

Echocardiography Screening in Neonates with Hypoxic-Ischemic Encephalopathy (HIE) and Hemodynamic Instability

NICU Clinical Guideline – Neonatal Hemodynamics

Background

Hypoxic-ischemic encephalopathy (HIE) occurs in approximately 1 to 3 per 1,000 live births in high-income countries, and therapeutic hypothermia (TH) remains the standard of care [1]. Cooling reduces the composite outcome of death or major neurodevelopmental disability but HIE is increasingly recognized as a multi-organ disease in which cardiac dysfunction is frequently under-recognized [2-4].

Hemodynamic instability is common during TH and is associated with increased mortality and brain injury on MRI. Reported prevalence in moderate-to-severe HIE is summarized below.

Parameter	Prevalence	Population / notes
Hemodynamic impairment	Up to 80%	Moderate-severe HIE
Pulmonary hypertension (PPHN)	17-25%	Moderate-severe HIE
Hypotension requiring inotropes	30-65%	Higher with PPHN (65% vs 30%)
Inhaled nitric oxide (iNO)	64%	HIE with PPHN
ECMO	12%	HIE with PPHN

Cited prevalence figures are drawn from published HIE studies [3-6].

Purpose

To standardize early, targeted echocardiographic assessment in neonates with HIE and hemodynamic instability, so that hemodynamic management is guided by the underlying physiology rather than by blood pressure alone.

Why Blood Pressure (BP) Alone Is Insufficient

Mean arterial pressure (MAP) is the product of cardiac output (CO) and systemic vascular resistance (SVR). In HIE, CO and SVR often change, so BP can appear normal while systemic perfusion is poor (low CO with compensatory high SVR) [7].

- Neonatal BP thresholds are imperfect and are not validated for HIE physiology; the “MAP $\approx$ gestational age” rule is a rough estimate only.
- Therapeutic hypothermia lowers heart rate and cardiac output without a proportionate change in BP. Sedation, ventilation, acid-base status, and PPHN further distort BP interpretation [8].
- A mean BP of at least 40-50 mmHg best preserves cerebral autoregulation; longer time spent outside this range is associated with more severe brain injury on MRI [9, 10].
- BP also does not identify which therapy is required. Surrogate markers of perfusion are unreliable during TH: lactate does not reliably reflect real-time cardiac output, capillary refill time is influenced by cooling, and urine output is confounded by perinatal renal injury [11, 12].

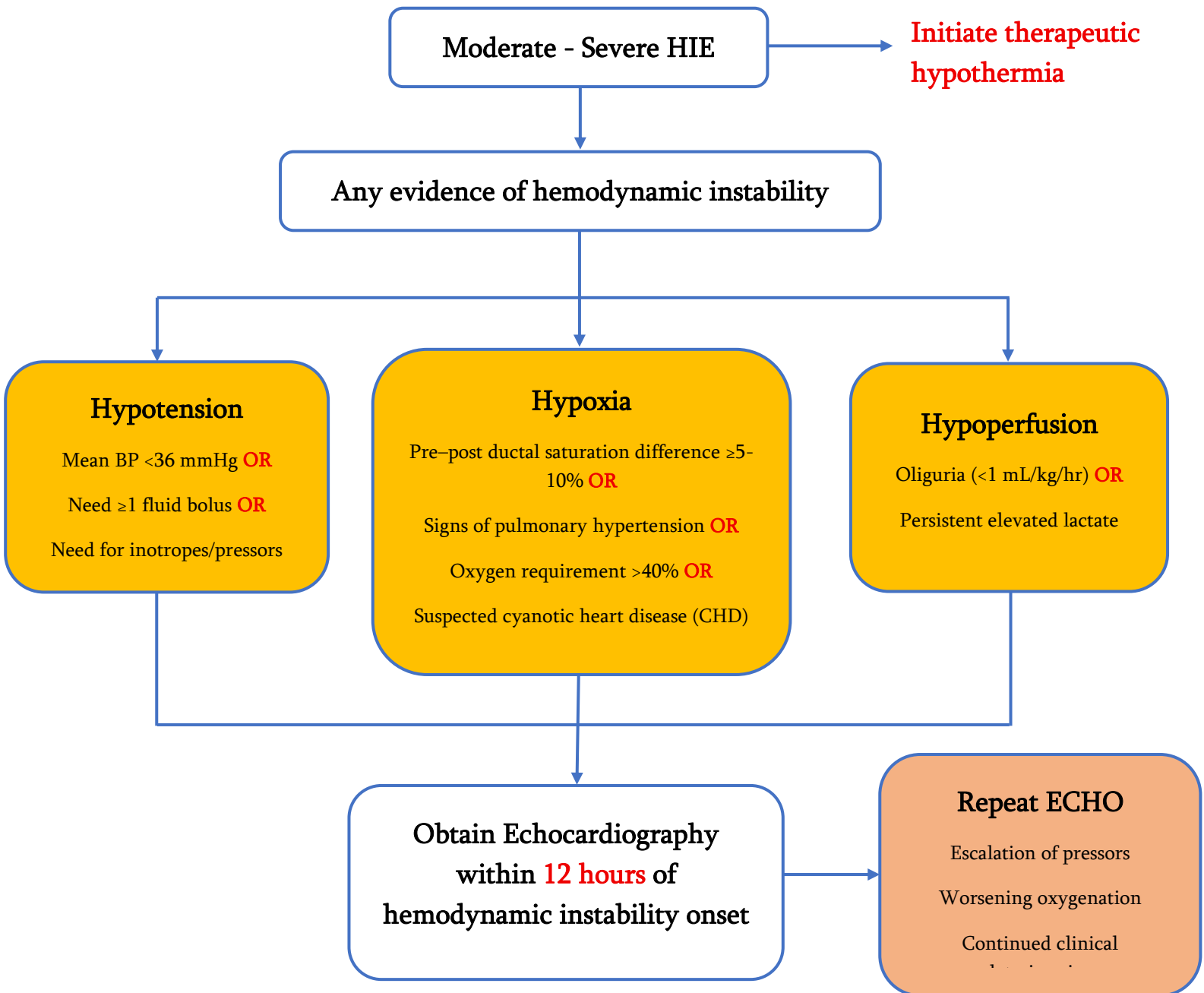
Role of Targeted Echocardiography

Targeted neonatal echocardiography characterizes the physiology that drives instability and allows therapy to be matched to the underlying lesion. Echocardiography can assess:

- Fluid status and ventricular filling
- Left ventricular systolic and diastolic function
- Right ventricular function
- PPHN severity and shunt direction
- Ductal and atrial shunting and PDA physiology
- Estimates of filling pressures (left atrial size, Doppler patterns)

Echocardiography-guided management aids inotrope and vasopressor selection, identifies the need for pulmonary vasodilators, avoids inappropriate fluid loading, and helps identify high-risk neonates associated with poorer outcomes.

Echocardiography screening guideline in neonates with
HIE & Hemodynamic Instability



References:

1. Bonifacio, S.L. and S. Hutson, *The Term Newborn: Evaluation for Hypoxic-Ischemic Encephalopathy*. Clin Perinatol, 2021. **48**(3): p. 681–695.
2. Zanelli, S.A., et al., *Therapeutic Hypothermia for Neonatal Hypoxic-Ischemic Encephalopathy: Clinical Report*. Pediatrics, 2026. **157**(2).
3. Azzopardi, D.V., et al., *Moderate hypothermia to treat perinatal asphyxial encephalopathy*. N Engl J Med, 2009. **361**(14): p. 1349–58.
4. Shankaran, S., et al., *Whole-body hypothermia for neonates with hypoxic-ischemic encephalopathy*. N Engl J Med, 2005. **353**(15): p. 1574–84.
5. Lakshminrusimha, S., et al., *Pulmonary Hypertension Associated with Hypoxic-Ischemic Encephalopathy-Antecedent Characteristics and Comorbidities*. J Pediatr, 2018. **196**: p. 45–51 e3.
6. Altit, G., et al., *Cardiac Dysfunction in Neonatal HIE Is Associated with Increased Mortality and Brain Injury by MRI*. Am J Perinatol, 2023. **40**(12): p. 1336–1344.
7. Rios, D.R., et al., *Hemodynamic optimization for neonates with neonatal encephalopathy caused by a hypoxic ischemic event: Physiological and therapeutic considerations*. Semin Fetal Neonatal Med, 2021. **26**(4): p. 101277.
8. Giesinger, R.E., et al., *Cardiovascular management following hypoxic-ischemic encephalopathy in North America: need for physiologic consideration*. Pediatr Res, 2021. **90**(3): p. 600–607.
9. Lee, J.K., et al., *Optimizing Cerebral Autoregulation May Decrease Neonatal Regional Hypoxic-Ischemic Brain Injury*. Dev Neurosci, 2017. **39**(1-4): p. 248–256.
10. Kent, A.L., et al., *Normative blood pressure data in the early neonatal period*. Pediatr Nephrol, 2007. **22**(9): p. 1335–41.
11. Surak, A., et al., *Hemodynamics in infants with hypoxemic ischemic encephalopathy: pathophysiology and beyond*. J Perinatol, 2025.
12. Eriksen, V.R., et al., *Lactate acidosis and cardiac output during initial therapeutic cooling in asphyxiated newborn infants*. PLoS One, 2019. **14**(3): p. e0213537.

Medical Legal Disclaimer:

Welcome to the UC Davis Health, Department of Pediatrics, Clinical Practice Guidelines Website. All health and health-related information contained within the Site is intended chiefly for use as a resource by the Department's clinical staff and trainees in the course and scope of their approved functions/activities (although it may be accessible by others via the internet). This Site is not intended to be used as a substitute for the exercise of independent professional judgment. These clinical pathways are intended to be a guide for practitioners and may need to be adapted for each specific patient based on the practitioner's professional judgment, consideration of any unique circumstances, the needs of each patient and their family, and/or the availability of various resources at the health care institution where the patient is located. Efforts are made to ensure that the material within this Site is accurate and timely but is provided without warranty for quality or accuracy. The Regents of the University of California; University of California, Davis; University of California, Davis, Health nor any other contributing author is responsible for any errors or omissions in any information provided or the results obtained from the use of such information. Some pages within this Site, for the convenience of users, are linked to or may refer to websites not managed by UC Davis Health. UC Davis Health does not control or take responsibility for the content of these websites, and the views and opinions of the documents in this Site do not imply endorsement or credibility of the service, information or product offered through the linked sites by UC Davis Health. UC Davis Health provides limited personal permission to use the Site. This Site is limited in that you may not:

- Use, download or print material from this site for commercial use such as selling, creating course packets, or posting information on another website.
- Change or delete propriety notices from material downloaded or printed from it. · Post or transmit any unlawful, threatening, libelous, defamatory, obscene, scandalous, inflammatory, pornographic, or profane material, any propriety information belonging to others or any material that could be deemed as or encourage criminal activity, give rise to civil liability, or otherwise violate the law.
- Use the Site in a manner contrary to any applicable law.

You should assume that everything you see or read on this Site is copyrighted by University of California or others unless otherwise noted. You may download information from this Site as long as it is not used for commercial purposes, and you retain the proprietary notices. You may not use, modify, make multiple copies, or distribute or transmit the contents of this Site for public or commercial purposes without the express consent of UC Davis Health.