Bayi prematur
  - **jaras hipotalamus-hipofise-adrenal belum matang**
  - **respon terhadap stres inadekuat**

- Deksametason dan hidrokortison
  - **meningkatkan tekanan darah** pada keadaan hipotensi persisten

- Rekomendasi dosis:
  - Hidrokortison 2–10 mg/KgBb/hari, 2–4 dosis
  - Deksametason 0,25 mg/KgBb, dosis tunggal

Table 1
Cardiovascular actions mediated by adrenergic, dopaminergic, and vascular vasopressin receptors

	Adrenergic, Dopaminergic, and Vasopressin Receptors					
	α_1/α_2[a]	β_2	α_1	β_1/β_2	DA_1/DA_2	V_{1a}
	Vascular	Vascular	Cardiac	Cardiac	Vascular/Cardiac	Vascular
Vasoconstriction	++++	0	0	0	0	++++
Vasodilation	0	++++	0	0	++++[b]	0
+Inotropy	0	0	++	++++	+/++	0
+Chronotropy	0	0	0	++++	0	0
Cond. velocity	0	0	0	++++	0	0

Abbreviations: Cond. velocity, conduction velocity; +Chronotropy, positive chronotropy; +Inotropy, positive inotropy.

Estimated relative vascular (vasoconstriction and vasodilation) and cardiac (inotropy, chronotropy, and conduction velocity) effects mediated by the cardiovascular adrenergic (α_1/α_2 and β_1/β_2), dopaminergic (DA_1/DA_1), and vasopressin (V_{1a}) receptor subtypes.

[a] α_2-Receptors cause arterial vasodilation and venous vasoconstriction.

[b] Renal, mesenteric, coronary circulation > pulmonary circulation > extracranial vessels of the neck.

Table 2
Estimated relative cardiovascular receptor stimulatory effects of inotropes, lusitropes, and vasopressors

	Adrenergic, Dopaminergic, and Vasopressin Receptors					
	α_1/α_2	β_2	α_1	β_1/β_2	DA_1/DA_2	V_{1a}
	Vascular	Vascular	Cardiac	Cardiac	Vascular/Cardiac	Vascular
Phenylephrine	++++	0	+	0	0	0
Norepinephrine	++++	0/+	++	++++	0	0
Epinephrine	++++	++++	++	++++	0	0
Dopamine[a]	++++	++	++	+++	++++	0
Dobutamine[b]	+/0	++	++	++++	0	0
Isoprenaline	0	+++	0	++++	0	0
Vasopressin	0	0	0	0	0	++++
PDE-III inhibitors	0	0	0	0	0	0
PDE-V inhibitors	0	0	0	0	0	0

Abbreviations: $\alpha_1/\alpha_2/\beta_1/\beta_2$, subtypes of α- and β-adrenoreceptors; DA, dopamine; DOB, dobutamine; PDE, phosphodiesterase enzyme; PDE-III inhibitors used in neonates, amrinone, milrinone; PDE-V inhibitors used in neonates, sildenafil; V_{1a}, vasopressin receptor expressed in the vasculature.

[a] Dopamine also has serotoninergic actions.

[b] Efficacy of dobutamine is independent of its affinity for adrenoreceptors.

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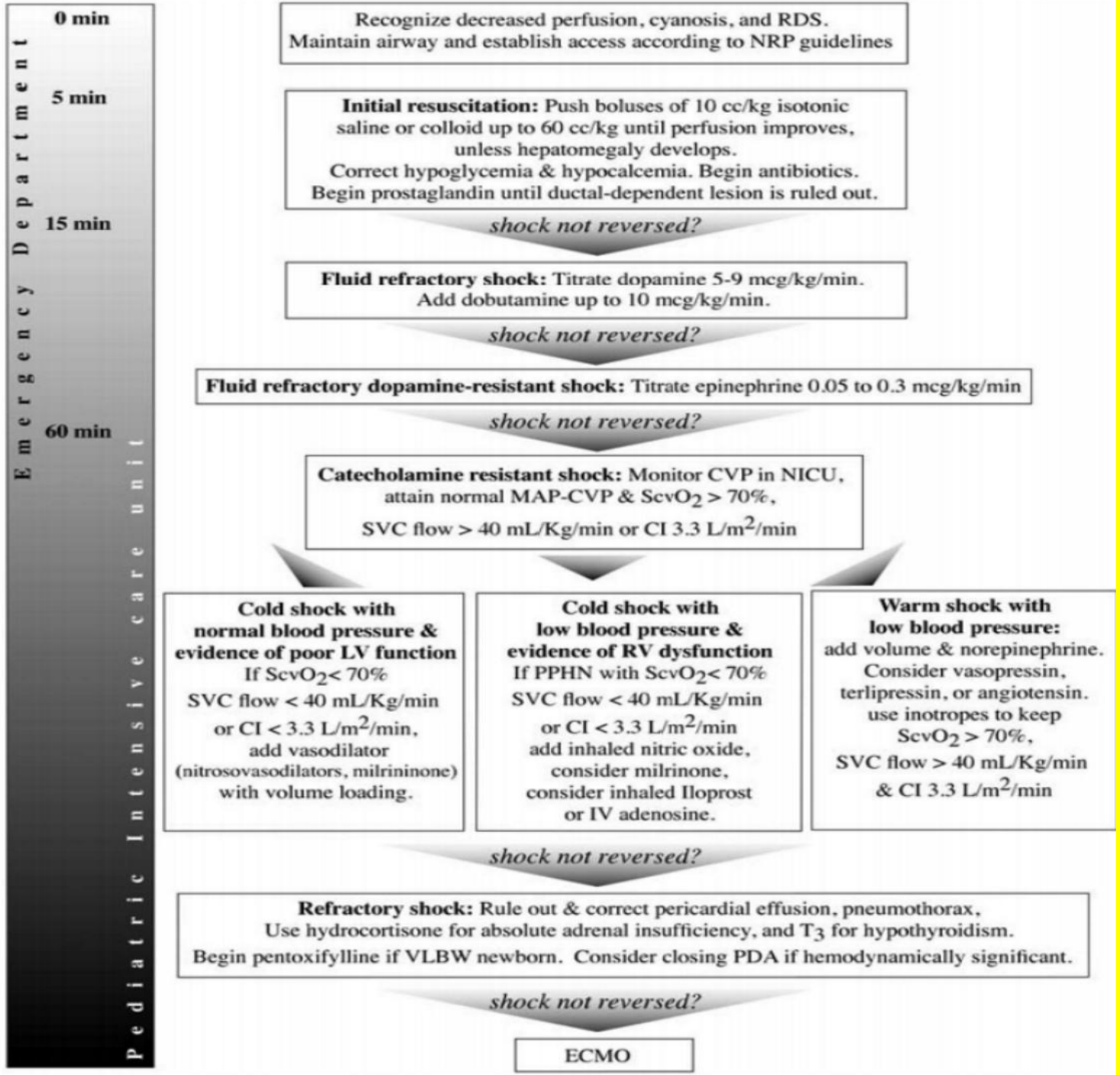

Algorithm for time sensitive, goal directed stepwise management of hemodynamic support in newborn

Brierley J, et al. Clinical practice parameters for hemodynamic support of pediatric and neonatal septic shock: 2007 update from American College of Critical Care Medicine. Crit Care Medicine 2009; 37(2): 666-88

Simpulan

- Monitor hemodinamika dini sangat krusial dalam mendeteksi dini keadaan instabilitas hemodinamik
- Parameter hemodinamika dapat dilihat dari klinis, laboratoris, hasil tindakan invasif dan non invasif
- Kombinasi dari berbagai parameter monitoring lebih baik dibandingkan dengan satu parameter
- Dalam keadaan klinis tertentu dibutuhkan inotropik untuk mengoptimalkan fungsi kardiovaskular
- Pilihan jenis dan dosis obat disesuaikan dengan pertimbangan klinis yang komprehensif

TERIMA KASIH