CONGENITAL HEART DISEASE

Delayed diagnosis of congenital heart disease worsens preoperative condition and outcome of surgery in neonates

K L Brown, D A Ridout, A Hoskote, L Verhulst, M Ricci, C Bull

Heart 2006;**92**:1298–1302. doi: 10.1136/hrt.2005.078097

See end of article for authors' affiliations

Correspondence to: K L Brown, Cardiac Unit, Great Ormond Street Hospital for Children NHS Trust, London WC1N 3JH, UK; BrownK@gosh.nhs.uk

Accepted 5 January 2006
Published Online First 31 January 2006

Objectives: To assess whether the route by which neonatal congenital heart disease (CHD) is first recognised influences outcome after surgery.

Methods: Surgical neonates admitted to a tertiary cardiac unit between March 1999 and February 2002 were retrospectively reviewed with analysis of risk factors for outcome. Three routes to initial recognition of CHD were compared: antenatal diagnosis, detection on the postnatal ward, and presentation after discharge to home. Outcome measures were mortality and duration of perioperative ventilation.

Results: 286 neonates had cardiac surgery with a median duration of ventilation of 101 h and in-hospital mortality of 12%. Recognition of CHD was antenatal in 20%, on the postnatal ward in 55% and after discharge to home in 25%. Multiple regression analyses, including the cardiac diagnosis, associated problems and other risk factors, indicated that severe cardiovascular compromise on admission to the cardiac unit was significantly related to mortality and prolonged ventilation. Considered in isolation, the route to recognition of heart disease did not influence mortality or ventilation time. Route to initial recognition did, however, influence the patient's condition on admission to the cardiac unit. Cardiovascular compromise and end organ dysfunction were least likely when recognition was antenatal and most common when presentation followed discharge to home.

Conclusion: The setting in which neonatal CHD is first recognised has an impact on preoperative condition, which in turn influences postoperative progress and survival after surgery. Optimal screening procedures and access to specialist care will improve outcome for neonates undergoing cardiac surgery.

In the current era many paediatric cardiac centres have directed resources towards facilities for the antenatal diagnosis of congenital heart disease (CHD). Antenatal diagnosis has been shown to benefit outcome in certain studies but this topic is controversial.[1–4] CHD that remains undetected by prenatal evaluation will come to light in postnatal life either during screening for CHD or when symptoms develop.[5] Outcome benefit as a result of screening for CHD is inferred from studies that show that children with undiagnosed CHD die[2 6–8] but is otherwise unproved.

In the context of reduced early mortality from CHD focus has increased on morbidity-related and long-term outcome measures. Certain adverse early variables—increased length of stay in hospital,[9] preoperative asphyxia and perioperative seizures—have been recognised to have long-term repercussions for neurodevelopment.[10–13] Assessing factors that can potentially influence preoperative condition and operative morbidity is therefore appropriate to better understand and improve the long-term outcome.

We aimed at assessing whether the route to initial diagnosis of CHD influenced outcome, in terms of morbidity and mortality, for neonates who required early surgery at our centre. We aimed at including preoperative variables that have a potential ''knock on'' effect. We hypothesised that the earliest possible diagnosis of CHD (prenatal) would be the most optimal and that the latest diagnosis (CHD missed by all screening opportunities) would be the worst-case scenario for the patient.

METHODS

Ethical committee approval was obtained for a retrospective review of medical records of neonates admitted to cardiac intensive care unit (ICU) between March 1999 and February 2002. Included patients had a primary diagnosis of CHD and underwent surgery in the first month of life. Demographic information was collected for each patient: weight, age on admission to the cardiac ICU, presence of a chromosomal or other associated abnormality and the main type of CHD. Included CHD types were two functional ventricles and left heart obstruction (LHO), transposition of the great arteries (TGA), pulmonary atresia (PA) or total anomalous pulmonary venous connection (TAPVC), a single ventricle with arch obstruction (SV). and other complex lesions including truncus arteriosus and isomerisms. The paediatric index of mortality score at admission to the ICU was included for descriptive purposes.

Three routes to the patients' initial diagnosis of CHD were considered: (A) antenatal diagnosis; (B) diagnosis before discharge from the maternity unit; and (C) diagnosis after discharge to home.

Patients with an antenatal diagnosis were delivered at an allied hospital where staff are experienced in treating neonates with CHD. These babies were stabilised and transferred to our centre. The patients' clinical condition on admission to the cardiac unit was evaluated with respect to two adverse clinical syndromes: (1) significant cardiovascular compromise, defined as a requirement for mechanical ventilation plus resuscitation with 20 ml/kg colloid with or without dopamine or epinephrine infusion; and (2) end organ dysfunction, defined as acidosis of pH < 7.1 and lactate > 3 mmol/l with one or more of renal failure,

Abbreviations: CHD, congenital heart disease; ICU, intensive care unit; LHO, left heart obstruction; PA, pulmonary atresia; SV, single ventricle with arch obstruction; TAPVC, total anomalous pulmonary venous connection; TGA, transposition of the great arteries

necrotising enterocolitis, cerebrovascular accident or hepatic dysfunction.[14]

The first outcome measure was total duration of perioperative mechanical ventilation. We operate a uniform policy whereby ventilation is initiated only on the basis of clinical need. For example, a neonate who is stabilised by prostaglandin E treatment is not necessarily ventilated but is always transferred with specialist staff in attendance. The second outcome measure was death before discharge to home independent of age. If a neonate was transferred to another hospital, including to a chronic care facility, and ultimately died, this was counted as a death.

Statistical methods

Regression analysis of risk factors was performed for outcome measure 1 (ventilation time) and logistic regression was used for outcome 2 (death). A value of $p = 0.05$ was deemed significant. To account for the effect of CHD type on outcome, each type was compared with the lowest mortality diagnosis, PA. For outcome measure 1, as ventilation time data were skewed, a log transformation was used to comply with regression assumptions. Factors found significant on univariate analysis were evaluated further with multiple regression analyses. For outcome measure 1, a multiple regression model included route to CHD recognition, CHD type, significant cardiovascular compromise, number of end organ dysfunctions and number of associated medical problems. For outcome measure 2, a multiple logistic regression model included route to CHD recognition, CHD type, 1/patient age, significant cardiovascular compromise and number of end organ dysfunctions. For the regression models, a backward elimination selection procedure was used and the normality assumption was checked by the Shapiro–Francia W dash test.

A χ^2 test for trend was used to assess whether progressive delay in initial recognition of CHD (from route A to B to C) had an effect on patient outcome measures 1 and 2. The test for trend was also applied to evaluate whether delayed recognition of CHD influenced the patient's condition on admission to the cardiac unit. For these analyses, patient condition was described in terms of cardiovascular compromise and end organ dysfunction. These analyses were performed for the cohort as a whole and for the individual CHD types separately. The software package Stata was used throughout (2003; StataCorp LP, College Station, Texas, USA).

RESULTS

Demographics

The study examined 286 neonates, of whom 33 (12%) died before discharge to home. The median age at death was 14 days (range 2 days to 67 weeks). Six patients died more than 30 days after surgery (four while at another hospital) and 27 (9.4%) died at our institution within 30 days. The median intubation time was 101 h (range 1 h to 67 weeks) and the mean paediatric index of mortality score was 0.126. Initial recognition of CHD was antenatal (route A) in 56 (20%), before discharge from the maternity ward (route B) in 157 (55%) and after discharge to home (route C) in 73 (25%). The main CHD type requiring surgery was LHO in 89 (31%) patients, TGA in 77 (27%), PA in 44 (15%), SV in 22 (8%), TAPVC in 18 (6%) and another type in 36 (13%).

Univariate analysis of risk factors

For outcome 1 (ventilation time), regression coefficients have been anti-logged to original units (hours) and are shown unadjusted for other risk factors. For binary data such as cardiovascular compromise, the regression coefficient corresponds to the ratio of the geometric means of two groups. For continuous factors, such as weight, the regression coefficient estimates the average amount by which intubation time increased for each unit increase in the particular risk factor. For outcome measure 2 (death), odds ratios are presented unadjusted for any other risk factor.

Prolonged perioperative ventilation was more likely when cardiovascular compromise or end organ dysfunction was present on admission, if there was an associated syndrome or medical problem, if the diagnosis was TGA, SV or TAPVC and if the route to recognition was from the maternity unit rather than through antenatal screening.

Death was more likely when cardiovascular compromise or end organ dysfunction was present on admission, the patient presented to the cardiac unit at a younger age, the diagnosis was SV or TAPVC, and the route to recognition was antenatal rather than after discharge to home ($p < 0.05$ for all) (table 1).

Multiple analyses of risk factors

Multiple analyses indicated that cardiovascular compromise or end organ dysfunction on admission, the presence of associated medical problems and a diagnosis of either TGA or TAPVC were independently associated with prolonged perioperative ventilation. Only cardiovascular compromise

Table 1 Univariate analysis

		Outcome 1 (ventilation time)		Outcome 2 (death)	
Risk factor	Quantity*	Regression coefficient (95% CI)	p Value	Odds ratio (95% CI)	p Value
CHD recognition route					
B versus A		1.51 (1.05 to 2.16)	0.03	0.50 (0.23 to 1.12)	0.09
C versus A		1.10 (0.73 to 1.67)	0.64	0.33 (0.11 to 0.94)	0.04
B versus C		1.37 (0.98 to 1.90)	0.06	1.54 (0.59 to 4.03)	0.38
Cardiovascular compromise	109 (38%)	1.79 (1.35 to 2.36)	<0.01	5.5 (2.54 to 11.90)	<0.01
⩾1 end organ dysfunctions	41 (14%)	1.39 (1.22 to 1.59)	<0.01	1.53 (1.16 to 2.01)	<0.01
Weight (kg)	3.2 (1–5.1)	0.97 (0.79 to 1.18)	0.75	0.77 (0.47 to 1.25)	0.28
Admit age (days)	8.5 (0–31)	1.03 (0.94 to 1.11)	0.55	1.37 (1.13 to 1.67)	<0.01
⩾1 syndromes or associated problems	71 (25%)	1.23 (1.02 to 1.49)	0.03	1.21 (0.79 to 1.84)	0.38
CHD type					
LHO versus PA	89 (31%)	1.12 (0.73 to 1.70)	0.61	2.07 (0.42 to 10.21)	0.37
TGA versus PA	77 (27%)	1.92 (1.25 to 2.95)	<0.01	1.46 (0.27 to 7.85)	0.66
SV versus PA	22 (8%)	1.79 (0.99 to 3.25)	0.05	30.33 (5.80 to 158.52)	<0.01
TAPVC versus PA	18 (6%)	3.02 (1.59 to 5.71)	<0.01	6.0 (0.99 to 36.37)	0.05
Other versus PA	36 (13%)	1.46 (0.87 to 2.44)	0.15	3.39 (0.62 to 18.62)	0.16

*Data are number (%) or median (range).
CHD, congenital heart disease; LHO, left heart obstruction; PA, pulmonary atresia; route A, antenatal diagnosis; route B, diagnosis before discharge from the maternity unit; route C, diagnosis after discharge to home; SV, single ventricle with arch obstruction; TAPVC, total anomalous pulmonary venous connection; TGA, transposition of the great arteries.

Table 2 Multiple analyses

Risk factor	Outcome 1 (ventilation time) Regression coefficient (95% CI)	p Value	Outcome 2 (death) Odds ratio (95% CI)	p Value
Cardiovascular compromise	1.46 (1.08 to 1.99)	0.02	5.68(2.04 to 15.84)	<0.01
End organ dysfunction	1.36 (1.18 to 1.57)	<0.01	1.22 (0.85 to 1.76)	0.28
Syndrome or associated problem	1.34 (1.12 to 1.61)	<0.01	N/A	N/A
CHD type				
LHO versus PA	0.79 (0.53 to 1.19)	0.27	1.06 (0.18 to 6.25)	0.95
TGA versus PA	1.98 (1.32 to 2.97)	<0.01	1.82 (0.30 to 11.08)	0.51
SV versus PA	1.54 (0.88 to 2.69)	0.13	27.03 (4.59 to 159.16)	<0.01
TAPVC versus PA	2.31 (1.26 to 4.22)	<0.01	2.79 (0.42 to 18.55)	0.29
Other versus PA	1.18 (0.73 to 1.90)	0.50	2.67 (0.44 to 16.28)	0.29
1/patient admit age	NA	NA	1.13 (0.88 to 1.44)	0.33

CHD, congenital heart disease; LHO, left heart obstruction; NA, not applicable; PA, pulmonary atresia; SV, single ventricle with arch obstruction; TAPVC, total anomalous pulmonary venous connection; TGA, transposition of the great arteries.

on admission and a diagnosis of SV significantly increased the risk of death when other factors were considered ($p < 0.05$). Multiple analyses showed no relationship between route to recognition of CHD and the outcome measures (table 2).

Relationship between route to initial recognition of CHD, patient condition on admission and outcome

Ventilation time

No trend for the three routes to CHD recognition was observed for outcome measure 1 (table 3). The multiple analyses presented in table 2 indicated that ventilation time was influenced by several risk factors (cardiovascular compromise, end organ dysfunction, associated medical problems and diagnosis of either TGA or TAPVC). These risk factors for prolonged ventilation were unevenly distributed within groups A, B and C. Associated medical problems, which independently predisposed to longer ventilation, were least common in group C. This is in contrast to cardiovascular compromise and end organ dysfunction (also independent risk factors for longer ventilation), which were most common in group C.

Death

There was a trend towards increased mortality in the antenatal diagnosis group (route A) with the lowest mortality among the patients who presented after discharge to home (route C) (table 3). Most (68%) of the patients with a single ventricle and aortic arch obstruction had an antenatal diagnosis and this diagnosis was an independent mortality risk (see table 2). Antenatal diagnosis was made relatively infrequently for other CHD types (LHO 19%, PA 27%, TGA 6%, TAPVC 0%) that had lower mortality risk.

Table 3 Trend in outcome and patient condition by route of presentation by which congenital heart disease was recognised

Outcome measure	Route A	B	C	p Value*
Ventilation time (median hours)	71	117	90	0.64
Death	21%	12%	8%	<0.05
Cardiovascular compromise	23%	33%	60%	<0.01
End organ dysfunction	5%	11%	29%	<0.01

*χ^2 test for trend.

Patient condition

When neonates with all diagnoses were considered together, there was a significant trend towards increased rates of cardiovascular compromise and end organ dysfunction the later CHD was recognised. Furthermore, for individual CHD types, there was a significant trend towards a higher rate of cardiovascular compromise with later CHD recognition for LHO, TGA and SV lesions considered separately. There was also a significant trend towards a higher rate of organ dysfunction with later CHD recognition for LHO and SV lesions considered separately ($p < 0.05$ for all).

DISCUSSION

For neonates in this study, there was a relationship between poor preoperative condition and worse outcome after heart surgery. Poor presenting patient condition was defined as cardiovascular compromise requiring mechanical ventilation and resuscitation or evidence of metabolic acidosis with end organ dysfunction. Neonates with CHD who presented preoperatively with these clinical features were at a disadvantage in terms of duration of ventilation associated with heart surgery and mortality in the case of cardiovascular compromise. Adverse preoperative condition or critical illness was most common when CHD was recognised late, after the neonate had been discharged home, and was least common when recognition was antenatal. The route to recognition of CHD was not directly related to the outcome measures, most likely because a range of interrelated and unevenly distributed factors contributed.

Certain cardiovascular risk factors may render a neonate particularly vulnerable to preoperative deterioration[15 16]—for example, obstructed pulmonary venous return in the context of SV.[16] Thus, there are neonates with CHD in critical condition for whom the most optimal preparation and access to tertiary care will not avert severe cardiovascular compromise. These children are likely to present very early in life with critical illness. Our study suggests, however, that, as well as predetermined cardiovascular risk factors, the route by which CHD is first recognised also plays a major part in determining preoperative condition, as this determines access to and timing of appropriate treatment.

A major limitation of this study is the small number of patients included; an investigation of a larger cohort would be informative. Furthermore, the results must have been influenced by local factors and practice patterns. For example, the rates of antenatal diagnosis for our dataset were as follows: for all patients, 20%; in SV, 68%; in LHO, 19%; in PA 27%; in TGA, 6%; and in TAPVC, 0%. A recently presented program-based rate of antenatal diagnosis for a

neonatal cohort was noticeably higher at 54% (A Dorfman and G Wernovsky, personal communication). Previously reported rates for SV range from 36.6%[17] to 86%[18] and for TGA a rate of 26% has been reported.[2] We are a tertiary centre with no obstetric service on site and the median age on admission to the unit was 8.5 days. Other series report a median admission age for neonates with CHD of between 0 and 4 days,[2 17 18] suggesting a late presenting age in our patients. This is in keeping with what we believe may be a high proportion of missed patients with CHD, which was 25%. Furthermore, 77 (27%) neonates in the cohort received ICU care at another institution before transfer to our centre for cardiac surgery. This aspect may have influenced the proportion of patients who required intubation and subsequent escalation of intensive care support mainly for the purpose of transfer.

Our first outcome measure, ventilation time, is a summary measure of morbidity that is less vulnerable to extraneous factors than length of hospital stay. We note with interest a recent study that associated prolonged stay after neonatal arterial switch operation with adverse neurological outcome in childhood.[9] The median duration of ventilation for our cohort was 101 h, or 4.2 days. Data directly comparable with ours are not readily available in the literature, although a program-based cohort recently reported a median length of hospital stay of 13 days (A Dorfman and G Wernovsky, personal communication). In general, the total duration of ventilation is likely to be greater for patients who require preoperative intubation, and a high proportion of neonates requiring this will influence the median value. We note that cardiovascular compromise for which mechanical ventilation was required was present in 109 (38%) of our patients and this proportion rose to 60% in the missed neonates of group C. A published series of patients with TGA reported preoperative mechanical ventilation in 38% in patients with postnatal diagnosis,[2] although we are unaware of other surveys of neonates with missed CHD.

The mortality among our patients was 9.4% at 30 days and 12% to hospital discharge. This is broadly comparable with a recently published UK summary of overall neonatal mortality for 2000–1 that reported that 9.1% of 780 patients died before 30 days and 13.9% died by 1 year of age.[19] Neonatal operative mortality is influenced by case mix, prematurity, associated syndromes or anomalies, and low Apgar scores.[20 21] Although we found a strong relationship between adverse preoperative patient condition and mortality (and other authors have presented related information[1 2]), risk adjustment based on critical illness scores is not usual.[21] Our data are supported by reports that use severity of illness scores such as paediatric index of mortality, as these are known to be useful in populations of critically ill children that include those with CHD.[22]

Several examples in the literature describe the evolution of dangerous or fatal CHD symptoms when access to appropriate specialist care is impaired. A 1998 study from Toronto indicated that 4% of neonates with simple TGA died preoperatively from hypoxaemia due to a restrictive atrial septum.[15] A 1994 study from the UK found that, of 185 patients who died of CHD in one region, 30% had died without a diagnosis.[6] A further 1999 study from France of 250 patients with TGA detected eight previously unrecognised cases at autopsy.[2] A 1999 regional study from the USA identified, among 800 infants who died of CHD, 76 who died before a diagnosis was made.[7] A UK study that evaluated patterns of postnatal CHD presentation concluded that, of those discharged without a diagnosis, 35% became unwell or died before the age of 6 weeks.[5] Our study evaluated the effect of variations in access to appropriate care in a population that was actually treated with cardiac surgery. In addition to the 286 included patients, we know of five further neonates with CHD who were referred to our centre during the study period and died without access to surgery. All died from severe cardiovascular compromise and end organ injury that were related to late diagnosis. All these studies are salient reminders that neonatal CHD remains a significant source of morbidity and mortality.

In conclusion, for the individual patient, optimised preoperative condition will be associated with minimised operative morbidity and mortality. Early identification of CHD will facilitate access to appropriate care and is in the patient's best interests. The ideal scenario for CHD screening and triage is beyond the scope of this paper, but dialogue and feedback between cardiac units and the communities responsible for obstetric and neonatal screening may be required to promote best practice.

ACKNOWLEDGEMENTS

We thank Dr AP Goldman and Mrs G Oldham, who are responsible for the data resources available to this study, for their assistance. We also thank Professor Gil Wernovsky, Dr Aaron Dorfman and the staff of the cardiac intensive care unit at the Children's Hospital of Philadelphia for sharing their data and experience with us. Research performed at NHS Trusts benefits from research and development funding received from the NHS Executive.

Authors' affiliations

K L Brown, A Hoskote, L Verhulst, M Ricci, C Bull, Cardiac Unit, Great Ormond Street Hospital for Sick Children, London, UK
D A Ridout, The Centre for Paediatric Epidemiology and Biostatistics, Institute of Child Health, London, UK

Competing interests: None declared

The project was registered with and approved by the Research and Development Office at the Institute of Child Health, London

REFERENCES

1 **Franklin O**, Burch M, Manning N, *et al*. Prenatal diagnosis of coarctation of the aorta improves survival and reduces morbidity. *Heart* 2002;**87**:67–9.
2 **Bonnet D**, Coltri A, Butera G, *et al*. Detection of transposition of the great arteries in foetuses reduces neonatal morbidity and mortality. *Circulation* 1999;**99**:916–8.
3 **Fesslova V**, Nava S, Villa L. Evolution and long term outcome in cases with foetal diagnosis of congenital heart disease: Italian multi-centre study. *Heart* 1999;**82**:594–9.
4 **Bull C**, on behalf of the British Paediatric Cardiac Association. Current and potential impact of foetal diagnosis on the prevalence and spectrum of serious congenital heart disease at term. *Lancet* 1999;**354**:1242–7.
5 **Wren C**, Richmond S, Donaldson L. Presentation of congenital heart disease in infancy: implications for routine exam. *Arch Dis Child Fetal Neonatal Ed* 1999;**80**:F49–53.
6 **Abu-Harb M**, Hey E, Wren C. Death in infancy from unrecognised congenital heart disease. *Arch Dis Child* 1994;**71**:3–7.
7 **Kuehl KS**, Loffredo CA, Ferencz C. Failure to diagnose congenital heart disease in infancy. *Pediatrics* 1999;**103**:743–7.
8 **Allan LD**, Sharland GK, Milburn A, *et al*. Prospective diagnosis of 1006 consecutive cases of congenital heart disease in the foetus. *J Am Coll Cardiol* 1994;**23**:1452–8.
9 **Newburger JW**, Wypij D, Bellinger DC, *et al*. Length of stay after infant heart surgery is related to cognitive outcome at age 8 years. *J Pediatr* 2003;**143**:67–73.
10 **Gomelsky A**, Holden EW, Ellerbeck KA, *et al*. Predictors of developmental outcomes in children with complete transposition. *Cardiol Young* 1998;**8**:352–7.
11 **Hovels-Gurich HH**, Seghaye MC, Dabritz S, *et al*. Cognitive and motor development in pre-school and school-aged children after neonatal arterial switch operation. *J Thorac Cardiovasc Surg* 1997;**114**:578–85.
12 **Sharma R**, Choudhary SK, Mohan MR, *et al*. Neurologic evaluation and intelligence treating in the child with operated congenital heart disease. *Ann Thorac Surg* 2000;**70**:575–81.
13 **Majnemer A**, Limperopoulos C. Developmental progress of children with congenital heart defects requiring open heart surgery. *Semin Pediatr Neurol* 1999;**6**:12–9.
14 **Carter BS**, Mcnabb F, Merenstein GB. Prospective validation of a scoring system for predicting neonatal morbidity after acute perinatal asphyxia. *J Pediatr* 1998;**132**:619–23.

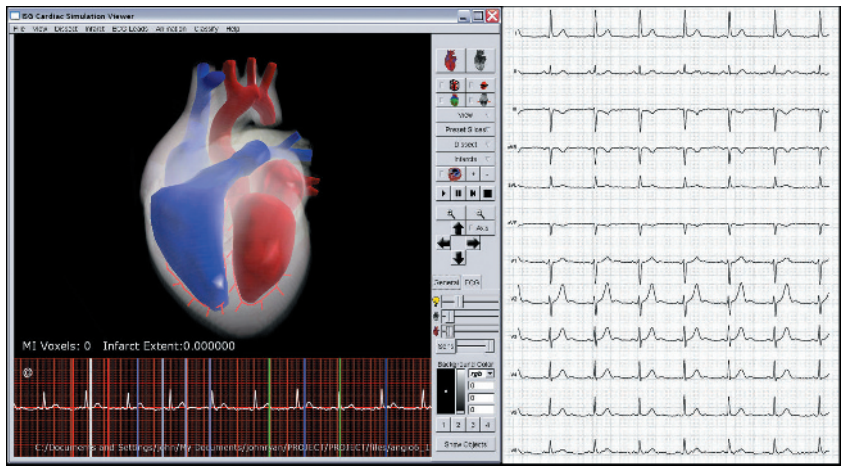
15 **Soonswang J**, Adatia I, Newman C, *et al*. Mortality in potential arterial switch candidates with transposition of the great arteries. *J Am Coll Cardiol* 1998;**32**:753–7.
16 **Perry SB**, Lang P, Keane JF, *et al*. Creation and maintenance of adequate intra atrial communication in left atrioventricular valve atresia and stenosis. *Am J Cardiol* 1986;**58**:622–6.
17 **Mahle WT**, Clancy RR, McGaurn SP, *et al*. Impact of prenatal diagnosis on survival and early neurologic morbidity in neonates with hypoplastic left heart syndrome. *Pediatrics* 2001;**107**:1277–82.
18 **Andrews R**, Tulloh R, Sharland G, *et al*. Outcome of staged reconstructive surgery for hypoplastic left heart syndrome following antenatal diagnosis. *Arch Dis Child* 2001;**85**:474–7.
19 **Gibbs JL**, Monro JL, Cunningham D, *et al*. Survival after surgery or therapeutic catheterisation for congenital heart disease in children in the UK. *BMJ* 2004;**328**:611–6.
20 **Clancy RR**, McGaurn SA, Wernovsky G, *et al*. Preoperative risk-of-death prediction model in heart surgery with deep hypothermic circulatory arrest in the neonate. *J Thorac Cardiovasc Surg* 2000;**119**:347–57.
21 **Jenkins KJ**, Gauvreau K, Newburger JW, *et al*. Consensus-based method for risk adjustment for surgery for congenital heart disease. *J Thorac Cardiovasc Surg* 2002;**123**:111–8.
22 **Shann F**, Pearson G, Slater A, *et al*. Paediatric risk of mortality (PIM): a mortality prediction model for children in intensive care. *Intensive Care Med* 1997;**23**:201–7.

IMAGES IN CARDIOLOGY

doi: 10.1136/hrt.2005.083584

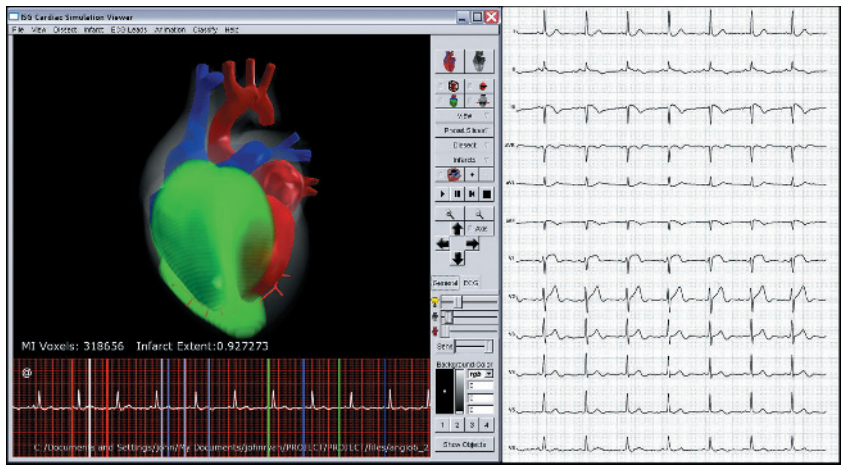
Real time three dimensional imaging of zone of ischaemia derived from surface 12 lead ECG during percutaneous coronary revascularisation

A 71-year-old man presented with accelerated angina over a two week period. He had no significant ECG ischaemia on presentation and troponin T was detectable at 0.04 µg/l. He was also diagnosed with new onset type 2 diabetes during his admission. His diagnostic angiogram revealed moderate atheroma in the left anterior descending and left circumflex coronary arteries with no significant stenosis. The right coronary artery (RCA) was dominant and had two critical stenoses in the proximal and mid segments of the vessel. He underwent uncomplicated percutaneous coronary intervention to the two RCA lesions; two drug eluting stents were implanted, a 2.75 × 13 mm Cypher proximally and a 2.75 × 18 mm Cypher stent in the mid segment. ECG before stent implantation was normal. Stent implantation induced ST segment elevation in leads II, III, aVF, and V1–V3. The ECG signals were converted into a real time three dimensional interactive image of the zone of ischaemia induced by balloon inflation.



Right panel shows 12 lead ECG taken before stent deployment in the proximal right coronary artery (RCA). Left panel shows real time three dimensional image of the heart with the right ventricular chamber in blue and the left ventricular chamber in red derived directly from the 12 lead ECG.

N T Mulvihill
J T Ryan
C Bell
mulvihn@tcd.ie



Right panel shows 12 lead ECG taken after 60 seconds of balloon inflation during stent deployment in the proximal RCA. This ECG demonstrated balloon induced ST segment elevation in leads II, III, aVF, and V1–V3. Left panel shows real time three dimensional image of the heart with the right ventricular chamber in blue, the left ventricular chamber in red, and the zone of transient balloon induced ischaemia in green.