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Review Article

Neurodevelopmental and Psychiatric Outcomes in Children With Congenital Heart Disease: A Clinical Review

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Congenital heart disease (CHD) is one of the most common major congenital anomalies, and improved survival has shifted clinical attention toward long-term developmental, cognitive, behavioral, and psychiatric outcomes. This clinical review synthesizes major scientific statements, collaborative neurodevelopmental recommendations, cohort data, meta-analyses, and neuroimaging studies relevant to children with CHD. The evidence consistently shows that neurodevelopmental vulnerability is not a rare secondary complication. Children with complex CHD, chronic cyanosis, infant cardiac surgery with cardiopulmonary bypass, single-ventricle physiology, genetic syndromes, prematurity, perioperative neurologic complications, feeding or growth difficulty, and socioeconomic adversity have an elevated risk for developmental delay, lower average cognitive performance, executive dysfunction, language weakness, motor impairment, academic difficulty, anxiety, depression, and attention-deficit/hyperactivity disorder. The pathophysiology is cumulative and begins before surgery, with fetal cerebral dysmaturation, preoperative white matter vulnerability, perioperative hemodynamic and inflammatory stress, and post-discharge environmental and family factors all contributing to the outcome. Clinically, CHD care should include a parallel neurodevelopmental and mental health pathway. High-risk children require early identification, age-windowed assessment, timely referral to early intervention and rehabilitation services, school-based support, neuropsychological evaluation when indicated, and routine screening for internalizing and attentional symptoms. A practical care model should connect fetal and neonatal risk recognition with infancy surveillance, preschool reassessment, school-age educational planning, adolescent mental health screening, and transition preparation. The goal is not only to detect impairment after it becomes obvious but also to anticipate risk, intervene early, and preserve function across childhood and adolescence.

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Introduction

Congenital heart disease (CHD) affects about 8 to 10 per 1,000 live births worldwide and remains the most common major congenital anomaly [1]. Advances in fetal

diagnosis, neonatal stabilization, cardiopulmonary bypass, intensive care, staged palliation, catheter-based reintervention, and longitudinal cardiology follow-up have shifted CHD from a frequently fatal diagnosis to a chronic lifespan condition for most children [2][3]. This survival success has changed the clinical endpoint. Cognition, language, motor function, school participation, mental health, and family functioning now shape long-term outcomes as directly as anatomy and ventricular function [2][3]. The burden is substantial. In a 2016 cohort of 3,147 publicly insured children who underwent infant heart surgery, the cumulative prevalence of at least one neurodevelopmental diagnosis by 5 years of age was 51.7%, and 82.9% had received at least one neurodevelopmental service [4]. Screening was more common than in general pediatric samples, but only 6.6% underwent psychological or neuropsychological evaluation and only 8.5% underwent a comprehensive developmental evaluation [4]. This gap suggests that need is common while formal assessment remains underused. The phenotype is also quantifiable. In a meta-analysis of school-aged children operated on for CHD, the summary estimate for total IQ was 96.03, below the population mean of 100, and executive function was moderately worse than in controls, with a standardized mean difference of -0.56 [5]. Preschool children with critical CHD have lower language performance than population norms, and motor impairment affects approximately one-third of children across infancy, childhood, and adolescence [6] [7]. The psychiatric burden is also increased; in one pediatric CHD cohort, 18.2% of patients had an anxiety or depression diagnosis or had been prescribed treatment, compared with 5.2% of peers without CHD [8]. Academic consequences are evident, with children with complex CHD at increased risk for substandard academic outcomes and special educational service use [2][9]. This review is intended for clinical use. It begins with a targeted methodologic statement, then organizes the evidence around epidemiology, pathophysiology, risk stratification, age-based evaluation, referral pathways, school supports, mental health care, and practical management. The central question is not whether neurodevelopmental risk exists in CHD; the evidence already answers that. The more useful question is how clinicians should identify high-risk children early, route them to appropriate services, and keep families connected to care long enough for intervention to matter. Accordingly, the aim of this targeted narrative clinical review is to provide pediatricians and pediatric cardiologists with a practical framework for neurodevelopmental and psychiatric surveillance, risk recognition, and timely referral for intervention in children with CHD.

Methods and Search Strategy

This is a targeted narrative clinical review rather than a systematic review or meta-analysis. The review was structured around major scientific statements, collaborative clinical recommendations, cohort studies, systematic reviews, meta-analyses, and neuroimaging literature relevant to neurodevelopmental and psychiatric outcomes in children with CHD. A focused literature search and source update were performed using PubMed/MEDLINE and Google Scholar, with emphasis on literature available through May 2026. Search concepts included congenital heart disease, critical congenital heart disease, neurodevelopmental outcomes, neuropsychological evaluation, executive function, language outcomes, motor impairment, brain MRI, white matter injury, early intervention, school outcomes, anxiety, depression, ADHD, and mental health. Priority was given to American Heart Association scientific statements, Cardiac Neurodevelopmental Outcome Collaborative recommendations, meta-analyses, systematic reviews,

large cohort studies, and fetal/neonatal neuroimaging studies. Articles were selected for clinical relevance to risk recognition, counseling, surveillance, referral timing, evaluation, and management across pediatric primary care, cardiology, neurodevelopmental, educational, and mental health settings. The cited studies were retained when they provided directly usable clinical recommendations or quantitative estimates for prevalence, service use, cognition, executive function, language, motor impairment, psychiatric diagnoses, academic outcomes, fetal/neonatal brain findings, or age-specific evaluation. Because this was a narrative review, no formal risk-of-bias scoring, pooled analysis, or PRISMA flow diagram was performed.

Epidemiology and Clinical Phenotype

The most clinically useful epidemiologic data are those that guide follow-up. CHD is common enough that pediatricians, neonatologists, intensivists, cardiologists, developmental pediatricians, psychologists, therapists, and school teams will all encounter affected children [1]. Neurodevelopmental difficulty should not be framed as a rare complication limited to a few syndromic or perioperative outliers. It is a predictable part of the disease burden, especially in children with complex lesions, chronic cyanosis, infant surgery requiring cardiopulmonary bypass, single-ventricle physiology, prematurity, genetic syndromes, perioperative neurologic complications, feeding or growth difficulty, recurrent hospitalization, or socioeconomic adversity [2][3].

CHD should not be read as a single-risk category. It ranges from minor lesions such as small septal defects or mild valve disease to critical lesions requiring neonatal intervention or staged palliation. Most children with CHD do not have severe lesions, neonatal bypass exposure, or demonstrated fetal cerebral impact; therefore, the risk framework in this review is most applicable to children with complex CHD, chronic cyanosis, infant cardiopulmonary bypass, single-ventricle physiology, prematurity, genetic syndromes, perioperative neurologic complications, or persistent medical and social risk modifiers [2][3].

A domain-based phenotype is most useful clinically. Cognitively, many children with CHD do not have profound global intellectual disability, but the mean shifts downward and the lower tail widens, so more children fall into a range that affects classroom performance and adaptive function [5]. Executive deficits are particularly important because they drive many symptoms reported by families and teachers: slow processing, weak working memory, poor organization, reduced cognitive flexibility, distractibility, inconsistent task completion, and difficulty managing multistep demands [2][5]. Language weakness may be missed until preschool or early school demands increase. A child may have adequate basic conversation but still struggle with vocabulary growth, expressive formulation, narrative structure, comprehension of complex instructions, and later literacy [6]. Motor impairment may appear earlier through delayed head control, poor truncal stability, delayed walking, balance problems, weak coordination, or slow graphomotor skills [7]. Psychiatric and behavioral symptoms often coexist with these developmental vulnerabilities, especially anxiety, depressive symptoms, attention-deficit/hyperactivity disorder (ADHD), and emotional dysregulation [8][10]. The clinical implication is direct: if a child with CHD struggles in school, fatigues in therapy, has weak organization, shows delayed language, appears anxious, or falls behind in motor milestones, these findings should not be dismissed as unrelated or unexpected. They are part of the recognized risk profile

and should prompt structured evaluation rather than reassurance alone [2][3][4][5][6][7][8][9].

Measure	Best-supported number	Clinical implication
CHD birth prevalence	About 8–10 per 1,000 live births [1]	Developmental surveillance should be expected, not exceptional.
Any neurodevelopmental diagnosis by age 5 after infant heart surgery	51.7% [4]	More than half of children in a recent cohort had a documented diagnosis by age 5.
Any neurodevelopmental service by age 5 after infant heart surgery	82.9% [4]	Service use was more common than formal diagnosis.
Psychological or neuropsychological evaluation by age 5 after infant heart surgery	6.6% [4]	Formal evaluation remains underused relative to burden.
Comprehensive developmental evaluation by age 5 after infant heart surgery	8.5% [4]	Many high-risk children are not receiving a full assessment.
Summary estimate for total IQ after CHD surgery	96.03 [5]	Average cognition is shifted downward enough to affect school performance at the population level.
Executive function vs controls	Standardized mean difference −0.56 [5]	Executive weakness is common and clinically meaningful.
Preschool language performance	Overall SMD −0.46; expressive −0.45; receptive −0.32 [6]	Language weakness is measurable before formal school age.
Motor impairment	Approximately one-third of children have delayed motor skills [7]	Motor surveillance should be routine.
Anxiety/depression in one pediatric CHD cohort	18.2% in CHD vs 5.2% in peers without CHD [8]	Mental health burden is high enough to justify routine screening.
Academic outcomes in complex CHD	Approximately 25% higher risk of substandard academic outcomes and 50% greater likelihood of needing special educational services [2]	School planning should be proactive, not reactive.

Table 1. Selected epidemiologic and outcome estimates relevant to clinical practice

Stepwise Pathophysiology of Neurodevelopmental Vulnerability

The pathophysiology is best understood as cumulative rather than as a single perioperative event. Step 1: Fetal cerebral dysmaturation begins before surgery. Complex CHD can alter fetal circulation and reduce cerebral oxygen and substrate delivery during a critical period of brain growth. Fetal MRI studies have shown smaller brain volumes and abnormal brain metabolism in fetuses with CHD, and fetal brain volume has been associated with later neurodevelopmental outcomes [11][12][13]. Delayed brain maturation may therefore be detectable before complete surgical recovery and, in some infants, before surgery [12][13]. Step 2: The newborn brain may enter surgery already vulnerable. Preoperative MRI studies have demonstrated clinically silent brain injury before congenital heart surgery, and postoperative imaging has shown new lesions after surgery in infancy [14]. More recent data suggest that postoperative neonatal brain injury has declined over time, which is encouraging, but preoperative white matter vulnerability and developmental immaturity remain important [15]. Step 3: Perioperative stress adds hemodynamic and inflammatory risk. Cardiopulmonary bypass, deep hypothermic circulatory arrest, embolic load, altered cerebral perfusion, reperfusion injury, seizures, low cardiac output, and prolonged intensive care may worsen the developmental trajectory [2][3][14][15]. The phenotype is therefore not explained by one lesion, one operation, or one MRI abnormality. It reflects cumulative exposure across fetal life, surgery, and recovery. Step 4: Chronic physiology and environment continue to shape outcomes after discharge. Cyanosis, heart failure physiology, recurrent hospitalization, feeding difficulty, poor sleep, limited activity tolerance, and missed school can interfere with learning and development [2][3]. Social context modifies the final phenotype. Socioeconomic strain, reduced access to developmental services, and parental anxiety or depression can amplify risk, whereas coordinated follow-up, early intervention, and family support may improve functional outcomes [2][8][10].

Risk Stratification: Which Children Should Raise Early Concern?

The 2024 American Heart Association scientific statement is clinically useful because it organizes risk into categories that can be applied at the bedside [2]. Two groups should be treated as high risk even before formal testing: children who undergo cardiac surgery with cardiopulmonary bypass during infancy and children with chronic cyanosis [2]. In practice, many infants with single-ventricle physiology, transposition physiology requiring neonatal repair, truncus arteriosus, interrupted aortic arch, complex arch lesions, and other major neonatal operations should enter a neurodevelopmental surveillance pathway from the outset. Risk increases further with genetic syndromes or pathogenic variants, fetal or neonatal brain injury on neuroimaging, prematurity or low birth weight, perioperative seizures, cardiopulmonary resuscitation, extracorporeal membrane oxygenation or ventricular assist support, transplantation, persistent feeding delay, growth failure, socioeconomic disadvantage, and major parental psychological distress [2]. These factors matter because they lower the threshold for formal neurodevelopmental referral, early intervention enrollment, therapy, school planning, and mental health support. A practical mental checklist is useful: infant bypass surgery; chronic cyanosis; single-ventricle pathway; genetic syndrome;

prematurity; postoperative seizures or brain injury; persistent feeding or growth problems; school difficulty; and anxiety, depression, or ADHD symptoms. If any of these are present, routine developmental surveillance alone is usually insufficient [2][10][16][17].

Evaluation Pathway Across the Continuum of Care

The most useful evaluation pathway follows the child's clinical course rather than organizing each decision by test name. Prenatal and fetal stage. When CHD is diagnosed prenatally, the first clinicians involved are usually maternal-fetal medicine, fetal cardiology, and obstetrics. Their initial neurodevelopmental role is to identify lesions likely to require neonatal intervention, counsel the family that brain vulnerability may begin before birth, and plan delivery in a center that can provide neonatal cardiology, cardiac intensive care, and surgical support when needed [2][11][12][13]. The goal at this stage is risk recognition, family preparation, and avoidance of preventable perinatal instability. Neonatal hospitalization and cardiac intensive care. The lead teams are neonatology, cardiology, cardiac surgery, cardiac intensive care, bedside nursing, feeding specialists, and rehabilitation services when available. Their role is to classify neurodevelopmental risk, document neurologic complications, monitor feeding and growth, minimize avoidable hypoxemia and hemodynamic instability, and begin family-centered developmental care in the intensive care environment [2][15][16]. Neurology should be involved when seizures, abnormal examination findings, unexplained encephalopathy, or significant neuroimaging abnormalities are present. Discharge planning and the first year. This is a common point of system failure. Before discharge, the team should determine whether the infant needs referral to a cardiac neurodevelopmental follow-up program, state early intervention services, feeding therapy, physical therapy, occupational therapy, speech-language pathology, nutrition, social work, or psychology [2][16]. The birth-through-5 Cardiac Neurodevelopmental Outcome Collaborative (CNOC) recommendations are useful because they specify what a first-year assessment should include: a detailed medical and social history, family stress and supports, a neurologic examination, a feeding and sleep review, developmental progress across domains, and actionable therapy recommendations [16]. Structured follow-up in infancy and preschool. CNOC recommends formal neurodevelopmental evaluation windows centered at approximately 6 months, 18 months, 36 months, and 60 months [16]. These ages align with practical decision points: early motor and feeding delay, toddler language and behavior, preschool readiness, and school entry. In the United States, the transition around age 3 years is especially important because public school systems assume a major role in determining educational services after early intervention programs end [16]. School age and adolescence. The clinical questions change. A child who appeared to be developing adequately in toddlerhood may later show inattention, weak working memory, slow reading, organizational difficulty, anxiety, social withdrawal, or depressive symptoms when academic and social demands rise. CNOC recommends a formal school-age neurodevelopmental evaluation that integrates medical history, school records, family report, and direct testing, with referral to a cardiac neurodevelopmental follow-up program when available [17]. Adolescence should be treated as a second high-risk window because executive demands rise, mental health burden becomes more visible, adherence becomes more autonomous, and transition planning begins [2][17][10].

Setting/age	Who usually leads	Key actions	Clinical rationale
Prenatal diagnosis	Fetal cardiology, maternal-fetal medicine, obstetrics	Identify high-risk lesions; counsel family; plan delivery location and postnatal resources.	Brain vulnerability may start before surgery; risk recognition should start before birth [2][11][12][13].
Neonatal hospitalization/CICU	Cardiology, cardiac surgery, neonatology/CICU, bedside nursing; neurology when indicated	Classify neurodevelopmental risk; document seizures/brain injury; monitor feeding, growth, and recovery; involve therapy and feeding teams early.	High-risk status becomes explicit, and secondary injury may still be reduced [2][15][16].
Discharge to first year	Cardiology plus primary care; early intervention coordinator; rehabilitation and feeding specialists	Place referrals before delay is entrenched; connect family to Early Intervention, PT/OT/SLP, feeding therapy, and social work as needed.	Missed referrals at discharge lose time during a highly plastic period [2][16].
6, 18, 36, and 60 months	Cardiac neurodevelopment clinic, developmental pediatrics, psychology/neuropsychology, rehabilitation	Perform formal age-windowed evaluation; adjust therapies; document school-readiness needs.	CNOC windows align with major developmental transitions [16].
School age	Neuropsychology/psychology, developmental pediatrics, school team, cardiology, primary care	Assess attention, executive function, language, reading, mathematics, behavior, and adaptive skills; support IEP/504 planning.	Problems often become clearer when classroom demands increase [5][6][9][17].
Adolescence	Cardiology, psychology/psychiatry, neuropsychology, primary care, school team	Screen for anxiety, depression, ADHD, school difficulty, adherence problems, and transition readiness.	Mental health and executive burdens often rise in adolescence [2][8][17][10].

Table 2. CHD neurodevelopment pathway: responsibilities, timing, and clinical rationale

Evaluation and Workup in the Clinic, Hospital, and School

Evaluation should be domain-based and setting-based. It begins with risk identification rather than indiscriminate testing. Step 1: Decide whether the child needs routine surveillance or formal referral. Any child with infant bypass surgery, chronic cyanosis, a genetic syndrome, prematurity, a perioperative neurologic complication, feeding delay, growth failure, school difficulty, or an emotional/behavioral concern should move quickly from surveillance to formal evaluation [2][16][17]. Step 2: Ask focused questions by domain. In infancy, ask about head control, rolling, sitting, crawling, walking, use of both hands, feeding endurance, weight gain, sleep, and early communication. In preschool, ask about vocabulary growth, phrase use, play skills, frustration tolerance, attention, and behavior. In school-age children and adolescents, ask directly about reading, mathematics, writing, test time, attention, organization, memory, fatigue, anxiety, mood, peer relationships, and school accommodations [5][6][7][8][9][16][17]. Step 3: Examine with development in mind. This means more than a cardiology examination. Observe tone, asymmetry, postural control, balance, coordination, fine motor skill, language output, affect, attention, and adaptive independence. A child who answers questions politely in clinic may still have executive dysfunction that becomes obvious only when school records or parent reports are reviewed [5][16][17].

Step 4: Review modifiers that may be attributed incorrectly to CHD alone. Hearing and vision problems, sleep disturbance, feeding disorders, iron deficiency, repeated absences from school, family stress, and undertreated anxiety or depression can worsen neurodevelopmental performance and should be addressed in parallel [2][16][17][10]. Step 5: Match the workup to the problem. Gross motor delay should lead to physical therapy and often occupational therapy. Fine motor or visuomotor weakness should lead to occupational therapy. Language delay should lead to a speech-language evaluation. Broad school difficulty, slow processing, executive dysfunction, or uncertainty about learning style should lead to a neuropsychological or psychoeducational assessment. Anxiety, depression, trauma symptoms, or severe irritability should trigger a psychology or psychiatry referral. Feeding fatigue, oral aversion, poor weight gain, or prolonged mealtimes should trigger a feeding and nutrition evaluation [2][16][17][10]. Step 6: Use the school system deliberately. Many children with CHD need either an individualized education program (IEP) or a Section 504 accommodation plan, depending on the main problem. The clinician's role is not to write the school plan in isolation but to describe the child's functional profile clearly enough that the school can act: slower processing speed, reduced working memory, attentional dysregulation, fatigue, weak handwriting, language disorder, frequent medical absences, anxiety, or need for therapy access [9][16][17].

Management: Referrals, Therapies, Mental Health Care, and Supportive Interventions

Management is not a single medication or clinic visit. It is a coordinated package of services matched to the child's deficits and family context. Developmental and rehabilitation therapies. Physical therapy is the core intervention for delayed gross motor skills, reduced endurance, poor balance, and coordination problems. Occupational therapy addresses fine motor skills, visuomotor integration, self-

care, sensory regulation, and sometimes feeding. Speech-language therapy addresses expressive and receptive language, pragmatic communication, and later literacy-related weaknesses. These therapies are not adjuncts reserved only for the most affected children; they are the main treatment when a delay is identified [7][16][17]. Early intervention and school supports. For infants and toddlers, the usual route is Early Intervention or equivalent public developmental services. After age 3 years, school districts become central to evaluation and service delivery. Many children need periodic re-referral because needs change with age. A child discharged from physical therapy at 18 months may later need occupational therapy for handwriting or neuropsychology for executive dysfunction at 8 years [16][17]. Psychological therapies. Cognitive-behavioral therapy is the most evidence-based psychotherapeutic approach for anxiety and depression in CHD and should be considered first-line when symptoms are impairing and the child can engage developmentally [10]. Family-based work is often as important as individual therapy because parental anxiety, avoidance, and medical trauma can maintain symptoms in the child [10].

Medication for anxiety and depression. When psychotherapy alone is insufficient or symptoms are moderate to severe, selective serotonin reuptake inhibitors are generally preferred first-line pharmacologic options in pediatric anxiety and depression. In CHD, medication decisions should be individualized with attention to rhythm history, baseline QTc, concomitant QT-prolonging medications, electrolytes, hepatic dysfunction, ventricular function, and heart failure status when relevant [10]. The pediatrician, child psychiatrist, or developmental-behavioral clinician typically prescribes and titrates medication, while cardiology helps interpret lesion-specific or rhythm-specific safety concerns. Medication for ADHD. Psychostimulants and nonstimulant ADHD medications are not categorically contraindicated across CHD. The approach should be individualized: review blood pressure, heart rate, exercise symptoms, arrhythmia history, ventricular dysfunction, dynamic outflow obstruction, prolonged QT, and lesion-specific concerns; involve cardiology more closely when these are present; then weigh the academic and behavioral consequences of undertreated ADHD against the usually modest hemodynamic effects of medication [10]. Family and social interventions. Social work, care coordination, and parent mental health support should be considered part of neurodevelopmental management. Transportation barriers, missed therapy because of insurance or scheduling problems, school misunderstanding of CHD-related fatigue, and untreated parental anxiety can derail an otherwise appropriate care plan [2][8][10].

Clinical problem	Most appropriate referral/intervention	What it treats
Delayed sitting or walking, weak balance, poor endurance	Physical therapy	Gross motor delay, postural control, coordination, activity tolerance
Poor handwriting, clumsy fine motor work, weak visuomotor skills, difficulty with dressing/self-care	Occupational therapy	Fine motor skills, visuomotor integration, self-care, regulation
Late words, weak sentence formation, poor narrative skills, language-based learning concerns	Speech-language therapy	Expressive/receptive language, pragmatic communication, literacy foundations
Slow reading, mathematics difficulty, weak attention/organization, inconsistent classroom performance	Neuropsychology or school psychoeducational evaluation; IEP/504 planning	Executive function, learning profile, academic accommodations
Anxiety, depression, avoidance, medical trauma, poor coping	Psychology/cognitive-behavioral therapy; psychiatry if needed	Internalizing symptoms, coping, functional participation
ADHD symptoms impairing school or home function	Behavioral supports plus ADHD medication when indicated, with cardiology context if higher-risk lesion or rhythm history	Attention, impulsivity, school function
Feeding fatigue, oral aversion, poor growth	Feeding therapy, nutrition, sometimes occupational/speech involvement	Oral-motor skills, intake, growth, parent-child mealtime stress
Family overwhelm, missed appointments, social barriers	Social work, care coordination, parent mental health referral	Adherence, access, family functioning

Table 3. Selected interventions and common indications

Clinical Care Sequence

A structured sequence can reduce the risk that children are lost between cardiology follow-up and neurodevelopmental care.

1. Identify the lesion and risk category early. Any infant surgery with bypass or chronic cyanosis should immediately raise neurodevelopmental concern [2].
2. Ask about development and mental health at every cardiology and primary care visit, not only when the family volunteers concerns [2][16][17].
3. Refer early rather than waiting for severe delay. In CHD, delayed referral often results in lost developmental time [4][16].
4. Reassess at developmental transition points: infancy, toddlerhood, preschool, school entry, and adolescence [16][17].

5. Treat the domains that are impaired: physical therapy for gross motor delay, occupational therapy for fine motor or adaptive skills, speech-language therapy for language, school services for learning, and psychotherapy or medication when needed for mental health [16][17][10].
6. Keep the family in the plan. A technically correct referral that the family cannot access is unlikely to be effective [2][10].

Clinical Implications

The practical implication is that CHD follow-up should not be limited to anatomy, oxygen saturation, ventricular function, arrhythmia surveillance, and medication adjustment. Developmental and psychiatric risk should be visible in the problem list and revisited at predictable developmental transitions. The cardiology visit, primary care visit, cardiac intensive care discharge, early intervention evaluation, school evaluation, and adolescent transition visit should function as connected points in one pathway rather than as separate systems. For the pediatrician, the central tasks are to recognize high-risk physiology, ask domain-specific developmental questions, document functional concerns clearly, and route the child to early intervention, school evaluation, therapy, psychology, psychiatry, or neuropsychology when indicated. For cardiology, the task is to identify lesion-specific risk, normalize developmental surveillance as part of cardiac care, and help other clinicians interpret cardiac safety issues when psychiatric or ADHD medications are considered.

Limitations

This review has limitations. It is a narrative clinical review and not a systematic review or meta-analysis. The literature was selected for clinical relevance rather than through a formal protocol with duplicate screening, risk-of-bias assessment, or pooled quantitative synthesis. The studies cited vary in lesion complexity, age at surgery, era of surgical care, neurodevelopmental instruments, follow-up duration, insurance status, socioeconomic context, and definitions of impairment. These differences limit direct comparisons across cohorts. The review also emphasizes pediatric clinical application and therefore does not fully address adult congenital heart disease, adult neurocognitive aging, health economics, or implementation science. In addition, many available studies describe associations rather than causal pathways, and some domains, such as family trauma, sleep, school absenteeism, and real-world therapy access, remain less consistently measured than cognitive or motor outcomes.

Conclusion

Children with CHD should be managed as children with a parallel cardiac and neurodevelopmental condition. The epidemiologic burden is sufficient to justify that shift: developmental diagnoses, therapy use, cognition, executive function, language, motor skills, school performance, and mental health are all affected often enough to require routine attention. A useful clinical model is sequential. Fetal and neonatal risk recognition should be followed by structured infancy surveillance, preschool reassessment, school-age neuropsychological and educational evaluation when indicated, and routine mental health attention in adolescence. The goal is not simply to document impairment; it is to identify the right child at the right time, route that child to the right service, and keep the family connected to care long enough for intervention to matter.

Statements and Declarations

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Potential Competing Interests

The author declares no potential competing interests.

Ethics

This review used publicly available published literature and did not involve direct human participant enrollment, patient records, identifiable patient information, animal subjects, or new experimental data. Institutional review board approval and informed consent were not applicable.

Data Availability

No new datasets were generated or analyzed for this review. All information discussed in this article is derived from publicly available published literature cited in the reference list.

Author Contributions

Abenezer F. Kebede: Conceptualization, literature review, interpretation of the literature, writing—original draft, writing—review and editing, final approval of the submitted version, and accountability for all aspects of the work.

Use of Generative AI

During manuscript preparation, the author used OpenAI ChatGPT for language clarity, organization, and formatting support. The author independently reviewed, edited, and verified the scientific content, references, and final wording and takes full responsibility for the accuracy, originality, and integrity of the manuscript. No generative AI tool was listed as an author, and no generative AI tool was used to create or alter images.

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