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NỘI DUNG

- Tổng quan trẻ sơ sinh TBS nguy kịch
- Chẩn đoán sớm, kịp thời
- Hồi sức trước phẫu thuật
- Giảm thiểu nguy cơ: nhiễm khuẩn, tổn thương não

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TỔNG QUAN

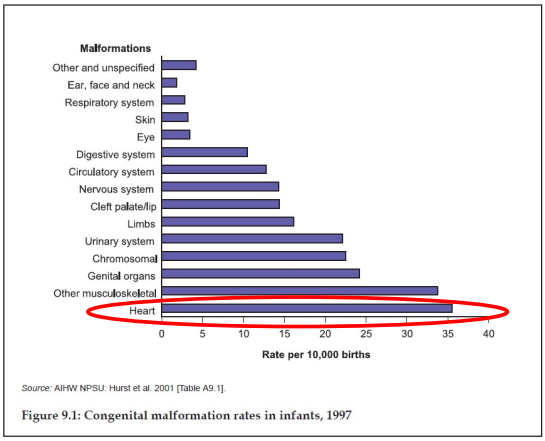

Figure 9.1: Congenital malformation rates in infants, 1997

- In 1997, malformations of the heart were the most common congenital malformations (35.5 per 10,000 births, accounting for 20% of all congenital malformations). These were followed by malformations of the musculoskeletal system (33.7, or 19%), malformations of the genital organs (24.2, or 14%), chromosomal malformations (22.5, or 13%), and malformations of the urinary system (22.1, or 13%).

- Tim bẩm sinh: DTBS thường gặp nhất, tần suất 6 – 8 / 1000 trẻ sanh sống
- Tử vong do TBS chiếm 3% tử vong trẻ em
- Can thiệp sớm giúp giảm TV sơ sinh do TBS từ 2 → 0,6 – 0,8 / 1000 trẻ sanh sống
- 3 nhóm chính của TBS theo phương diện LS:
  - Life-threatening CHD:
  - Clinically significant CHD
  - Clinically non-significant CHD

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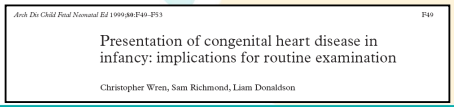
Chẩn đoán TBS

- 33% trẻ TBS trong gđ sơ sinh có biểu hiện triệu chứng không do tim trước
- Chẩn đoán sót: giai đoạn SS: 50%, 6 tuần tuổi: 33%
- Ba bệnh cảnh chính của trẻ TBS nặng
  - Tím (tắc nghẽn hoặc hạn chế máu lên phổi)
  - Sốc (tắc nghẽn máu nuôi cơ thể)
  - Suy tim (quá tải)
- Nghĩ đến nguyên nhân TBS trong bệnh cảnh trẻ nặng

Table 1. Ductus Dependent CHDs

Left sided obstructive lesions (ductus dependent systemic circulation)	Right sided obstructive lesions (ductus dependent pulmonary circulation)
hypoplastic left heart syndrome (HLHS)	TOF with pulmonary atresia (PA)
critical aortic stenosis (AS)	pulmonary atresia (PA)
"shone" complex variants	pulmonary atresia with intact ventricular septum (PA /IVS)
coarctation of the aorta (COA)	critical pulmonary stenosis (PS)
interrupted aortic arch (IAA)	tricuspid atresia, with PS/PA (with/without VSD)
	univentricular heart with PA /PS
	severe Ebsteins anomaly
	complete transposition of the great arteries* with intact ventricular septum (TGA/ IVS)


Arch Dis Child Fetal Neonatal Ed 1999;80:F49–F53 F49

Presentation of congenital heart disease in infancy: implications for routine examination

Christopher Wren, Sam Richmond, Liam Donaldson

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Sàng lọc dị tật tim trước sanh

TABLE 4-1. Indications for Fetal Echocardiography

Maternal factors
- Maternal congenital cardiovascular malformation
- Exposure to known cardiovascular teratogen (anticonvulsants, alcohol, rubella, etc.)
- Metabolic disorder (diabetes mellitus, phenylketonuria)
- Connective tissue disease
- Maternal anxiety*
- Advanced maternal age*

Familial factors
- Previous child or fetus with congenital cardiovascular malformation
- Paternal congenital cardiovascular malformation
- Family history of genetic syndrome (especially DiGeorge and related syndromes, Holt-Oram, Noonan, Marfan, Williams, long QT syndrome)
- Family history of malformation syndrome
- Family history of other birth defects

Fetal factors
- Suspected structural cardiovascular malformation on obstetrical ultrasound
- Extracardiac malformation
- Chromosomal abnormality
- Twin-to-twin transfusion syndrome
- Hydrops fetalis
- Arrhythmia
- Increased nuchal translucency

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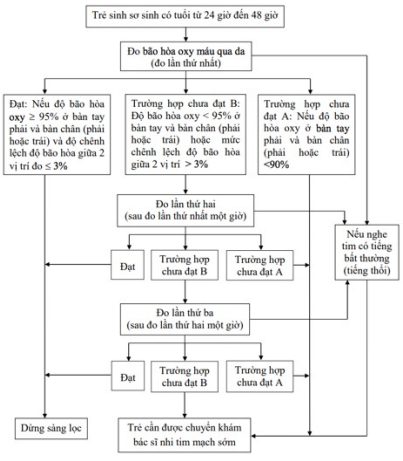
Sàng lọc dị tật tim sau sanh



- Sàng lọc SpO2 sau sanh:
  - Thời điểm đo: ≥ 24 giờ tuổi hoặc ngay trước xuất viện
  - KQ dương tính:
    - SpO2 < 90% hoặc SpO2 < 95% trong 3 lần đo cách 1 giờ
    - Khác biệt SpO2 tay P và chân > 3% trong 3 lần đo cách 1 giờ
  - Giá trị tiên đoán âm: 99,9%
  - Giá trị tiên đoán dương: 47%
    - Tim bẩm sinh
    - SHH hoặc NTSS sớm

Table 1 Congenital heart disease lesions divided by likelihood of being detected by pulse oximetry

Primary targets	Secondary targets	Possibly screenable	Not screenable
Hypoplastic left heart syndrome	Interrupted aortic arch/aortic atresia	Aortic stenosis with PDA	Coarctation of the aorta without a PDA
Pulmonary atresia	Coarctation of the aorta with PDA	Pulmonary stenosis	Ebstein's anomaly without right-to-left shunt
Total anomalous pulmonary venous connection	Ebstein's anomaly	Complete atrioventricular canal	Aortic stenosis without PDA
Transposition of the great arteries	Double-outlet right ventricle		Other left-to-right shunting lesions
Tetralogy of Fallot	Single ventricle physiology		
Tricuspid atresia			
Truncus arteriosus communis			

Notes: Primary and secondary targets are cardiac lesions that will have hypoxemia in the newborn period. Lesions are considered possibly screenable or not screenable because of lesser degree of hypoxemia.
Abbreviation: PDA, patent ductus arteriosus.

Medical Devices: Evidence and Research
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REVIEW

Pulse oximetry screening: a review of diagnosing critical congenital heart disease in newborns

This article was published in the following Dove Press journal:
Medical Devices: Evidence and Research
11 July 2014
Number of times this article has been viewed

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Abstract: Congenital heart disease (CHD) is one of the most common birth defects, with an incidence of nine out of every 1,000 live births. The mortality of infants with CHD has decreased over the past 3 decades, but significant morbidity and mortality continue to occur if not diagnosed shortly after birth. Pulse oximetry was recommended as a screening tool to detect critical CHD in 2011 by the American Academy of Pediatrics and the American Heart Association. Pulse oximetry is a tool to measure oxygen saturation, and based on the presence of hypoxemia, many

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Chẩn đoán di truyền trẻ TBS

Table 6.1 Typical cardiac anomalies associated with a syndromic diagnosis identifiable using chromosomal microarray

Syndromic		
Copy number variation		
22q11.2 deletion	DiGeorge	Left and right outflow tract
7q11.23 deletion	Williams	Supra aortic stenosis
XO	Turners	Coarctation of aorta
8p23.1 duplication		Atrioventricular septal defect Single ventricle
1q21.1 deletion		Aortic arch anomalies
Single gene anomaly		
PTPN11, RAS	Noonan	Pulmonary stenosis Hypertrophic cardiomyopathy
JAG1, NOTCH2	Alagille	Peripheral pulmonary stenosis
TBX5	Holt-oram	Atrial septal defect Ventricular septal defect
DHCR7	Smith-lemli-opitz	Atrioventricular septal defect Patent arterial duct Ventricular septal defect
TAZ	Barth	Cardiomyopathy

Table 6.2 Typical cardiac anomalies associated with a single gene anomaly identifiable using chromosomal microarray

Non-syndromic		
Familial	NOTCH1	Bicuspid aortic valve Aortopathy
	GATA4	Atrioventricular septal defect
	FBN1	Aortopathy
Mitochondial	ACAD9	Cardiomyopathy

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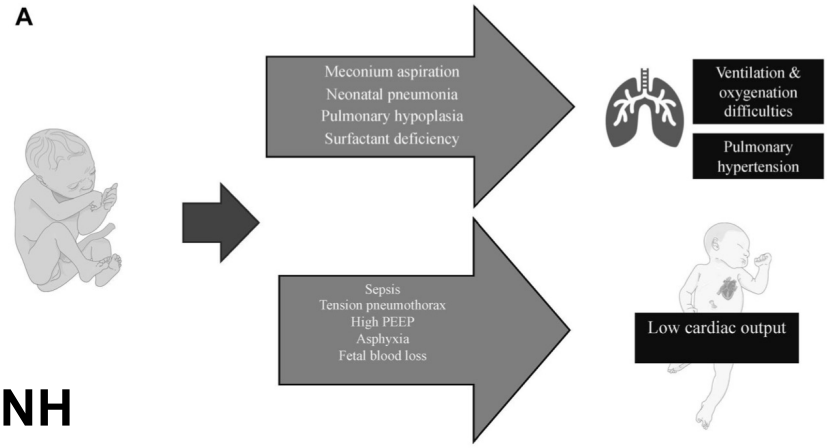
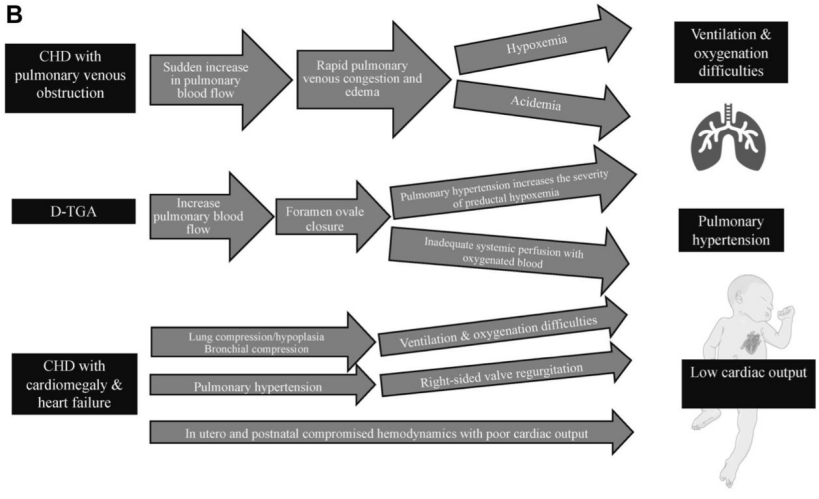
HỒI SỨC SAU SANH





Received: 30 March 2024 | Revised: 28 May 2024 | Accepted: 29 May 2024
DOI: 10.1002/pd.6417
REVIEW
PRENATAL DIAGNOSIS WILEY

Delivery room and early postnatal management of neonates with congenital heart disease

Noorjahan Ali[1] | Mary T. Donofrio[2]

CITY CHI

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Consensus Statement and Clinical Recommendations

- Neonates < 32+0 wk PMA
  - ON supplemental oxygen, target oxygen range of 90-94%
  - OFF supplemental oxygen, target oxygen range of > 90%
- Neonates ≥ 32+0 wks PMA
  - ON supplemental oxygen, target oxygen range of 92-98%
  - OFF supplemental oxygen, target oxygen range of >92%
- Neonates with Chronic Lung Disease > 36 +0 wks PMA
  - Oxygen targeting range of 92-98%

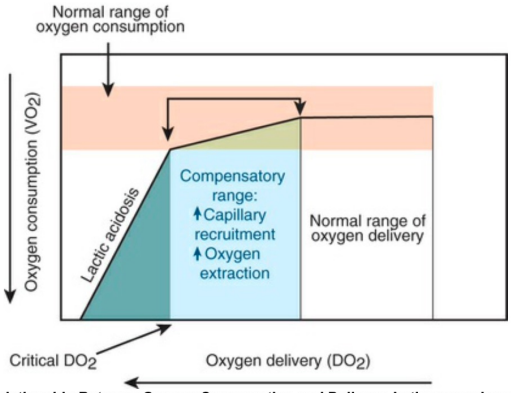

FIG. 51.4 Relationship Between Oxygen Consumption and Delivery. In the normal range of oxygen delivery, oxygen consumption is unaffected by changes in the rate of delivery of oxygen to the tissues. As oxygen delivery decreases below the normal range, tissue oxygen consumption remains in the normal range for a while because of activation of local compensatory mechanisms such as capillary recruitment and increased oxygen extraction. However, when oxygen delivery decreases to the "critical" point, compensatory mechanisms can no longer satisfy tissue oxygen demand, and anaerobic metabolism commences, resulting in significantly decreased ATP and increased lactate production.

Cyanotic heart disease and duct dependant cardiac lesions: A target range of 75- 85% is typically recommended for neonates with mixed circulation. Patient specific parameters must be determined by the treating cardiology or primary medical team.

*Limited evidence is available for a standardised oxygen saturation target range for neonates with cyanotic heart disease. Maintaining saturations >85% may be unachievable in these neonates without causing significant hyperoxia due to cardiac shunting.

Hyperoxia should be avoided in neonates with duct dependant cardiac lesions to avoid pulmonary over circulation, systemic hypo perfusion and sequelae.

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Tác dụng của các loại thuốc vận mạch trên lâm sàng

Drugs	Receptors	SVR	PVR	SV	HR	Cont-ractility	BP (MAP)	Dosage	Effects
Epinephrine	α_1 α_2, β_1 β_2	↑↑↑	↑	↑	↑↑↑	↑	↑↑	0.02 – 0.5 ug/kg/min **Start: 0.05 ug/kg/min**	↑ SVR↑ MAP ↑ contractility
Norepinephrine	α_1 α_2, β_1	↑↑↑	↑	↑	↑	No effect	↑↑	0.02 – 0.5 ug/kg/min **Start: 0.05 ug/kg/min**	↑ SVR > PVR ↑ MAP ↓ PA pressure
Dopamine	α_1 β_1 β_2 D	↑↑	↑↑↑	↑	↑↑↑	↑	↑↑	2 – 20 ug/kg/min **Start: 5 ug/kg/min**	↑ contractility ↑ SVR & PVR
Dobutamine	α_1 β_1 weak β_2	↓↓	No effect	↑↑	↑	↑	↓	5 – 20 ug/kg/min **Start: 5 ug/kg/min**	↑ contractility
Milrinone	PDE III inhibitor	↓↓	↓↓	↑↑	↑	↑	↓	0.2 – 1 ug/kg/min **Start: 0.33 ug/kg/min**	↑ contractility / ↓ SVR / PVR /↓ MAP
Vasopressin	v_1 v_2	↑↑↑	↓↓	↑	No effect	No effect	↑↑	0.1 - 1.2 miliu/kg/min **Start: 0.1 miliu/kg/min**	↑ SVR /↓ PVR ↑ MAP
Hydrocortisone		↑↑↑	No effect	No effect	↑	No effect	↑↑	1 mg/kg/dose q8 hours	↑ SVR ↑ MAP

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Hồi sức trước chuyển Trung tâm can thiệp tim mạch

Dominant physiology	Pathology	Management
Left to right shunt	AVSD, VSD, PDA, AP window	Diuretic therapy Accept saturation > 88% (usually >92%) Avoid unnecessary oxygen therapy Fluid restriction Avoid hyperventilation and alkalosis
Obstructed pulmonary veins	Obstructed TAPVR	Immediate transport for cardiac surgery
Cyanosis with unlimited PBF	Unobstructed TAPVR, Tricuspid atresia with no PS, Truncus arteriosus, single ventricle, DORV with no PS	Diuretic therapy Accept saturation > 75% Discharge after ensuring there is no significant PS and the PDA is almost closed Early follow-up in cardiac center
Cyanosis with limited BPF	Pulmonary atresia, severe PS, TOF, DORV with severe PS, Tricuspid atresia with severe PS, severe Ebstein anomaly	Prostaglandin E1 infusion Accept saturation > 75% Increase the intravascular volume Vasopressors to augment pulmonary flow Transport to a cardiac center
Parallel circulations	TGA with intact ventricular septum, DORV with malposed great arteries	Prostaglandin E1 infusion Accept saturation > 75% Evaluate the need for balloon atrial septostomy Treat pulmonary HTN aggressively if desaturated with adequate ASD. Transport to a cardiac center
Obstructed systemic circulation	HLHS, Tricuspid atresia with malposed great arteries and small VSD, Aortic stenosis, Interrupted aortic arch, Coarctation of the aorta	Prostaglandin E1 infusion Accept saturation > 75% Avoid hyperventilation, oxygen therapy and alkalosis. Fluid restriction Utilize ventilation with higher PEEP Transport to a cardiac center

Alkhushi The Cardiothoracic Surgeon (2023) 31:1
https://doi.org/10.1186/s43057-022-00090-7

The Cardiothoracic Surgeon

REVIEW Open Access

The management of newborns with critical congenital heart diseases prior to transport to a cardiac center

Naif Alkhushi[1,2,3*]

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NGUY CƠ NHIỄM KHUẨN

Early Hum Dev. 2012 May ; 88(Suppl 2): S92–S97. doi:10.1016/S0378-3782(12)70025-7.

Sepsis in Young Infants with Congenital Heart Disease

Simon B. Ascher, BS[1], P. Brian Smith, MD, MPH, MHS[1,2,*], Reese H. Clark, MD[3], Michael Cohen-Wolkowiez, MD[1,2], Jennifer S. Li, MD, MHS[1,2], Kevin Watt, MD[1,2], and Daniel K. Benjamin Jr., MD, PhD, MPH[1,2]

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[2]Department of Pediatrics, Duke University, Durham, North Carolina, USA;

[3]Pediatrix-Obstetrix Center for Research and Education, Sunrise, Florida, USA

Background—We sought to describe the incidence, pathogen distribution, and mortality associated with blood culture-proven sepsis in young infants with congenital heart disease (CHD) admitted to a neonatal intensive care unit (NICU).

Methods—Cohort study of all blood cultures obtained from infants with CHD between 4 and 120 days of age cared for in250 NICUs managed by the Pediatrix Medical Group in the United States between 1996 and 2007.

Results—Of 11,638 infants with CHD, 656 (6%) had 821 episodes of sepsis: a cumulative incidence of 71/1000 admissions. Gram-positive organisms were the most common cause (64%), and coagulase-negative *Staphylococcus* and *Staphylococcus aureus* were the most frequently isolated species. On multivariable regression, infants with sepsis were more likely to die compared to infants with sterile blood cultures (odds ratio [OR] = 1.53 [95% confidence interval: 1.09, 2.13]). Infants with gram-negative bacteremia and candidemia were more likely to die than infants with sterile blood cultures (OR = 2.01 [1.20, 3.37], and OR = 3.18 [1.60, 6.34], respectively).

Conclusion—Infants with CHD have a high incidence of culture-proven sepsis, especially with staphylococcal organisms. Gram-negative bacteremia and candidemia are strongly associated with increased mortality in this group of young infants.

Sepsis in premature newborns with congenital heart disease

Emina Hadzimuratovic [1], Senka Mesihovic Dinarevic, Admir Hadzimuratovic

Affiliations + expand

PMID: 21087428 DOI: 10.1111/j.1747-0803.2010.00406.x

Abstract

Objective: To determine the incidence and distribution of neonatal sepsis in premature newborns with congenital heart disease (CHD) according to gestational and postnatal age, time of onset, and type and frequency of causing agents, and compare it with premature newborns without CHD.

Design, setting, patients: A clinical investigation on 80 premature newborns admitted to neonatal intensive care unit (NICU) of Pediatric Clinic University Medical Center Sarajevo, Bosnia and Herzegovina, between October 23, 2007 and March 18, 2009. We analyzed the incidence and distribution of neonatal sepsis in premature newborns with CHD according to gestational and postnatal age, time of onset, and type and frequency of causing agents, and compared it with premature NICU patients without CHD.

Results: Of the 80 premature newborns included in our study, 14 had CHD with patent ductus arteriosus as the most common type of anomaly. Culture-proven sepsis was diagnosed in 28.57% premature newborns with CHD and 12.12% premature newborns without CHD. The three most common causing agents were Staphylococcus aureus, Klebsiella species, and Serratia species.

Conclusion: Premature newborns with CHD have a higher risk of acquiring sepsis during hospitalization in NICU, probably because of longer duration of hospitalization and need for invasive procedures such as mechanical ventilation, central venous catheters, and parenteral nutrition.

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NGUY CƠ TỔN THƯƠNG NÃO

Brain Injury in Neonates with Complex Congenital Heart Disease: What Is the Predictive Value of MRI in the Fetal Period?

M. Brossard-Racine, A. du Plessis, G. Vezina, R. Robertson, M. Donofrio, W. Tworetzky, and C. Limperopoulos

ABSTRACT

BACKGROUND AND PURPOSE: Brain injury in neonates with congenital heart disease is an important predictor of adverse neurodevelopmental outcome. Impaired brain development in congenital heart disease may have a prenatal origin, but the sensitivity and specificity of fetal brain MR imaging for predicting neonatal brain lesions are currently unknown. We sought to determine the value of conventional fetal MR imaging for predicting abnormal findings on neonatal preoperative MR imaging in neonates with complex congenital heart disease.

MATERIALS AND METHODS: MR imaging studies were performed in 103 fetuses with confirmed congenital heart disease (mean gestational age, 31.57 ± 3.86 weeks) and were repeated postnatally before cardiac surgery (mean age, 6.8 ± 12.2 days). Each MR imaging study was read by a pediatric neuroradiologist.

RESULTS: Brain abnormalities were detected in 17/103 (16%) fetuses by fetal MR imaging and in 33/103 (32%) neonates by neonatal MR imaging. Only 9/33 studies with abnormal neonatal findings were preceded by abnormal findings on fetal MR imaging. The sensitivity and specificity of conventional fetal brain MR imaging for predicting neonatal brain abnormalities were 27% and 89%, respectively.

CONCLUSIONS: Brain abnormalities detected by in utero MR imaging in fetuses with congenital heart disease are associated with higher risk of postnatal preoperative brain injury. However, a substantial proportion of anomalies on postnatal MR imaging were not present on fetal MR imaging; this result is likely due to the limitations of conventional fetal MR imaging and the emergence of new lesions that occurred after the fetal studies. Postnatal brain MR imaging studies are needed to confirm the presence of injury before open heart surgery.

ABBREVIATIONS: BCH = Boston Children's Hospital; CHD = congenital heart disease; CNMC = Children's National Medical Center; GA = gestational age; GMH-IVH = germinal matrix hemorrhage and intraventricular hemorrhage; NHPI = nonhemorrhagic parenchymal injury; pWMI = punctate white matter injury; SNAP = Scores for Neonatal Acute Physiology

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NGUY CƠ TỔN THƯƠNG NÃO

Pediatr Crit Care Med. 2018 January ; 19(1): 56–63. doi:10.1097/PCC.0000000000001374.

Intraventricular Hemorrhage in Moderate to Severe Congenital Heart Disease

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Abstract

Objective—Determine the incidence of intraventricular hemorrhage (IVH) in infants with moderate to severe congenital heart disease (CHD), investigate the impact of gestational age, cardiac diagnosis, and cardiac intervention on IVH, and compare IVH rates in preterm infants with and without CHD.

Methods—A retrospective review was performed from 2007–2012 of all infants admitted to St. Louis Children's Hospital with moderate to severe CHD requiring cardiac intervention in the first 90 days of life and all preterm infants without CHD or congenital anomalies/known genetic diagnoses admitted during the same time period. Cranial ultrasound (CUS) data were reviewed for presence/severity of IVH. Head computed tomography (CT) and brain magnetic resonance imaging (MRI) data were also reviewed in the CHD infants. Univariate analyses were undertaken to determine associations with IVH and a final multivariate logistic regression model was performed.

Results—There were 339 infants with CHD who met inclusion criteria and 25.4% were born preterm. IVH was identified on CUS in 13.3% of infants, with the majority of IVH being low-grade (Grade I/II). The incidence increased as gestational age decreased such that IVH was present in 8.7% of term infants, 19.2% of late preterm infants, 26.3% of moderately preterm infants, and 53.3% of very preterm infants. There was no difference in IVH rates between cardiac diagnoses. Additionally, the incidence of IVH did not increase after cardiac intervention, with only three infants demonstrating new/worsening high-grade (Grade III/IV) IVH after surgery. In a multivariate model, only gestational age at birth and African American race were predictors of IVH. In the subset of infants with CT/MRI data, there was good sensitivity and specificity of CUS for presence of IVH.

TYPE Original Research
PUBLISHED 25 April 2024
DOI 10.3389/fped.2024.1326804

Neonatal outcomes in preterm infants with severe congenital heart disease: a national cohort analysis

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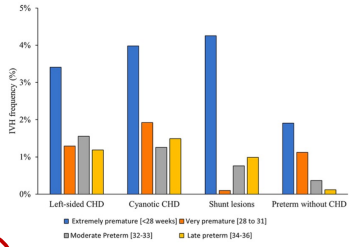
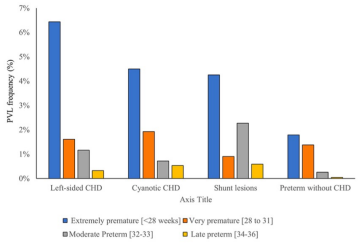
[1]Department of Cardiology, Boston Children's Hospital, Boston, MA, United States, [2]Department of Pediatrics, Harvard Medical School, Boston, MA, United States, [3]Department of Pediatrics, Nassau University Medical Center, East Meadow, NY, United States, [4]Department of Neonatology, Cleveland Clinic, Cleveland, OH, United States





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KEY MESSAGES

- Phát hiện sớm TBS đặc biệt nhóm nguy kịch giúp xử trí chủ động, kịp thời từ trước sanh đến sau sanh
- Hồi sức và xử trí TBS nguy kịch tuỳ thuộc:
  - Loại bất thường cấu trúc
  - 3 yếu tố chính: mức oxy hoá máu, lưu lượng tim và lưu lượng mạch máu phổi
  - Yếu tố bệnh lý kèm: ngạt, viêm phổi, nhiễm trùng huyết, bất thường di truyền
- Giảm thiểu các nguy cơ ảnh hưởng kết cục: nhiễm khuẩn, tổn thương não, …

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THANK YOU

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