CONGRÈS MÉDICO-CHIRURGICAL DE LA FCPC 15e

Role of prenatal diagnosis in predicting the outcomes of a new-born with congenital heart disease

Damien Bonnet on behalf of the PARSIFAL group

Unité médico-chirurgicale de Cardiologie Congénitale et Pédiatrique
Hôpital Universitaire Necker Enfants malades – APHP, Université Paris Descartes, Sorbonne Paris Cité
IcarP Cardiology, Institut Hospitalo-Universitaire IMAGINE

Centre de Référence Maladies Rares
Malformations Cardiaques Congénitales Complexes-M3C
Centre de Référence Maladies Rares
Maladies Cardiaques Héréditaires- CARDIOGEN

M3C

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Centre de référence des Maladies Cardiaques Héréditaires

European Reference Network
for rare or low prevalence complex diseases
Network Respiratory Diseases (ERN-LUNG)

European Reference Network
for rare or low prevalence complex diseases
Network Heart Diseases (ERN GUARD-HEART)

Disclosure

- **None**

The French system during pregnancy

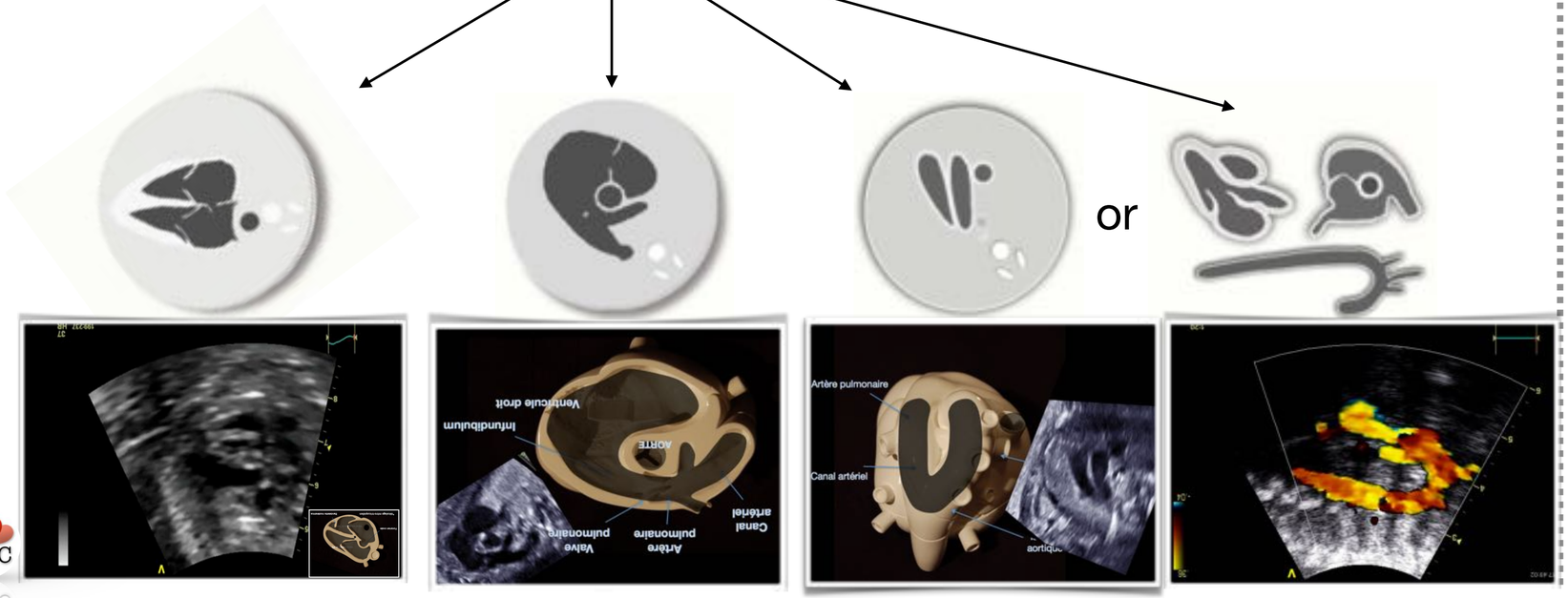
3 systematic foetal echo scans - ***Level 1***

11 Weeks **18-22 Weeks** **32-34 Weeks**

Nuchal translucency
Term
Number fetuses

Morphology

Growth
Malformations



In case of anomaly or difficulty in assessing normality

Level 2

Expert foetal echography

If heart anomaly is confirmed

Level 3

Fetal echocardiography by expert

PARSIFAL organization after fetal diagnosis of CHD

If heart anomaly is confirmed

↓

Level 3

↓

Fetal echocardiography by expert

→

Information
Diagnosis
Prognosis
Need for karyotype
Search for associated anomalies

→ Parental decision to continue pregnancy → *Level 4*

Information
Organisation of perinatal care
Decision of in utero transfer if needed

→ Parental decision to terminate pregnancy → *Level 4*

Prenatal diagnosis
national center
Multidisciplinary decision

Prevalence, pre- and post-natal diagnosis, and infant mortality of newborns with congenital heart defects
A population-based study using the International Paediatric and Congenital Cardiac Code (IPCCC)
The EPICARD Study Group 2005-08

Proportion of prenatal diagnosis

All CHDs

ACC-CHD categories	% of prenatal diagnosis
All cases excluding chromosomal anomalies	25.6
All cases excluding chromosomal and other extra cardiac anomalies	23
All cases excluding chromosomal, other anomalies and simple VSD	40.2

In categories of CHDs

ACC-CHD categories	% of prenatal diagnosis (n)
Heterotaxy	89.2 (37)
Anomalies of venous connections	16.0 (25)
Anomalies of atria	4.3 (164)
Anomalies of AV junction and AV valves	67.0 (91)
Complex anomalies of AV junction	100.0 (13)
Functionally univentricular heart	92.5 (133)
Ventricular septal defects	9.6 (1353)
Anomalies of ventriculo-arterial connections	39.2 (503)
Anomalies of extra pericardial trunks	44.7 (143)
Congenital anomalies of coronary arteries	0 (9)

Specific CHDs

Type of CHD	% of prenatal diagnosis
Congenitally corrected transposition of the great	100
Functionally univentricular heart	92.5
TGA	74
DORV	98

M3C

Khoshnood B et al. Heart 2012;98:1667-73

Prenatal diagnosis of CHD and TERMINATION OF PREGNANCY

What is the impact ?

Prevalence, pre- and post-natal diagnosis, and infant mortality of newborns with congenital heart defects:
A population-based study using the International Paediatric and Congenital Cardiac Code (IPCCC)
The EPICARD Study Group 2005-08

Proportion of Termination of pregnancy

All CHDs

ACC-CHD categories	% TOP
All cases excluding chromosomal anomalies	9.8
All cases excluding chromosomal and other extra cardiac anomalies	6.4
All cases excluding chromosomal, other anomalies and simple VSD	14.0

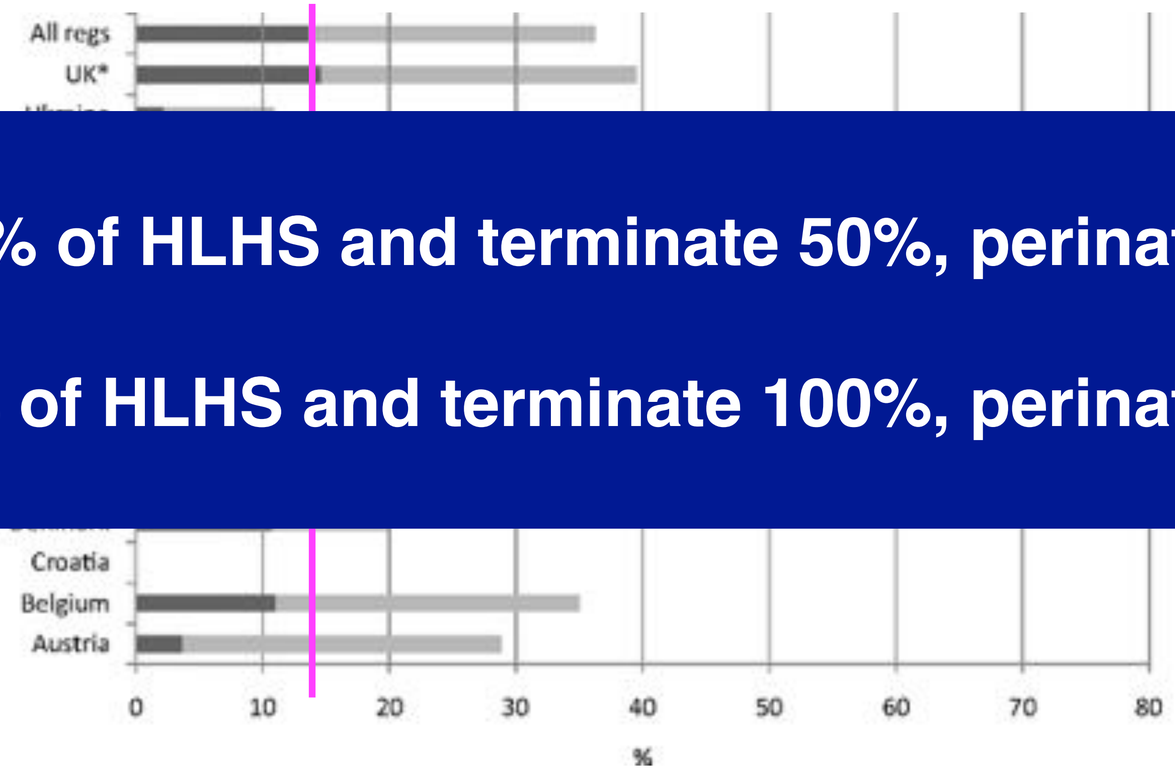
In categories of CHDs

ACC-CHD categories	% TOP
Heterotaxy	75.7
Anomalies of venous connections	16.1
Anomalies of atria	4.4
Anomalies of AV junction and AV valves	42.7
Complex anomalies of AV junction	46.2
Functionally univentricular heart	62.7
Ventricular septal defects	5.7
Anomalies of ventriculo-arterial connections	18.5
Anomalies of extra pericardial trunks	23.5
Congenital anomalies of coronary arteries	0

M3C

Khosnood B et al. Heart 2012;98:1667-73

Proportion of nonchromosomal SI/SII congenital heart defect cases prenatally diagnosed (PD)* by pregnancy outcome (terminations of pregnancy for fetal anomaly [TOPFA] or birth), by country, 2000 to 2005



If you diagnose 100% of HLHS and terminate 50%, perinatal mortality is 50%

If you diagnose 20% of HLHS and terminate 100%, perinatal mortality is 20%

Severity I (SI): single ventricle, hypoplastic left heart, hypoplastic right heart, Ebstein anomaly, and tricuspid atresia
Severity II (SII): pulmonary valve atresia, common arterial truncus, atrioventricular septal defects, aortic valve atresia/stenosis, transposition of great vessels, tetralogy of Fallot, total anomalous pulmonary venous return, and coarctation of aorta, without additional CHD subgroups classified as severity I.
Severity III (SIII): ventricular septal defect (VSD), atrial septal defect, and pulmonary valve stenosis, without additional CHD subgroups classified as SI or SII.

American Heart Association.

Helen Dolk et al. Circulation. 2011;123:841-849

Outcomes informations influence the decision to perform TOP in UVH

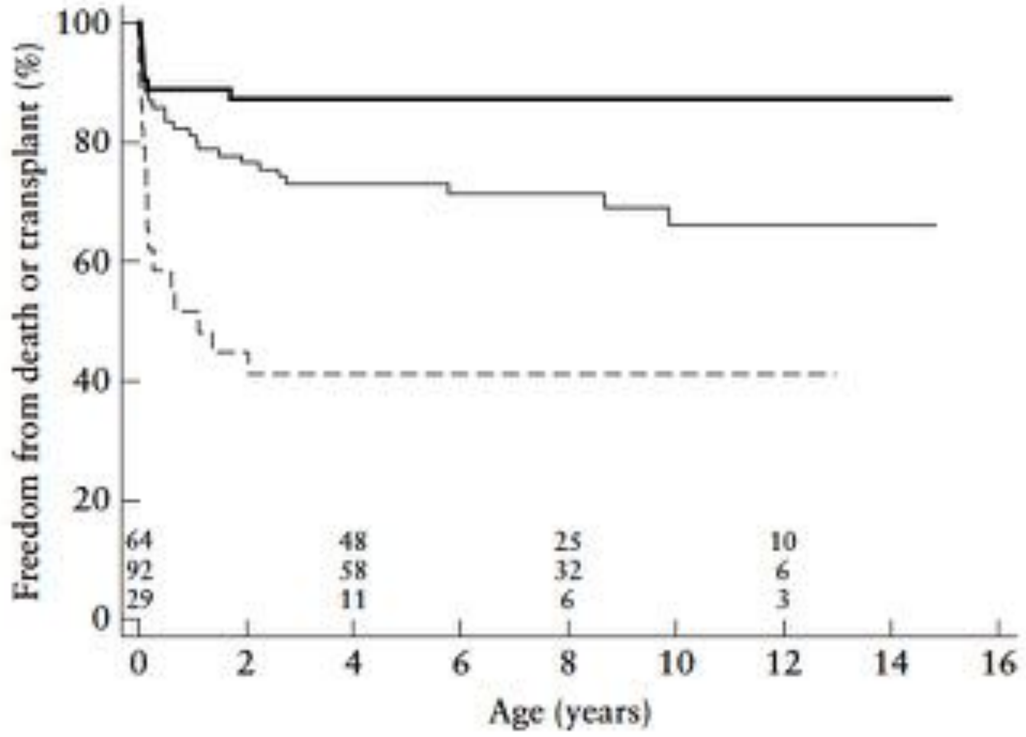


Figure 2 Kaplan–Meier survival plot showing difference in postnatal transplantation-free survival after Fontan procedure of all live births with prenatal diagnosis of dominant LV (——, $n = 65$) and dominant RV categorized into high-risk (– – –, $n = 30$) or standard-risk (——, $n = 95$) hypoplastic left heart syndrome. Log rank test $P < 0.001$. Values at bottom of plot indicate number of patients alive and not lost to follow-up at each time point, displayed in same order as curves.

Dominant LV

Dominant RV/low risk

Dominant RV/high risk

Beroukhim RS et al. Ultrasound Obstet Gynecol 2015; 45: 657–663

Cardiologists and parents factors in deciding to continue pregnancy

Factor in Deciding to Continue Pregnancy (lay terminology)	Number of Cardiologists n = 38 (%)	Number of Parents n = 41 (%)
Moral/religious beliefs	7 (18)	13 (32)
Quality of life of the child	34 (89)	21 (51)
Potential neurodevelopmental delay (learning disabilities and delay)	23 (61)	3 (7)
Probability of child surviving until birth	2 (5)	11 (27)
Probability of child surviving into early childhood	1 (3)	9 (22)
Probability of child surviving into adulthood	8 (21)	10 (24)
Impact on quality of life for the family/siblings	10 (26)	3 (7)
Potential need for repeated surgeries and hospitalizations	5 (13)	3 (7)
Need for Fontan palliation vs. two-ventricle repair (severity of heart disease)	10 (26)	9 (22)
Potential need for a heart transplant	3 (8)	2 (5)

(Participants were asked to rank the 3 most important factors; all 3 factors are included for each subject. Therefore percentage summation is greater than 100.)

Arya B et al. Cong Heart Dis 2014

Infant MORTALITY in newborns with congenital heart defects
The EPICARD Study Group

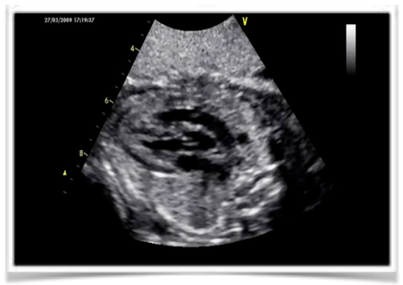
ACC-CHD categories	Prenatal diagnosis	Postnatal diagnosis			Infant mortality	
	N	<7days %	8-28 days %	29 days-1 year %	%	95%CI
Heterotaxy	8	25.0	0.0	12.5	37.5	8.5-75.5
Anomalies of venous	26	3.9	11.5	11.5	26.9	11.6-47.8
Anomalies of atria	174	0.6	0.6	2.3	3.5	1.3-7.3
Anomalies of AV junction and AV valves	109	8.3	7.3	12.8	28.4	20.2-37.0
Complex anomalies of AV	7	0.0	0.0	14.3	14.3	0.4-57.9
Functionally univentricular	48	41.7	12.5	4.1	58.3	43.2-72.4
Ventricular septal defects	1396	0.2	0.5	0.9	1.6	1.0-2.4
Anomalies of ventriculo-arterial connections	447	2.3	2.0	4.0	8.3	5.9-11.2
Anomalies of extra pericardial trunks	124	3.2	6.5	2.4	12.1	6.9-19.2
Congenital anomalies of coronary arteries	9	0	0	11.1	11.1	0.3-48.2
All	2348	2.1	1.8	2.5	6.4	5.5-7.5
All except chromosomal anomalies and /or anomalies of other systems and IVSD	**784**	**2.9**	**2.2**	**3.6**	**8.7**	**6.8-10.9**

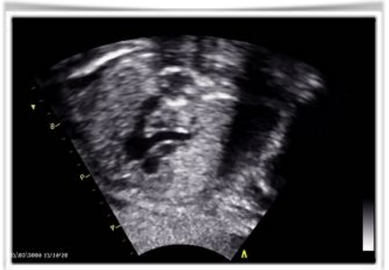
Prenatal diagnosis of CHD and MORTALITY

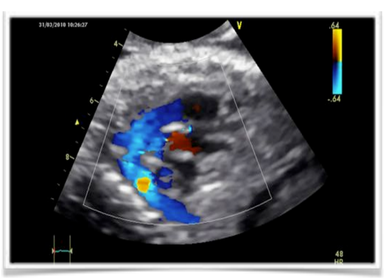
Is there any impact ?

Prenatal diagnosis of TGA reduces neonatal mortality

TGA







Preoperative mortality in TGA = 4-6%
(*vs./+)* Surgical mortality = 1-2%

Comparison of Characteristics of Patients in the Prenatal and Postnatal Groups

	Postnatal Group	Prenatal Group	*P*
Isolated TGA	304	57	NS
Associated defects	48	11	NS
VSD	31	5	NS
VSD + CoA	14	3	NS
CoA	1	1	NS
Age at admission, h	75 ± 210	2.2 ± 3.8	<0.01
Mechanical ventilation	95 (28)	13 (17.8)	<0.01
Metabolic acidosis + MOF	56	6	<0.05
PGE_1 infusion	95	32	NS
BAS	168	54	NS
Preoperative mortality	15	0	<0.05
Coronary artery pattern	233 (68)	49 (65)	
Normal	168	47	NS
Abnormal	65	26	NS
Preoperative mortality	30	0	<0.01
Hospital stay, d	30 ± 17	24 ± 11	<0.01

VSD indicates ventricular septal defect; CoA, coarctation; MOF, multiorgan failure; PGE_1, prostaglandin E_1; BAS, balloon atrioseptostomy; and ASO, arterial switch operation. Values are n (%).

Bonnet D et al. Circulation 1997
van Velzen CL et al. Ultrasound Obstet Gynecol 2015

Recent studies show that prenatal diagnosis DOES NOT impact neonatal CHD mortality

Table 3 Association between prenatal diagnosis and risk of infant mortality for four specific congenital heart defects (CHDs), EPIdémiologie des CARDiopathies congénitales (EPICARD) Population-Based Cohort Study

	Prenatal diagnosis		Infant mortality				
CHD		n*	n†	%	95% CI	Risk ratio	95% CI
Functionally univentricular heart‡	No	7	3	42.9	9.9 to 81.6		
	Yes	32	17	53.1	34.7 to 70.9	1.2	0.5 to 3.1
d-Transposition of the great arteries‡	No	24	1	4.2	0.1 to 21.1		
	Yes	57	5	8.8	2.9 to 19.3	2.1	0.3 to 17.1
Tetralogy of Fallot‡	No	18	2	11.1	1.4 to 34.7		
	Yes	36	1	2.8	0.07 to 14.5	0.3	0.02 to 2.6
Coarctation of the aorta‡	No	44	3	6.8	1.4 to 18.7		
	Yes	29	2	6.9	0.8 to 22.8	1.0	0.2 to 5.7

*N = number of live births (denominator data).
†n= number of deaths (numerator data).
‡Cases with the specific International Paediatric and Congenital Cardiac Code for the given CHD; whether or not other CHD codes were also included, all cases with chromosomal or others anomalies were excluded.

Khoshnood B et al. BMJ open 2017
van Velzen CL et al. BJOG 2016;123:400–407

Prenatal diagnosis has a limited (TGA) or no impact on neonatal mortality

Study or subgroup	Experimental Events	Total	Control Events	Total	Weight	Odds ratio M-H, Fixed, 95% CI
TGA						
Bonnet 1999	0	57	20	200	11.6%	0.08 [0.00, 1.31]
Kumar TGA 1999	0	14	4	20	3.8%	0.19 [0.01, 3.74]
Subtotal (95%CI)		71		232	15.4%	0.11 [0.01, 0.82]
Total events	0		24			
Heterogeneity: Chi^2=0.19, df=1 (P=0.67); I^2=0%						
Test for overall effect: Z=2.14, (P=0.03)						
HLHS						
Glatz 2007	9	26	3	12	3.5%	1.59 [0.34, 7.38]
Kumar HLHS 1999	13	27	21	47	10.3%	1.15 [0.45, 2.97]
Tworetzky 2001	0	14	13	38	9.4%	0.07 [0.00, 1.18]
Subtotal (95% CI)		67		97	23.1%	0.78 [0.38, 1.56]
Total events	22		37			
Heterogeneity: Chi^2=4.31, df=2 (P=0.12); I^2=54%						
Test for overall effect: Z=0.71 (P=0.48)						
LHO						
Espen 1998	6	15	8	30	3.9%	2.08 [0.57, 7.68]
Subtotal (95% CI)		15		30	3.9%	2.08 [0.57, 7.68]
Total events	6		8			
Heterogeneity: Not applicable						
Test for overall effect: Z=1.10 (P=0.27)						
DORV						
Lagopoulos 2010	2	63	3	48	4.3%	0.49 [0.08, 3.07]
Subtotal (95% CI)		63		48	4.3%	0.49 [0.08, 3.07]
Total events	2		3			
Heterogeneity: Not aplicable						
Test for overall effect: Z=0.76 (P=0.45)						
Mixed						
Copel 1997	9	45	18	54	16.9%	0.50 [0.20, 1.26]
Fuchs 2007	2	49	21	208	9.9%	0.38 [0.09, 1.67]
Verheijen 2001	14	81	62	327	26.4%	0.89 [0.47, 1.69]
Subtotal (95% CI)		175		589	53.3%	0.67 [0.41, 1.10]
Total events	25		101			
Heterogeneity: Chi^2=1.73, df=4 (P=0.42); I^2=0%						
Test for overall effect: Z=1.58 (P=0.11)						
Total (95% CI)		391		999	100.0%	0.66 [0.46, 0.94]
Total events	55		173			
Heterogeneity: Chi^2=12.78, df=9 (P=0.17); I^2=30%						
Test for overall effect: Z=2.29 (P=0.024)						

Fig. 6. Forest plot for the comparison of postoperative mortality between the prenatal and postnatal diagnosis groups. Significant difference in postoperative mortality was found between the prenatal and postnatal diagnosis groups, with an odds ratio of 0.66 (95% CI, 0.46, 0.94, P=0.02). No heterogeneity was detected (P=0.17, I^2=30%). HLHS: hypoplastic left heart syndrome; TGA: transposition of great arteries; LHO: left heart obstruction; DORV: double outlets right ventricle; CI: confidence interval; df: degree of freedom.

Li F et al. World J Pediatr. 2016;12:298-307.

Prenatal diagnosis still reduces mortality in countries with limited access to neonatal care

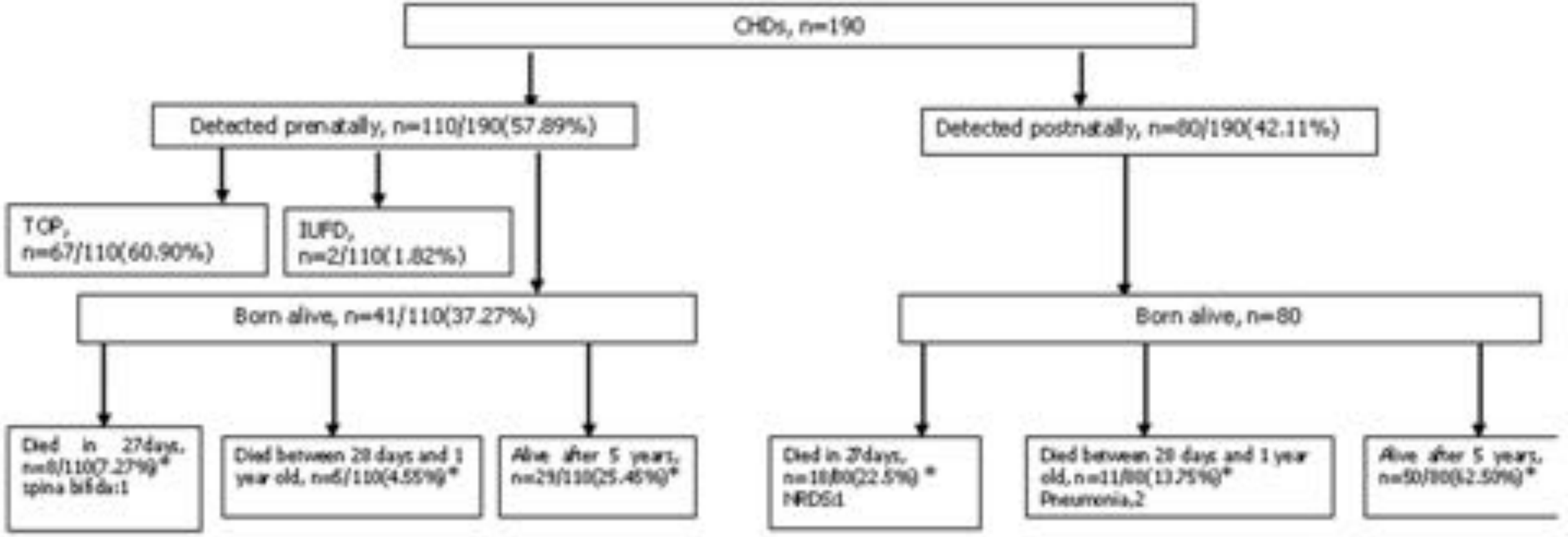
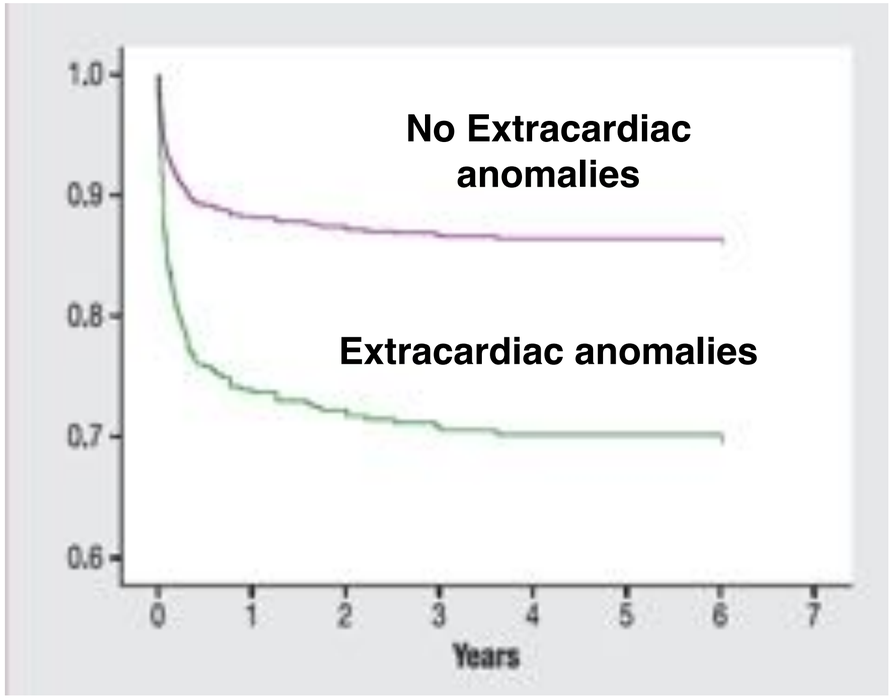


Fig 3. Outcomes and prognosis of infants with CHDs detected either prenatally or postnatally. *P<0.001.

Xie D, et al. Perinatal outcomes and congenital heart defect prognosis in 53313 non-selected perinatal infants. PLoS ONE 2017; 12(6): e0177229.

Death before hospital discharge in prenatally diagnosed « in-born » CHD

Type of CHD/predicted physiology	Mortality before discharge n (%)
at risk for Rashkind	8 (2.3)
ductal-dependent pulmonary flow	13 (12.1)
potentially ductal-dependent pulmonary flow	3 (2.1)
ductal-dependent systemic flow	**25 (39.6)**
potentially ductal-dependent systemic flow	16 (5.2)
TAPVR	1 (12.5)
AV block with CHD	0 (0)
a priori at no risk of early intervention	7 (6.5)
ALL	**73 (6.7)**



Bensemlali M et al. CiTY 2016
Bensemlali M et al. Arch Cardiovasc Dis 2016

Mortality is no more the important outcome in developed countries

MORBIDITY (early and late) should be the new end-point to scrutinize

Mortality is not an end-point

Does prenatal diagnosis of CHD reduce neonatal morbidity?

- Prenatal diagnosis reduces morbidity in life-threatening CHD ?
  - Not reproducible in TGA[1] nor in other defects
- Prenatal diagnosis allows to anticipate and prevent early demise ?
  - Through immediate interventions[2] after *in utero* transfer of fetuses at risk

1-Escobar-Diaz M et al. Ultrasound Obstet Gynecol. 2015 ; 45: 678–682
2-Bensemlali M et al. CiTY 2016

Prenatal diagnosis anticipates and prevents early demise *is in utero transfer a valid option ?*

Common indications for in utero transfer

- **Life threatening CHDs**
  - ***Ex: TGA, TAPVR, HLHS***
- **Evolutive defects**
  - ***Ex: Coarctation of the aorta***
- **Uncertain perinatal physiology**
  - ***Ex: Tetralogy of Fallot***
- **Highly variable/unpredictable postnatal outcome**
  - ***Ex: Ebstein***

Interventions in prenatally diagnosed « in-born » CHD

Type of CHD/predicted physiology	Number of patients	Intervention before discharge %
at risk for Rashkind	345	86
ductal-dependent pulmonary flow	94	74
potentially ductal-dependent pulmonary flow	163	23
ductal-dependent systemic flow	66	89
potentially ductal-dependent systemic flow	300	39
a priori at no risk of early intervention	98	7
ALL	1080	55

TGA

748 in born

21% early demise

87% intervention

Suspected coarctation

486 in born

35% intervention

ToF

287 in born

4% intervention

Bensemlali M et al. CiTY 2016
Bensemlali M et al. Arch Cardiovasc Dis 2016

Mortality is not an end-point

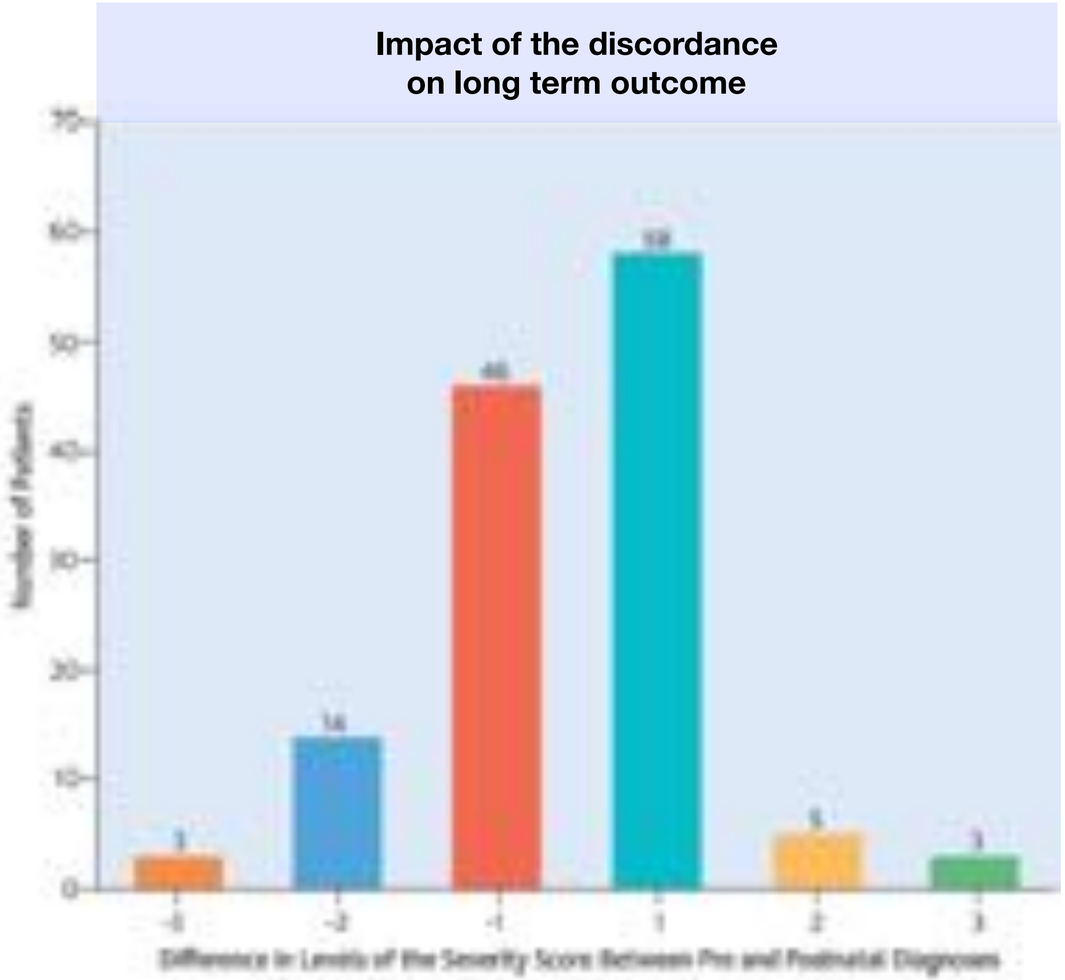
Does prenatal diagnosis of CHD reduce neonatal morbidity?

- Prenatal diagnosis reduces morbidity in life-threatening CHD ?
  - Not shown in TGA[1] nor in other defects
- Prenatal diagnosis allows to anticipate and prevent early demise ?
  - Through immediate interventions[2] after *in utero* transfer of fetuses at risk
  - Through *in utero* transfer to optimize diagnosis accuracy and tailor postnatal management

1-Escobar-Diaz M et al. Ultrasound Obstet Gynecol. 2015 ; 45: 678–682
2-Bensemlali M et al. CiTY 2016
3-Bensemlali M et al. JACC 2016

Impact of discordances between pre- and post-natal diagnosis on outcomes in in-born fetal CHD

Total number 1285	n	% of the total population	% of the discordances
Different diagnosis	36	2.9	9.8
Partially different diagnosis with MAJOR impact on the planned neonatal treatment	97	7.7	26.3
Partially different diagnosis with MINOR impact on the planned neonatal treatment	235	18.7	63.9



Bensemlali M et al. JACC 2016

Mortality is no more the important outcome in developed countries

Early morbidity reduction after prenatal diagnosis of CHD
is a valid end-point to be further confirmed
Indications for in utero transfer and prediction of early outcomes
should be further defined

Can late outcomes be predicted in fetal CHD ?

Prenatal diagnosis accuracy in predicting cardiac outcomes

- Prenatal diagnosis predicts cardiac mortality in live-birth prenatally diagnosed CHDs ?

  - Not shown in TGA[1] nor in other defects

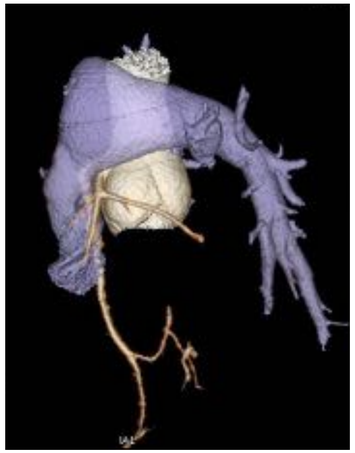
Is information on cardiac outcomes individualized ?

Mortality in simple TGA is mainly driven by coronary artery pattern
Should we attempt to diagnose coronary artery distribution before birth ?

1,7%

Single ostium

OR = 2.9



3,2%

Intramural course

OR = 6.5

If you claim that surgical mortality is 1% in simple TGA,

-you are close to reality in TGA with normal coronary pattern

-you are excessively optimistic in TGA with intramural course of coronary artery

Pasquali SK et al. Circulation. 2002;106:2575-80.

Prenatal diagnosis accuracy in predicting cardiac outcomes

- Prenatal diagnosis predicts cardiac mortality ?
  - Not shown in TGA nor in other defects
- Prenatal diagnosis predicts reparability of complex defects ?
  - Partially shown in PA-VSD and DORV

Is information on probability of complete repair individualized ?

Repair of PA-VSD is closely related to size of pulmonary artery branches

	Repair < 1y %	p
PA branches Normal vs. absent/hypoplastic	86 vs. 55	<0.001
PA trunk present vs. absent	79 vs. 16	0,003
MAPCAs present vs. absent	76 vs. 50	0.17

Kaguelidou F et al. Eur Heart J. 2008

Prenatal diagnosis accuracy in predicting cardiac outcomes

- Prenatal diagnosis predicts cardiac mortality ?
  - Not shown in TGA nor in other defects
- Prenatal diagnosis predicts reparability of complex defects ?
  - Partially shown in PA-VSD and DORV
- Interventions after prenatal diagnosis can modify cardiac outcomes
  - Partially shown in fetal aortic valve stenosis

Prenatal diagnosis accuracy in predicting NON-cardiac outcomes

- Prenatal diagnosis improves neurodevelopment outcomes ?
  - Shown in TGA[1]

Calderon J et al. J Pediatr 2012

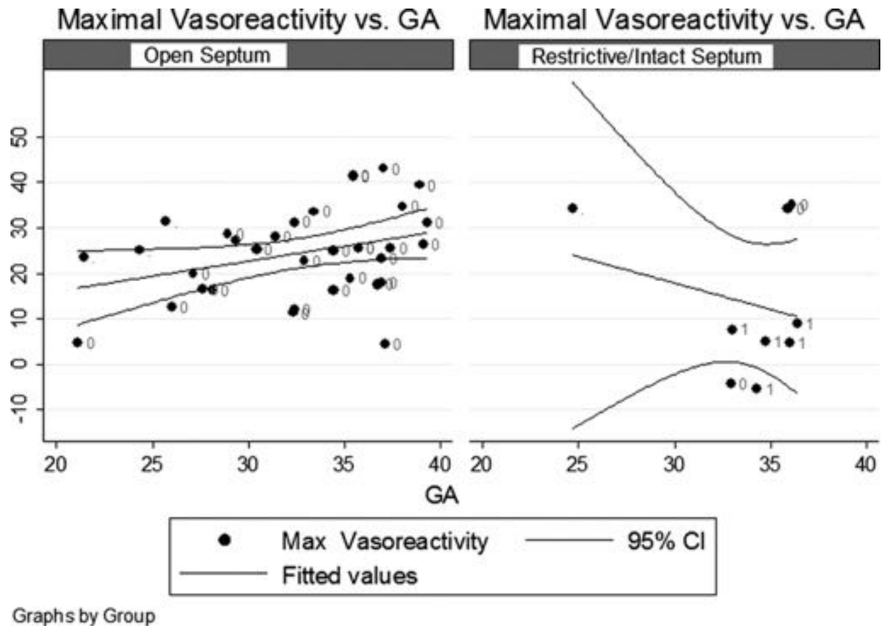
Neurodevelopmental morbidity is improved after prenatal diagnosis

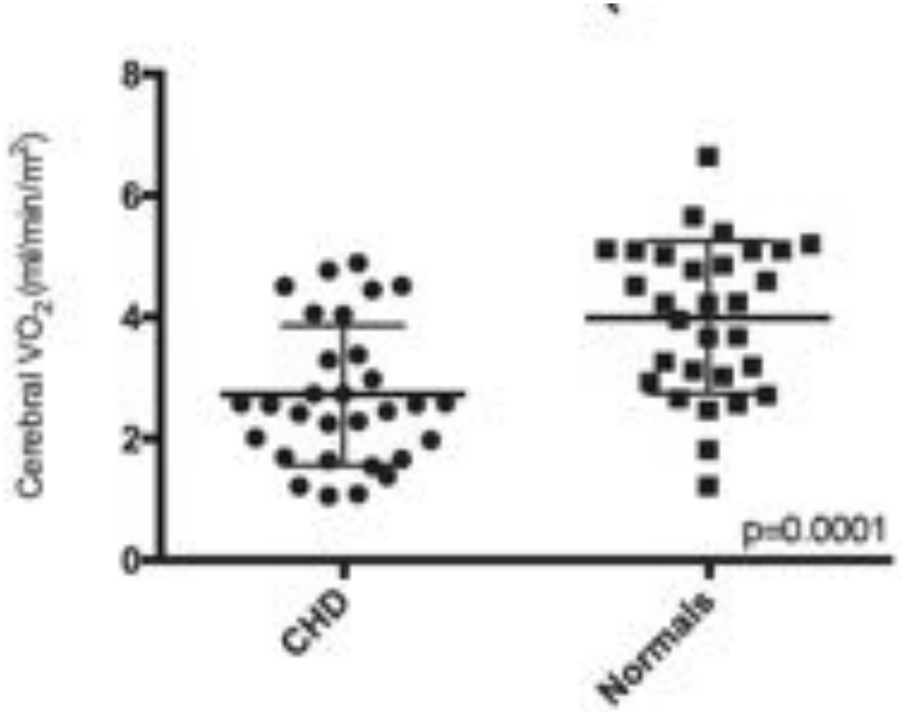
Cognitive Domain	Test	Prenatal (n=29)	Postnatal (n=16)	*p*
IQ	CMMS	114.5 (8.50)	112.4 (8.06)	0.4
Receptive Language	NEPSY - Comprehension	12.65 (0.55)	12.25 (1.12)	0.11
Response motor control	NEPSY – Knock and tap	24.31 (2.46)	24.14 (5.82)	0.89
Cognitive control	Stroop test (Number of errors)	2.41 (2.48)	4.31 (3.59)	0.04
	Stroop test (Reaction Time)	77.82 (28.05)	90.74 (36.71)	0.19
Verbal working memory	Digit span WISC IV	2.96 (2.48)	2.62 (2.57)	0.66
Spatial working memory	BEM-144 blocks	3.62 (2.0)	2.06 (2.01)	0.01
Cognitive flexibility	DCST	8.10 (2.65)	5.64 (2.61)	0.006
Social cognition	Theory of mind	1.31 (1.33)	0.31 (0.87)	0.01

Calderon J et al. J Pediatr 2012

Prenatal interventions can improve non-cardiac outcomes

Maternal interventions to improve outcome : hyperoxygenation





Pulsatility index and need for BAS in HLHS
Might improve LV filling in conditions leading to small LV
Hyperoxygenation may improve fetal brain VO2 and have an impact on white matter anomalies

Szwast A et al. Circ Cardiovasc Imaging 2010;3:172-178
Channing A et al. Ultrasound Obstet Gynecol 2015; 45: 664–669

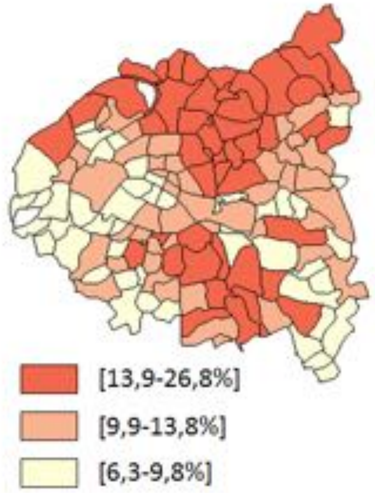
Prenatal diagnosis accuracy in predicting NON-cardiac outcomes

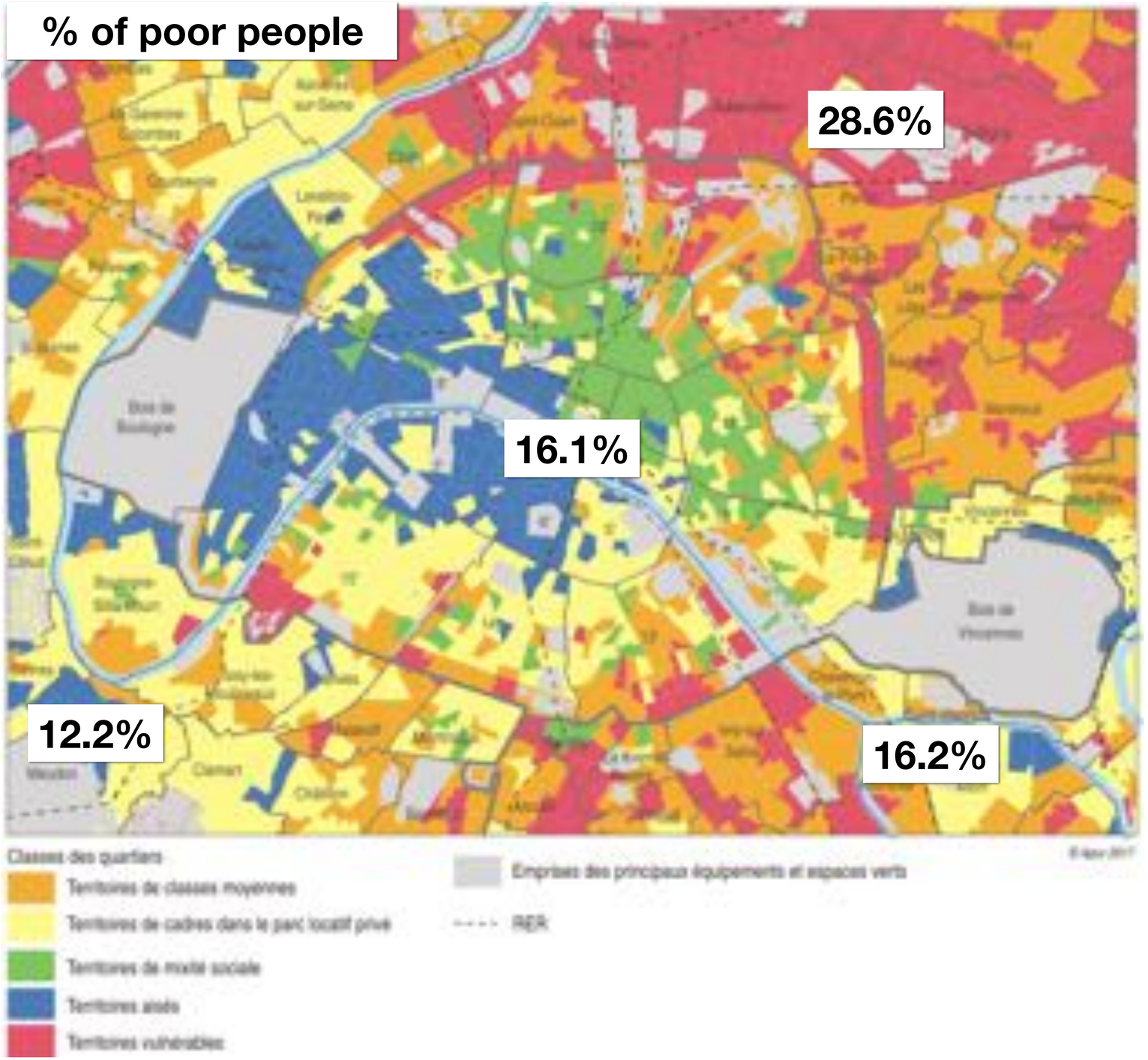
- Prenatal diagnosis improves neurodevelopmental outcomes ?
  - Shown in TGA[1]
- Prenatal diagnosis can help detecting social conditions at risk ?
  - Poverty is a major risk factor for poorer non-cardiac (and cardiac) outcomes

Calderon J et al. J Pediatr 2012
Khosnood B et al. 2017

Vulnerable areas

Schools, security, housing…





Socioeconomic disparities in healthcare are not inevitable
The EPICARD Study Group

Differences in prenatal diagnosis by maternal occupation, geographic origin and place of residence

The proportion of PND of CHD were similar across categories

- The health system organization allowed high availability of reimbursed specialized services that can provide similar access to PND for all socioeconomic groups

Income level and geographic origin does not influence access to prenatal diagnosis and postnatal care

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	All CHD				
	n 2867	% 29.1	Adj-OR	95%CI	p
Geographic origin					0.62
France	1370	27.7	Ref	Ref	
North African	526	28.1	1.0	0.8-1.3	
African	393	33.6	1.3	1.0-1.7	
Other	562	30.6	1.1	0.9-1.4	
Occupation					0.39
Professional	2139	68.9	Ref	Ref	
None	728	31.6	1.3	0.8-1.5	
Department of residence					0.31
Paris	972	28.8	Ref	Ref	
Hauts de Seine	702	29.5	1.1	0.8-1.4	
Val de Marne	509	26.3	0.7	0.5-1.0	
Seine-Saint Denis	684	25.6	0.9	0.7-1.2	

Khosnood B et al. 2017

Socioeconomic disparities in healthcare are not inevitable
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The association between TOP and maternal characteristics in fetuses with prenatal diagnosis of CHD are related to maternal geographic origin

	All CHD				
	n 835	% 41.4	Adj-OR	95%CI	p
Geographic origin					**<0.0004**
France	380	46.1	Ref	Ref	
North African	148	26.4	0.4	0.2-0.6	
African	132	34.9	0.6	0.3-1.0	
Other	172	48.8	1.1	0.7-1.8	
Occupation					**0.09**
Professional	165	43.0	Ref	Ref	
None	230	30.4	0.8	0.4-1.3	
Department of residence					**0.25**
Paris	280	42.1	Ref	Ref	
Seine-Saint Denis	214	38.8	0.9	0.6-1.5	

Proportion of TOP is related to maternal origin

Khosnood B et al. 2017

How socioeconomic differences in prenatal decision for TOP may influence outcomes ?

- The probability of TOPFA may represent women's preferences that should of course be respected.
- These differences in TOPFA can result in disparities in the spectrum of severity of CHD at birth and thereby, all else equal, in the risk of mortality, morbidity and long-term adverse developmental outcomes for newborns with CHD.
- In addition, families with fewer resources may become disproportionately responsible for the care of newborns with more severe types of CHD.
- The extent to which post-natal management can modify any such disparities needs to be examined.

State of play

- Prenatal diagnosis has a specific organization in France with a high rate of detection for complex CHD.
- While access to prenatal diagnosis is possible for all women, the proportion of TOP is stable for 25 years with extracardiac anomalies and univentricular heart being the major causes for TOP.
- Diagnosis accuracy has still to be improved to better organize perinatal care and predict short and long term outcomes.
- The future is to improve cardiac and extra cardiac outcomes of prenatally diagnosed fetuses with CHD:
  - Identification of fetuses at high risk before and after birth
  - Fetal cardiac interventions
  - Maternal/social interventions for cardiac or neurodevelopment end-points

PARSIFAL TEAM-M3C-NECKER


Bertrand Stos


Daniela Laux

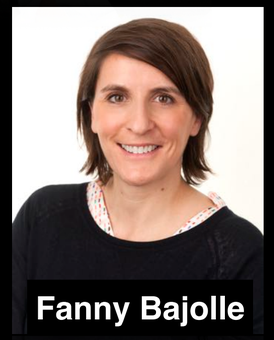

Marilyne Lévy


Eric Héry


Jérôme Le Bidois


Myriam Bensemlali


Yves Ville


Fanny Bajolle


Elsa Kermorvant


Laurent Salomon


Julien Stirnemann


Pascal Vouhé


Olivier Raisky


Régis Gaudin


Damien Bonnet


Alexandre Lapillonne


Philippe Roth

To read more www.carpedemm3c.com

40ème Séminaire
de Cardiologie Congénitale et Pédiatrique

Auditorium de l'Institut IMAGINE
24, Boulevard du Montparnasse
75015 Paris

21 et 22 Mars 2019
Imaginez demain

22 and 23 Novembre 2018

M3C-Necker

Cardiologie Congénitale et Pédiatrique

Bâtiment Laennec, 4ème étage

149, rue de Sèvres

75015 Paris

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Transposition of the great arteries

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