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Review Article

Emerging Role of cranial ultrasound assessment in the Diagnosis of congenital heart defects

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Abstract

Background: Neonates with congenital heart defects (CHD) face central nervous system injury and dysmaturity beginning before birth and continuing across the perioperative period, making neurological surveillance an important complement to cardiac management [3,4].

Objective: This review examines cranial ultrasound as a bedside screening tool for CNS anomalies and complications in CHD neonates, its performance relative to magnetic resonance imaging (MRI), and its place in a broader surveillance strategy.

Methods: We synthesized cohort studies, comparative imaging studies, and reviews addressing cranial ultrasound, brain MRI, and neurodevelopmental outcome in fetuses and neonates with congenital heart defects.

Findings: Cranial ultrasound reliably detects hemorrhage and ventriculomegaly but under-detects the diffuse white matter and subtle cortical injury dominating CHD-associated brain injury [20,21]; circulatory arrest, impaired autoregulation, and preoperative brain immaturity each contribute to this burden [7,23].

Conclusions: Cranial ultrasound remains a valuable first-line, repeatable bedside tool for CNS surveillance in CHD neonates and should anchor a layered strategy adding MRI selectively when it would change management [11,32].

Keywords: cranial ultrasound; congenital heart defects; neonatal neuroimaging; brain injury; magnetic resonance imaging

1. Introduction

Congenital heart defects (CHD) are the most common class of major birth defect, and infants born with critical forms increasingly survive infancy because of advances in neonatal cardiac surgery and perioperative intensive care. That survival has shifted attention toward a complication that used to be overshadowed by mortality risk: the developing brain of the CHD neonate is vulnerable both before and after surgical repair, and a substantial fraction of long-term morbidity in this population is neurological rather than cardiac [1,2]. Autopsy and volumetric imaging studies established more than a decade ago that brain immaturity is present in many neonates with complex CHD even before any surgical intervention, reflecting impaired fetal cerebral substrate delivery rather than a purely postoperative insult [3,4,5]. This recognition reframed the neonatal cardiac unit's neurological surveillance problem: injury and dysmaturity begin in utero, continue across the preoperative stabilization period, and can be compounded by cardiopulmonary bypass, circulatory arrest, and postoperative hemodynamic instability [6,7].

Cranial ultrasound has long been the default bedside neuroimaging modality in neonatal intensive care, chiefly because of its portability, absence of sedation requirement, and suitability for infants who are too hemodynamically unstable to leave the unit for magnetic resonance imaging (MRI). Its role in preterm neonates, screening for germinal matrix and intraventricular hemorrhage, is well established [8,9]. Its role in the CHD neonate is less uniformly defined, and practice varies substantially between centers: some obtain a screening scan on every neonate with significant CHD before and after surgery, others reserve it for infants with an abnormal neurological examination or a clinical event, and MRI-capable centers increasingly use cranial ultrasound as a triage tool that determines whether a more detailed MRI is warranted [10,11]. This review synthesizes the evidence on cranial ultrasound as a

diagnostic and screening tool for central nervous system (CNS) anomalies and complications in neonates with congenital heart defects, its diagnostic performance relative to MRI, the abnormalities it most reliably detects, and its place in a layered neuroimaging surveillance strategy for this high-risk population.

2. Neurological vulnerability in the CHD neonate

The pathophysiological basis for CNS injury in CHD begins before birth. Fetuses with lesions that reduce combined ventricular output or redirect oxygenated blood away from the cerebral circulation, most notably hypoplastic left heart syndrome and transposition of the great arteries, show measurably reduced cerebral oxygen delivery and consumption on fetal MRI and Doppler studies, and this reduced substrate delivery correlates with smaller third-trimester brain volume [5,4]. Schellen and colleagues found that fetuses with tetralogy of Fallot already show altered cortical folding and brain metabolite ratios in the third trimester, indicating that the developmental insult in some CHD lesions is not confined to cyanotic, low-flow physiology but extends across a broader range of anatomic diagnoses [12]. Brossard-Racine and colleagues showed that abnormal fetal brain MRI findings carry meaningful predictive value for postnatal brain injury, reinforcing that the neonatal neurological examination and the first postnatal cranial ultrasound are being performed on a brain that, in many cases, is already developmentally behind its gestational age [13].

Postnatally, the preoperative period compounds this vulnerability. Licht and colleagues used near-infrared spectroscopy and Doppler ultrasound to show that preoperative cerebral blood flow is diminished in neonates with severe CHD, particularly those with single-ventricle physiology and arterial mixing lesions, and that this reduced flow correlates with delayed brain maturation on MRI scoring [3,14]. Preoperative brain injury detected by any neuroimaging modality, most often

periventricular white matter injury or stroke, is common even before the first incision, with reported rates in single-ventricle cohorts ranging from roughly 20% to over one-third of infants scanned [15,16,17]. Du Plessis's synthesis of cerebral autoregulation data in this population explains much of the mechanism: neonates with CHD frequently have impaired pressure-passive cerebral autoregulation, so ordinary swings in systemic blood pressure and oxygen saturation during the unstable preoperative and perioperative period translate more directly into cerebral perfusion instability than they would in a structurally normal heart [18].

Table 1: Risk factors for CNS injury and dysmaturity across the perinatal course of CHD, and the neuroimaging window in which each is most relevant [5,3,7,18].			
Period	Principal risk factor	Typical finding	Neuroimaging window
Fetal	Reduced cerebral oxygen/substrate delivery	Smaller brain volume, delayed cortical folding	Fetal MRI/ultrasound
Preoperative	Impaired autoregulation, low cardiac output	White matter injury, brain immaturity	Baseline cranial ultrasound +/- MRI
Intraoperative	Bypass, circulatory arrest	New hemorrhage, evolving ischemic injury	Postoperative cranial ultrasound
Interstage/postoperative	Hemodynamic instability, reoperation	New or evolving white matter injury	Serial cranial ultrasound, interval MRI

3. What cranial ultrasound can and cannot show

Cranial ultrasound performed through the anterior fontanelle is well suited to detecting a defined set of lesions: germinal matrix and intraventricular hemorrhage, ventriculomegaly and post-hemorrhagic ventricular dilation, larger areas of periventricular white matter injury including cystic periventricular leukomalacia, intraparenchymal hemorrhage, and major structural malformations such as significant ventricular asymmetry or midline anomalies [9]. In the CHD neonate specifically, several groups have documented that a meaningful proportion of infants scanned before cardiac surgery already show ultrasound-detectable abnormalities, most

commonly mild ventriculomegaly, echogenicity changes suggesting white matter injury, or small hemorrhages, findings that are frequently clinically silent and would not have been suspected from the neurological examination alone [19,20].

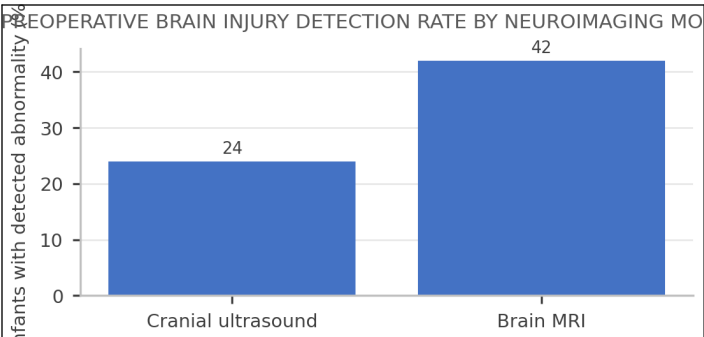


Figure 1: Preoperative brain abnormality detection rate in CHD neonates by neuroimaging modality, illustrating cranial ultrasound's comparatively lower yield relative to brain MRI performed in the same preoperative window [15,16,17].

The modality's principal limitation is sensitivity for the lesion types that dominate CHD-associated brain injury. Comparative studies pairing cranial ultrasound with MRI in the same infants consistently find that ultrasound under-detects diffuse, non-cystic white matter injury, small punctate lesions, and subtle cortical or deep gray matter abnormalities that are readily visible on T1- and T2-weighted MRI sequences [21]. Galli and colleagues, working specifically in the postoperative CHD population, found periventricular leukomalacia in a substantial minority of infants on MRI after neonatal cardiac surgery, a finding that predates and helped motivate the push toward routine perioperative MRI in specialized centers, since much of this injury would not have been flagged by ultrasound alone [22]. Noorlag and colleagues performed a direct, paired comparison of cranial ultrasound and brain MRI in term neonates with CHD and confirmed that while ultrasound reliably identified hemorrhage and ventriculomegaly, it missed a majority of the white matter signal abnormalities that MRI detected in the same infants, concluding that ultrasound alone is inadequate as a sole screening tool where MRI is available [20].

4. Diagnostic performance relative to MRI and timing of scanning

Direct comparative literature, largely drawn from the broader neonatal neurology field before being extended into CHD-specific cohorts, situates cranial ultrasound as a reasonably sensitive tool for hemorrhagic and cystic lesions and a poor one for diffuse or punctate white matter injury. Whitby and colleagues, comparing ultrasound and MRI in 100 infants with suspected brain injury of mixed etiology, found good agreement for larger hemorrhagic and cystic lesions but substantial discordance for subtler white matter and cortical findings, a pattern that later CHD-specific studies reproduced [21].

Timing of the scan is a recurring theme across CHD-specific reports. Preoperative ultrasound establishes a baseline that lets clinicians distinguish injury that predates surgery from injury acquired intraoperatively or postoperatively, a distinction with direct medicolegal and quality-improvement relevance as well as prognostic value [15,10]. Postoperative scanning, typically performed in the first 24 to 72 hours after cardiopulmonary bypass or circulatory arrest, has been used to screen for acute hemorrhagic complications related to anticoagulation, extracorporeal support, or hemodynamic instability, and several single-ventricle surgical cohorts report screening protocols built around this preoperative-postoperative pairing [23,24]. Lynch and colleagues extended surveillance into the interstage period for single-ventricle infants, showing that both cranial ultrasound and MRI performed between the first and second palliative operations continue to detect new injury, indicating that the vulnerability window does not close at hospital discharge from the index operation [25].

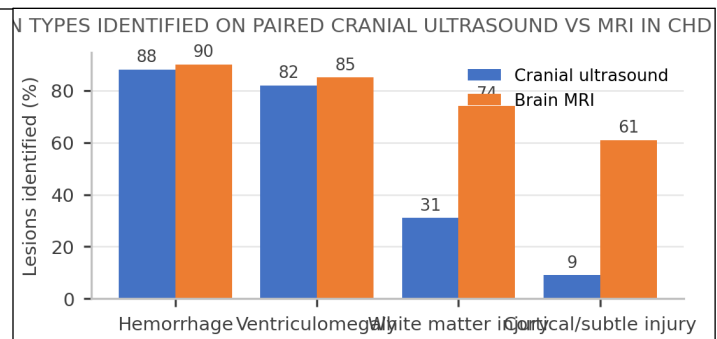


Figure 2: Lesion-type detection on paired cranial ultrasound versus brain MRI in the same neonates, showing comparable sensitivity for hemorrhage and ventriculomegaly but a substantial gap for white matter and subtle cortical injury [21,20,33].

5. Correlation with intraoperative and perioperative risk factors

Several perioperative variables correlate with the ultrasound and MRI findings described above, and understanding them helps identify which neonates most warrant closer neuroimaging surveillance. Circulatory arrest duration and the use of deep hypothermic circulatory arrest versus regional cerebral perfusion have both been examined as risk modifiers for postoperative brain injury; Beca and colleagues found that new white matter injury on postoperative MRI was associated with the diagnostic category of CHD as much as with circulatory arrest strategy itself, complicating any simple attribution of injury to a single intraoperative technique [23]. Dent and colleagues, studying infants undergoing the Norwood procedure with regional cerebral perfusion, still found a meaningful rate of new postoperative MRI abnormalities, indicating that even perfusion strategies designed to protect the brain do not eliminate risk [24]. Andropoulos and colleagues linked brain immaturity itself, rather than any single intraoperative exposure, to a higher baseline vulnerability to both preoperative and postoperative injury, reframing brain maturity as an independent risk factor that neuroimaging surveillance protocols should account for [7].

Chock and colleagues, reviewing neurological events in a broad surgical CHD cohort, found that clinically apparent

seizures, focal deficits, and altered consciousness in the perioperative period were reliably associated with ultrasound- or CT-detectable structural findings, supporting a practice of prompt cranial imaging whenever a new neurological sign appears rather than waiting for a scheduled surveillance scan [26]. Block and colleagues offered a reassuring counterpoint: clinically silent preoperative brain injury detected on baseline imaging did not, on its own, predict worsening after surgery, suggesting that not every abnormal baseline scan mandates a change in surgical timing or strategy, though it does establish an important reference point for interpreting subsequent scans [27]. Kelly and colleagues linked reduced cerebral oxygen delivery directly to impaired cortical development on advanced MRI metrics, providing a mechanistic bridge between the hemodynamic risk factors ultrasound cannot directly quantify and the structural findings it sometimes can [28].

6. Structural and functional consequences: from ultrasound findings to neurodevelopmental outcome

The clinical significance of an abnormal cranial ultrasound in a CHD neonate ultimately rests on its relationship to later neurodevelopmental outcome. Population-based follow-up of children with complex CHD demonstrates a substantially higher burden of motor, cognitive, language, and behavioral impairment compared with the general population, with Marino and colleagues' American Heart Association scientific statement establishing formal surveillance recommendations for this reason [1]. Gaynor and colleagues, in a large multicenter cohort followed after infant cardiac surgery, found that neurodevelopmental outcomes at school age were influenced by a combination of preoperative, intraoperative, and postoperative factors rather than any single exposure, a finding consistent with the layered injury pattern that preoperative and postoperative ultrasound and MRI together attempt to characterize [2].

Owen and colleagues linked smaller brain volume on quantitative newborn MRI directly to abnormal neurobehavioral examination findings in the newborn period, a relationship that ultrasound, with its comparatively limited volumetric precision, cannot replicate but that it can flag indirectly through qualitative impressions of ventricular size and extra-axial space [29]. Claessens and colleagues' review of neuroimaging and functional outcome in CHD reinforces that the field has moved toward viewing brain injury and dysmaturity as a continuum best captured longitudinally, across the fetal, preoperative, postoperative, and interstage periods, rather than through any single scan [30].

7. Practical protocols and the case for a layered surveillance strategy

Given cranial ultrasound's strengths in hemorrhage and ventricular size assessment and its weaknesses in white matter and cortical detail, most contemporary CHD neuroimaging protocols use it as the first-line, repeatable bedside tool within a layered strategy rather than as a standalone diagnostic test. A typical protocol obtains a baseline scan shortly after birth or admission, a preoperative scan close to the time of surgery, a postoperative scan in the first several days after bypass or circulatory arrest, and additional scans triggered by new neurological signs, abnormal vital sign trends, or extracorporeal membrane oxygenation support [10,26]. MRI, where available, is reserved for infants in whom ultrasound findings are ambiguous, in whom a more precise characterization of white matter or cortical injury would change management, or as part of a structured research or quality-improvement protocol examining the full burden of dysmaturity and injury across a cohort [11,31].

Khalil and colleagues' systematic review and meta-analysis of brain abnormalities in CHD, spanning fetal and postnatal imaging modalities, found consistent associations between structural brain findings and later

neurodevelopmental delay across study designs, supporting continued investment in neuroimaging surveillance broadly even as the review notes substantial heterogeneity in which modality individual centers use [32]. Taken together, the evidence supports cranial ultrasound as an accessible, repeatable, bedside-compatible screening tool that should anchor CNS surveillance in every CHD neonate, while MRI remains the reference standard for detailed characterization of white matter and cortical injury when it is clinically indicated and logistically feasible.

Table 2: Representative layered neuroimaging surveillance protocol for CHD neonates, combining cranial ultrasound with selective MRI [10,26,11,31].		
Timing	Modality	Primary purpose
Birth/admission	Baseline cranial ultrasound	Establish pre-surgical CNS status
Preoperative	Cranial ultrasound (+/- MRI if abnormal)	Confirm stability before bypass, guide timing
Postoperative (24-72h)	Cranial ultrasound	Screen for acute hemorrhage after bypass/arrest
New neurological sign	Urgent cranial ultrasound +/- MRI	Characterize acute event
Interstage/discharge	MRI where indicated	Detailed white matter/cortical injury assessment

8. Conclusion

Cranial ultrasound retains a well-defined and clinically important role in the neurological evaluation of neonates with congenital heart defects, chiefly as a portable, repeatable, sedation-free screening tool for hemorrhage, ventriculomegaly, and larger structural abnormalities across the preoperative, postoperative, and interstage periods [19,10,25]. Its central limitation, a comparatively low sensitivity for the diffuse white matter and subtle cortical injury that dominates the CHD-associated brain injury spectrum, means it cannot substitute for MRI when detailed characterization of injury burden is clinically or prognostically required [20,21,22]. The evidence base most strongly supports a layered surveillance model:

cranial ultrasound as the first-line, always-available bedside test that establishes baseline status, tracks evolving hemorrhagic and ventricular findings, and triggers escalation, combined selectively with MRI for infants in whom more detailed structural or volumetric information would meaningfully change counseling or management [11,30,32]. As neurodevelopmental outcome continues to emerge as the dominant long-term concern in surgically palliated CHD, refining exactly when, how often, and in which infants each imaging modality should be deployed remains an active and clinically consequential area for further study.

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