HYPOTENSION: HAEMODYNAMIC COMPROMISE

DEFINITION

- Characterised by low blood flow state, that clinically present with ≥1 of the following:
- low blood pressure
- acidosis
- raised lactate
- reduced urine output
- tachycardia
- prolonged capillary refill
- hypoxaemia
- **Do not** use mean blood pressure as sole indicator of haemodynamic compromise/treatment target
- Use gestational age based systolic and diastolic blood pressure centiles to guide management. See **Table 2** and **Table 3** for blood pressure values by gestational age or use blood pressure calculator https://neopeds.academy/bp/

RECOGNITION AND ASSESSMENT

- If signs of low blood flow state carry out thorough clinical evaluation
- if possible perform targeted neonatal echocardiography (TnECHO)
- Aim of evaluation, to establish underlying pathophysiology to help direct targeted management

CLINICAL INDICATORS OF COMPROMISED HAEMODYNAMIC STATUS

Table 1

Clinical indicator	Pathophysiology	Comments
Tachycardia	Increasing heart rate may increase cardiac output if stroke volume unchanged	• Affected by medications, pain, temperature, agitation • Avoid chronotropic agents to avoid compromising diastolic function
Systolic hypotension/narrow pulse pressure	Marker of decreased cardiac output	• Affected by transitional circulation, left-to-right shunts • Consider inotropes to augment myocardial contractility
Diastolic hypotension	Marker of systemic vascular resistance (SVR) and preload	• Affected by transitional circulation, left-to-right shunts • Consider vasopressors
Capillary refill time	Vasoconstriction of skin	• Affected by lighting, temperature, skin tone, anaemia
Pallor/acrocyanosis	Vasoconstriction of skin	• Affected by factors such as hypothermia, may be brisk in warm shock
Decreased level of consciousness	Decreased cerebral perfusion pressure	• Affected by sedative medications, meningitis, seizures
Elevated lactate	Anaerobic metabolism	• Also seen in some metabolic disorders,

		haemolysis, gluconeogenesis, placental insufficiency
Metabolic acidosis	Anaerobic metabolism	• Bicarbonate loss

GENERAL CONSIDERATIONS

- Tackle iatrogenic causes of low blood flow states in all cases, e.g. high mean airway pressure and over-ventilation
- Non-invasive blood pressure monitoring
- use appropriately sized blood pressure cuffs
- if not in keeping with clinical picture, consider repeating readings in different limb
- Invasive BP monitoring, ensure:
- transducer zeroed and set at appropriate height
- trace regular and not dampened
- no air bubbles in line (may be necessary to flush line)
- Optimise ionised calcium to ensure adequate myocardial function
- Do not initiate treatment based on a single marker of hemodynamic compromise
- Consider other causes, e.g.:
- pneumothorax
- pericardial effusion
- lung hyperinflation
- drug-induced compromise, e.g., opiates
- congenital heart disease
- metabolic and endocrine disorders
- Ensure adequate inotrope delivery before switching to a different agent (line patency, line dead-space etc.)
- Further management guided by underlying pathophysiology (see below)

Table 2 Normal blood pressure values by gestational age for day one (early)
(Mean +/- 95th CI for highest and lowest values)

Gestational age	Systolic			Diastolic			Mean (calculated)			Pulse pressure		
Weeks	95th	50th	5th	95th	50th	5th	95th	50th	5th	95th	50th	5th
22	55	39	22	31	23	14	39	28	17	18	12	8
23	56	40	23	32	24	15	40	29	18	18	12	8
24	57	42	25	33	25	16	41	31	19	18	12	8
25	58	43	26	34	26	17	42	32	20	18	12	8
26	60	44	27	35	27	18	43	33	21	18	12	8
27	61	45	29	36	28	19	44	34	22	18	12	8
28	63	47	31	37	29	20	46	35	24	19	13	9
29	64	48	33	38	30	21	47	36	25	19	13	9
30	66	50	35	39	31	22	48	37	26	19	13	9
31	68	51	36	40	32	23	49	38	27	20	14	10
32	69	52	37	41	33	24	50	39	28	20	14	10
33	70	53	38	42	34	25	51	40	29	20	14	10
34	71	55	40	43	35	26	52	42	31	20	14	10
35	73	57	41	44	36	27	54	43	32	20	14	10
36	75	59	42	45	37	28	55	44	33	20	14	10
37	76	60	44	46	38	29	56	45	34	20	14	10
38	77	61	46	47	39	30	57	46	35	21	15	12
39	79	62	47	48	40	31	58	47	36	21	15	12
40	81	64	48	49	41	32	60	49	37	21	15	12
41	82	65	50	50	42	33	61	50	39	22	15	12
42	84	67	51	51	43	34	62	51	40	22	15	12

Zubrow et al. Philadelphia Neonatal Blood Pressure Study Group. p. J of perinatology 1995

Table 3: Normal blood pressure values by corrected post conceptional age (Mean +/- 95th CI for highest and lowest values)

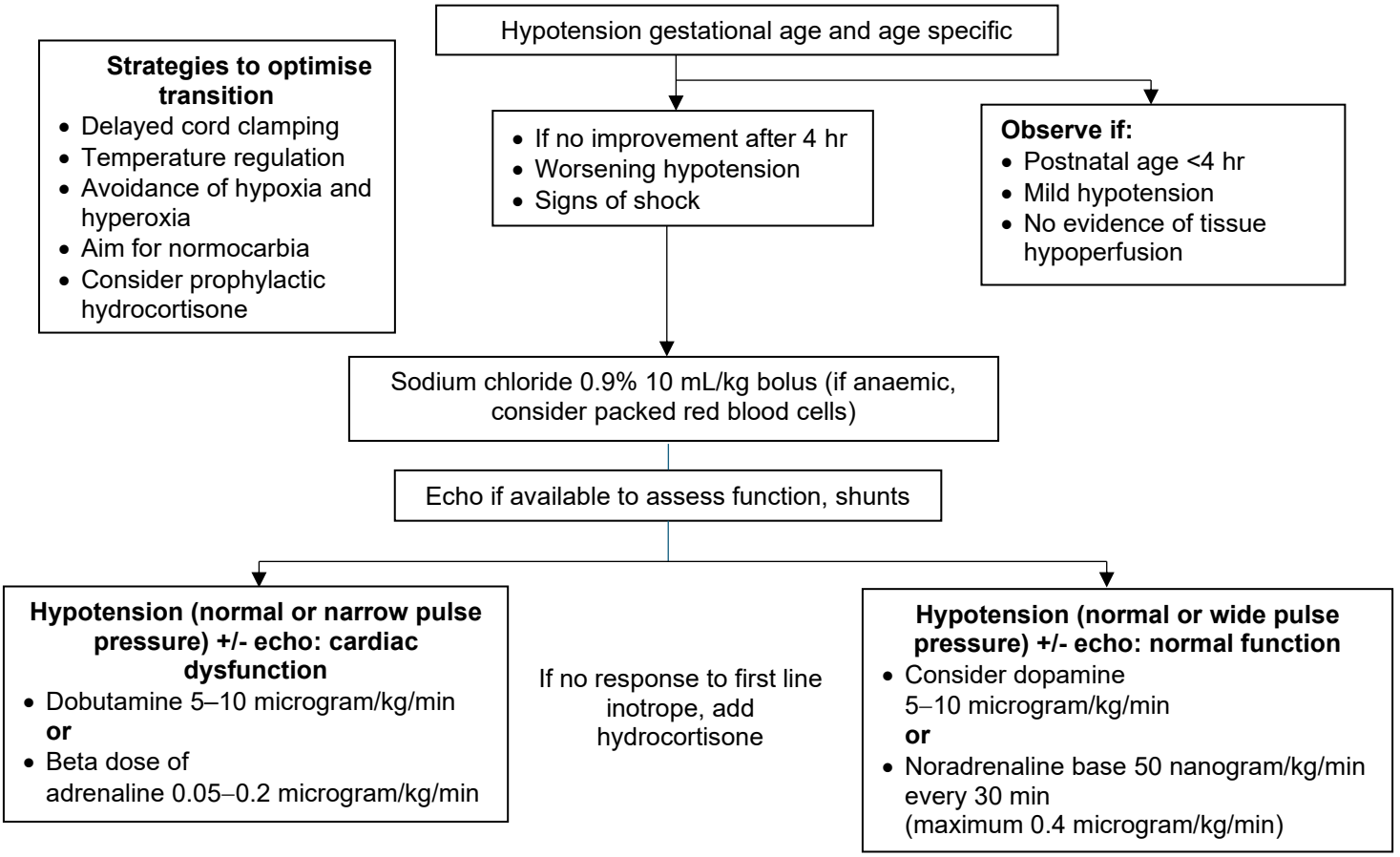
Age	Systolic			Diastolic			Mean (calculated)			Pulse pressure (calculated)		
Weeks	95th	50th	5th	95th	50th	5th	95th	50th	5th	95th	50th	5th
24	68	49	33	46	29	14	53	36	20	25	16	12
25	69	51	36	47	30	15	54	37	22	25	16	12
26	70	52	38	48	31	17	55	38	24	25	16	14
27	71	54	40	49	32	18	56	39	25	25	16	14
28	72	55	41	50	33	19	57	40	26	27	17	15
29	73	56	42	51	34	20	58	41	27	27	17	15
30	75	59	43	52	35	21	60	43	28	28	18	15
31	78	61	46	53	36	22	61	44	30	28	20	17
32	80	62	48	54	37	23	63	45	31	28	20	17
33	81	63	50	55	38	24	64	46	33	28	20	17
34	83	66	51	56	39	25	65	48	34	30	21	18
35	84	69	52	57	40	26	66	50	35	30	21	18
36	87	71	55	58	41	27	68	51	36	30	22	18
37	89	72	57	59	42	28	69	52	38	30	22	18
38	90	75	59	60	43	29	70	54	39	30	22	18
39	91	78	60	60	44	30	70	55	40	30	22	18
40	92	80	61	61	44	30	71	56	40	33	25	20
41	93	81	62	62	46	31	72	58	41	33	25	20
42	95	82	63	63	47	32	74	59	42	33	25	20
43	97	83	65	64	48	33	75	60	44	33	25	20
44	98	86	67	65	49	34	76	61	45	33	25	20
45	100	88	69	66	50	35	77	63	46	33	25	20
46	102	89	71	66	51	36	78	64	48	33	25	21

Zubrow et al. Philadelphia Neonatal Blood Pressure Study Group. p. J of perinatology 1995

TRANSITIONAL HAEMODYNAMIC COMPROMISE IN PRETERM BABIES

- Transitional haemodynamic compromise (33% of babies born <27 weeks) generally characterised by low diastolic BP (<5th centile), with normal or borderline low systolic BP, leading to low mean BP
- Condition is diagnosis of exclusion, rule out other causes, e.g. sepsis, if no response to first line agents

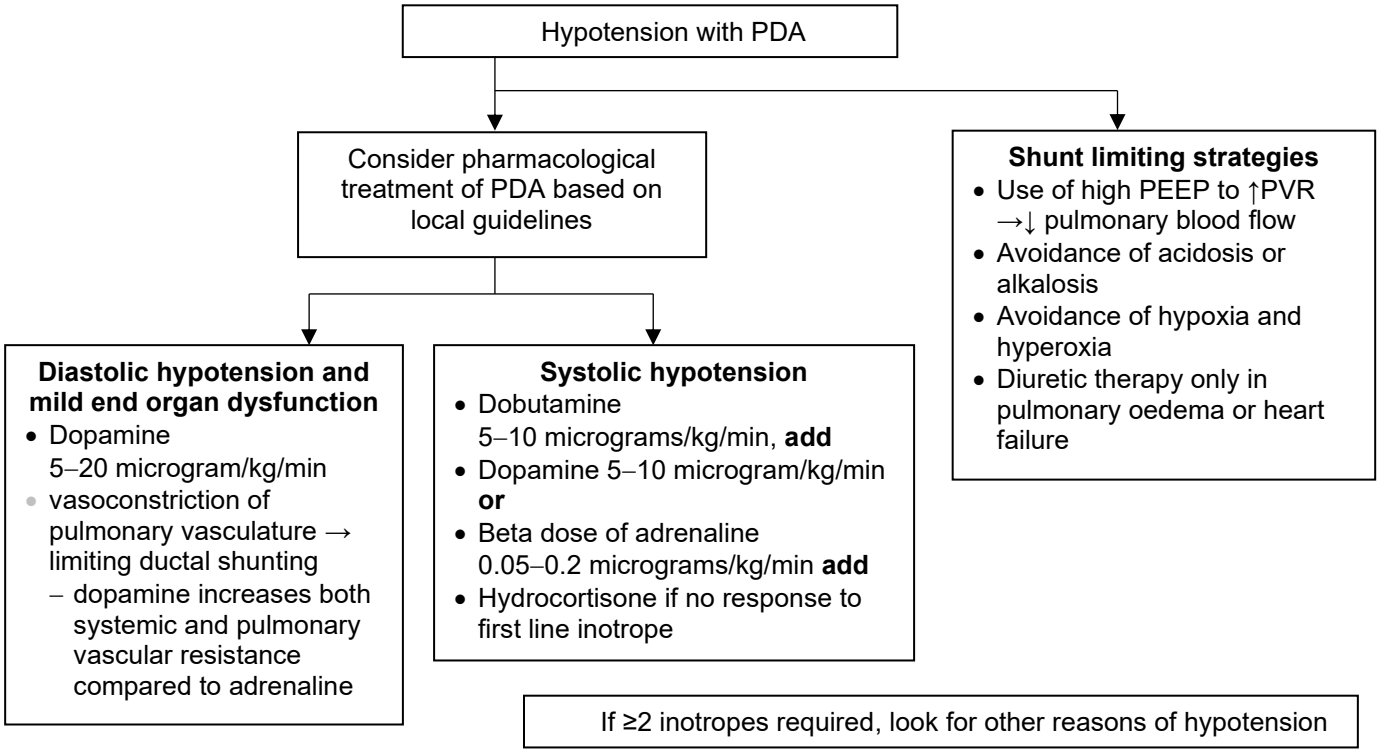
Flowchart 1: Management of transitional circulatory compromise in preterm babies



HAEMODYNAMIC COMPROMISE IN PDA

- Haemodynamically significant PDA can result in haemodynamic compromise after transitional period
- As pulmonary vascular resistance falls, transductal shunt volume increases leading to systemic steal and pulmonary over circulation
- Left ventricular (LV) stroke volume increases in response to increase preload
- classically presents as normal to high systolic BP with low diastolic BP (and wide pulse pressure) with/without signs of end organ dysfunction, e.g. acute kidney injury (AKI)
- Inability of the LV to augment systolic performance in response to increased preload will result in systolic hypotension and left atrial hypertension leading to pulmonary venous congestion
- Echocardiography essential to assess PDA and cardiac function
- Unusual for PDA to cause severe hypotension
- if more than first line agents required, look for other causes

Flowchart 2: Management of hypotension with PDA



HAEMODYNAMIC COMPROMISE IN HYPOXIC ISCHAEMIC INJURY (HIE)

Recognition and assessment

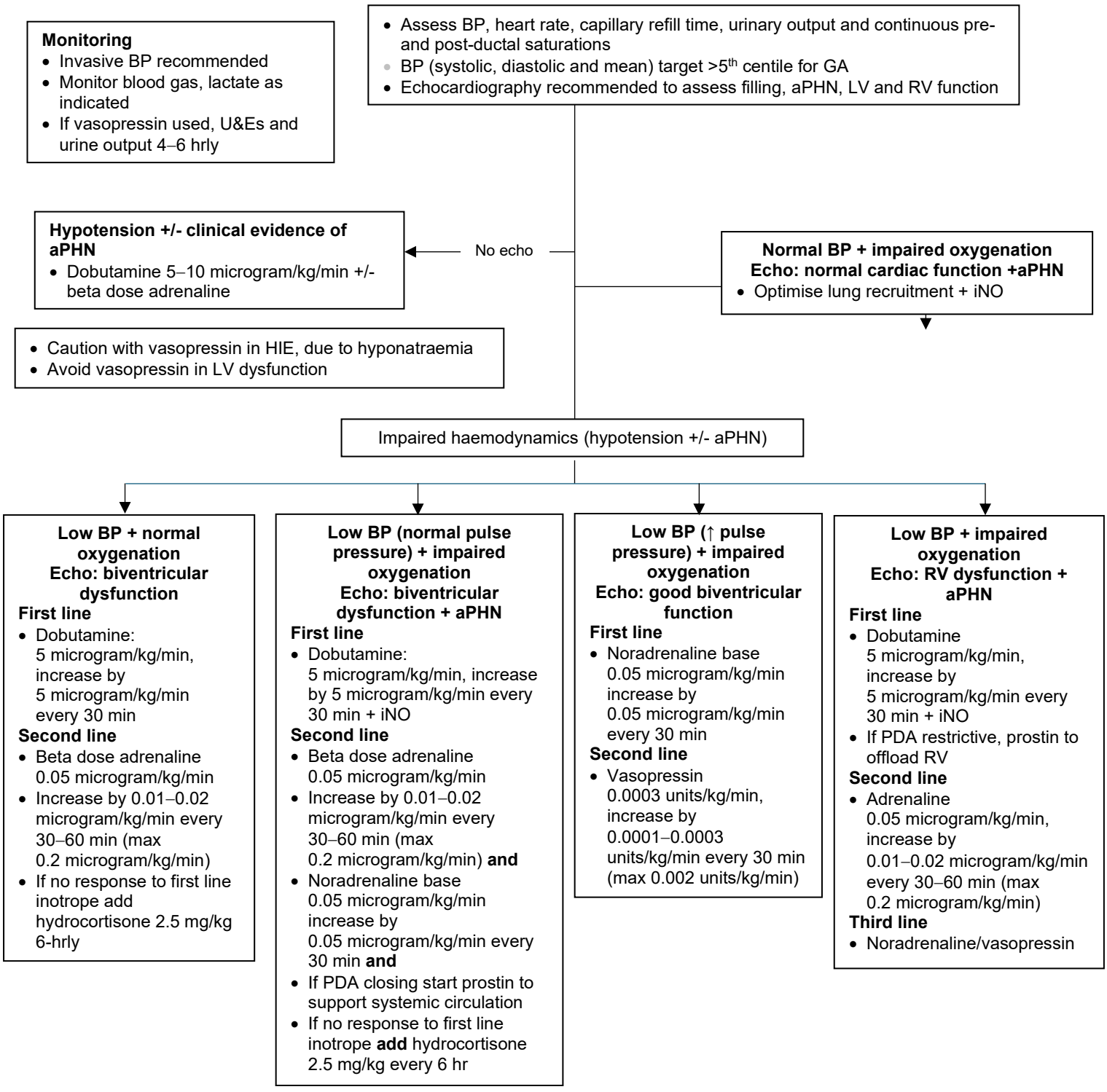
- Perinatal hypoxia results in transient myocardial ischaemia in about one-third of patients, which may result in reduced LV systolic performance and output
- Therapeutic hypothermia may mask hypotension by causing vasoconstriction and increasing diastolic BP
- HIE often associated with comorbidities, e.g. pulmonary hypertension and adrenal injury – may affect cardiovascular stability
- Presence of multiorgan dysfunction and therapeutic hypothermia makes assessment of haemodynamic status challenging due to deranged biochemical and clinical markers
- Echocardiography recommended to assess filling, LV, and right ventricular (RV) function, aPHN, PDA
- See **Recognition and assessment** and **General considerations** above

Management

- Avoid fluid bolus – will worsen cardiac dysfunction and may exacerbate cerebral oedema
- use fluid bolus only if evidence of intravascular depletion – up to 10 mL/kg over 30 min
- Isolated hypotension without impaired oxygenation
- start dobutamine 5 microgram/kg/min and titrate up to 10 microgram/kg/min
  - dose >10 microgram/kg/min associated with tachycardia
  - beta dose of adrenaline can be used
- In HIE complicated by pulmonary hypertension (aPHN), see **Pulmonary hypertension** guidelines)
- Isolated hypoxia and aPHN, optimise ventilation and start iNO
- Hypotension (normal pulse pressure) with hypoxia, commence dobutamine **or** beta dose of adrenaline
- if no response noradrenaline can be added
- Hypotension (wide pulse pressure) with hypoxia in presence of normal echo

- start noradrenaline base at 0.05 microgram/kg/min and titrate up to 0.4 microgram/kg/min
- vasopressin can be used cautiously as second line
- If restrictive PDA, start prostin either to support systemic circulation or offload right ventricle
- If no response to first line inotropes, add hydrocortisone.

Flowchart 3: Approach to haemodynamic optimisation in neonates with HIE



HAEMODYNAMIC OPTIMISATION IN LATE ONSET SEPSIS/NECROTISING ENTEROCOLITIS (NEC)

Definition

- Septic shock is a generalised, rapidly establishing tissue hypoperfusion due to dysregulated response to infection

Pathophysiology and assessment

- Sepsis and NEC cause systemic inflammatory response syndrome (SIRS) which lead to endothelial dysfunction associated with vasodilation and capillary leak
- leading to low SVR and low effective circulating volume
- LV compensates by going into a hyperdynamic state to meet the energy demands by increasing heart rate and cardiac output (warm shock)
- Increased demands and the direct effect of endotoxins may cause myocardial dysfunction resulting in low cardiac output (cold shock)
- Warm shock is the most common presentation in neonates, but infants may progress to or initially present with cold shock

Approach to management

- Distinguish between cold and warm shock as management guided by underlying pathophysiology
- Warm shock characterised by low diastolic BP with normal to high systolic BP, bounding pulses, flushed appearance, tachycardia, and brisk capillary refill
- Cold shock presents with low systolic BP or severe/combined hypotension, cool peripheries, and prolonged capillary refill
- Echocardiography to assess filling and function recommended
- Vasodilatory (warm) shock use:
- volume expansion (crystalloids/blood products up to 20 mL/kg) as the first line agent
- start noradrenaline base 0.05–0.4 microgram/kg/min
- consider vasopressin in babies with severe vasoplegia refractory to noradrenaline
- Vasoconstrictive (cold) shock
- use volume expansion cautiously in view of myocardial dysfunction
- adrenaline 0.05–0.2 microgram/kg/min recommended as first line agent to augment myocardial contractility and SVR
- both adrenaline and noradrenaline may be needed in severe refractory shock
- If no response to first line inotropes, add hydrocortisone – dysfunction of HPA axis is common in severe sepsis
- Monitor urine output, electrolytes and renal function with vasopressin every 4–6 hr
- In NEC, if baby requiring escalating inotropic support, discuss with KIDS NTS and surgeons at Birmingham Children’s Hospital

Flowchart 4: Approach to management of septic shock in late-onset sepsis/NEC