Journal of Paediatrics and Child Health

doi:10.1111/jpc.12066

ORIGINAL ARTICLE

Transporting newborns with transposition of the great arteries

Patricia Woods,[1,2] Kathryn Browning Carmo,[1,2,3] Margaret Wall[1] and Andrew Berry[1]

[1]Newborn and Paediatric Emergency Transport Service (NETS), Sydney Children's Hospitals Network (Westmead), [2]Grace Centre for Newborn Care, Sydney Children's Hospitals Network (Westmead) and [3]The University of Sydney, Sydney, New South Wales, Australia

Aims: The aim of the study was to examine the prevalence and management of outborn babies with a post-natally confirmed diagnosis of transposition of the great arteries (TGA) requiring transport by the Newborn and Paediatric Emergency Transport Service (NETS), New South Wales during the epoch 1991–2010.

Method: A retrospective audit of NETS database and case notes. The physiological status, interventions and any complications encountered from the point of referral to NETS (pre-transport), stabilisation (transport) and subsequent admission to the receiving hospital (post-transport) were evaluated.

Results: One hundred fifty-seven infants with TGA were transported, with an average of eight per year (1:11 598 births). Seven (4%) had an antenatal diagnosis, and 72 (46%) had a post-natal diagnosis prior to referral. Physiological and clinical parameters demonstrated overall clinical stability; however, 47% of the babies had a PaO_2 <30 mmHg, and approximately one-fifth had oxygen saturations <70%. Rates of mechanical ventilation and prostaglandin E_1 administration were approximately 50%. A quarter of transported babies encountered a transport-related event, including one death and two babies for which a decision was reached to forego life-sustaining treatment at the referring hospital.

Conclusions: Most newborns with TGA remain stable or improve during transport. There is a rate of adverse events; however, this reinforces the need to facilitate delivery where there is ready access to interventional paediatric cardiology services.

Key words: newborn; perinatal care; retrieval; transposition of great vessels.

What is already known on this topic

- Infants with transposition of the great arteries (TGA) risk rapid haemodynamic compromise following birth with inadequate circulatory mixing. Prompt specialist intervention in the form of atrial balloon septostomy and tertiary resuscitative care may be critical.
- A prenatal diagnosis of TGA has been demonstrated to reduce pre-operative morbidity and perioperative mortality and facilitates planned delivery in an appropriate perinatal environment.
- Infants with congenital heart disease are at a significant risk of transport-related morbidity, however, limited data exist documenting the transport process of newborns with TGA.

What this paper adds

- Detailed clinical and physiological data on transporting newborns with TGA over the past 20 years.
- Documentation of transport process from referral, stabilisation and during transport to a specialist centre providing definitive care.
- Information of the specific risks of post-natal transfer of TGA infants, including rates of adverse events.

Transposition of the great arteries (TGA) is defined as concordant atrioventricular and discordant ventriculoarterial connections. Simple TGA without ventricular septal defect (VSD) is demonstrated in 80% of cases (dextro-TGA); others are associated with more complex congenital heart disease (CHD).[1] TGA has an incidence of 0.5 per 1000 births (including live and stillborn infants) in New South Wales (NSW).[2] Liveborn infants risk rapid haemodynamic compromise with inadequate circulatory mixing at atrioventricular level despite patency of the ductus arteriosus (DA). Prompt specialist intervention, tertiary resuscitative care and/or balloon atrial septostomy (BAS) may be critical.[3] Mortality rates from TGA estimate 2.6–4%, and optimising pre-operative management is salient.[4–6]

Antenatal ultrasound detection is difficult, given the four-chamber cardiac view is essentially normal and the lack of crossing of the outflow tracts may not be appreciated.[7] Reported detection rates across the globe are variable from 15% to 53%.[8–13]

Systematic review demonstrates marked variation in the sensitivity of detection of CHD by screening fetal echocardiography by a cardiologist in unselected, low-risk populations; therefore, this practice is not routine.[13]

Correspondence: Dr Patricia Woods, Newborn and Paediatric Emergency Transport Service (NETS), Sydney Children's Hospitals Network (Westmead), Sydney, NSW 2145, Australia. Fax: +61 2 9633 8782; email: patricia.woods@sswahs.nsw.gov.au

Declaration of conflict of interest: None declared.

Accepted for publication 22 May 2012.

A subgroup of infants with TGA have identified predictive markers for being at high risk of morbidity and mortality.[6,14] Antenatal restriction of the foramen ovale and/or the DA at 36 weeks gestation had a high specificity at 84%, but low sensitivity (54%) in predicting babies who developed a critical cyanosis early after delivery.[14] Therefore, it is recommended that an antenatal diagnosis of TGA leads to a planned delivery in a centre with interventional cardiology services.[3]

Limited data documenting the transport process of newborns with TGA specifically exist. Yeager *et al*. reported 45% of infants with CHD transported had suboptimal clinical status regarding acidosis, hypothermia and low oxygen saturations.[15] There are highlighted episodes of physiological deterioration in up to 8% of transported neonatal patients, particularly if carried out by non-specialist retrieval teams.[16,17] The Newborn and Paediatric Emergency Transport Service (NETS) is a dedicated newborn and paediatric retrieval service for the Australian state of NSW, the Australian Capital Territory (ACT) and the islands in the South Pacific rim. NSW has a birth rate approaching 96 000, and NETS (http://www.nets.org.au) transports approximately 1000 critically unwell newborns each year.[4]

Study Methods

A retrospective audit of the NETS NSW database and case notes of all outborn babies with a post-natally confirmed diagnosis of TGA who required NETS retrieval during the epoch January 1991–August 2010 was conducted. Only the first transport of babies at presentation with suspected or confirmed diagnosis of TGA was evaluated. Exclusion criteria applied to newborns with a subsequent diagnosis other than TGA, any elective, non-primary transports of known TGA infants, transports by non-NETS retrieval teams and cases with associated complex CHD.

The timing of diagnosis of TGA was examined. Transportation data were collated on the duration, distance and modality of transport (fixed wing, rotary wing and road ambulance).

The total duration of the transport process was calculated from the time of NETS team tasking to the point of admission into the accepting specialist unit. Specific operational times by NETS teams were analysed: from 'first look' (initial assessment by NETS team on arrival) to 'stabilisation' (point at which the baby is deemed ready for transport); 'first look' to point of admission to receiving unit; and journey time (departure from referring hospital to admission to the receiving unit).

Interventions were examined; specifically, that of mechanical ventilation, use of prostaglandin E_1 (PGE_1) and inotropes and performance of urgent BAS upon admission to the specialist hospital. Physiological and clinical parameters were collated from three time points: pre-transport (point of referral) and/or NETS 'first look', at NETS 'stabilisation' and at admission to accepting specialist unit. The proportion exhibiting suboptimal physiological and clinical parameters was analysed. Values deemed suboptimal were pH $\leq$7.25, PaO_2 $\leq$30 mmHg, O_2 saturations $\leq$70%, base excess $\geq$−10, temperature <36.0°C, glucose <2.6 mmol/L, heart rate >160 bpm, respiratory rate >60 bpm, capillary refill time >3 s and mean arterial blood pressure <40 mmHg (<5th percentile for mean patient demographic of 38 weeks gestation, birthweight 3250 g and post-natal age <72 h).[18]

Rates of complications and adverse events were either transport specific (e.g. equipment failure) or related to clinical care (e.g. drug error).

Statistical Methods

Data were analysed using SPSS version 16 for Windows (IBM Corporation, Armonk, NY, USA). Baby demographics and physiological and clinical variables are expressed as means ± standard deviations and ranges. Transportation data are expressed as median values with ranges. One-way analysis of variance was utilised to compare the means of each variable between the three time points (NETS first look, stabilisation and admission post-transport). Bivariate analysis (χ^2) compared the proportion of TGA cases with suboptimal clinical status at each time point. Linear regression analysis explored the frequency of TGA transports over time. A *P*-value of <0.05 was considered statistically significant.

Results

Between January 1991 and August 2010, 199 babies were transported with TGA. Forty-two cases were excluded: 15 non-primary transfers of infants with known TGA, 19 transported by non-NETS teams and 8 cases with associated complex CHD in addition to TGA. NETS NSW was asked to transport a total of 157 babies with TGA. Three babies died after the NETS team 'first look' and prior to transport.

This is an average of eight infants with TGA transported per year (approximately 1:12 000 births). Epoch analysis demonstrated a decreasing trend in the frequency transported; however, it does not achieve statistical significance ($P = 0.08$). The male-to-female sex ratio was 2:1 (male 105, female 52).

Four per cent of the cases (seven) had an accurate antenatal diagnosis, while one additional case was diagnosed in error. The scan at 21 weeks gestation was suggestive of TGA, but the subsequent scan at 31 weeks gestation declared normal. A post-natal diagnosis was made in 46% (72) prior to referral, one of which had an antenatal diagnosis of isolated VSD. Thirteen cases (8%) were diagnosed following an initial retrieval and subsequently required a second transport to a tertiary cardiac institution. Sixty-three babies (40%) retrieved had no formal diagnosis of TGA until admission at the receiving specialist unit. One baby had a diagnosis of TGA and was confirmed only at post-mortem examination.

The babies who were referred and not transported were so moribund that a joint parental, referring and receiving clinician decision was made to forego life-sustaining treatment at the referring hospital ($n = 3$).

The majority of babies transported were of normal birthweight (3277 ± 699 g), term gestation (38 weeks, 5 days, ±16 days) and less than 72 h post-natal age (mean 65 ± 215 h).

Just over half of the babies were mechanically ventilated; 54% (84/157) and 57% (90/157) received PGE_1 infusion for transport. Of the babies who received PGE_1 infusion, 77% (70/90) were mechanically ventilated. Following commencement of PGE_1, 15% (14/90) developed pyrexia of $\geq$38.0°C, and 11% (18/157) received inotropic support. Upon admission to the specialist accepting unit, 39% (61/157) underwent urgent BAS.

Table 1 Duration and NETS operational times of TGA transports

TGA transports	Total duration of transport (team task to admission)		NETS stabilisation times (first look to stabilisation)		NETS case times (first look to admission)		NETS travel times (departure from referring hospital to admission)	
	Median (h : min)	Range	Median (h : min)	Range	Median (h : min)	Range	Median (h : min)	Range
Total TGA transports	03:44	01:01–21:25	01:05	00:04–04:15	02:35	00:49–13:45	00:58	00:08–13:00
TGA Australia	03:35	01:01–10:31	01:10	00:04–04:15	02:30	00:49–06:25	00:55	00:08–02:50
TGA international	16:40	15:00–21:25	00:32	00:13–04:13	08:23	07:04–13:45	05:57	05:06–13:00

NETS, Newborn and Paediatric Emergency Transport Service; TGA, transposition of the great arteries.

A total of 124 TGA transports occurred within NSW, and 19 were referred from the ACT. Sixty-one per cent were transported via road, 32% via rotary wing and 7% via fixed wing. Fourteen international retrievals to New Caledonia were undertaken (distance 1983 km, median duration 16 h, range 15–21 h 25 min). The median distance of all TGA transports within NSW/ACT was 33 km (range 0.5–600 km), and the median duration was 3 h 35 min (range 1 h 1 min–10 h 31 min) (Table 1).

In three cases, there was a decision to forego life-sustaining treatment at the referring hospital. In 1992, case 1 was a 4-kg term infant with an antenatal diagnosis of TGA who was referred to deliver at a tertiary centre with specialist cardiology services in Sydney. With the onset of a precipitous labour, however, the infant was delivered at an alternative tertiary unit in closer proximity. The infant developed marked respiratory distress with cyanosis (O_2 saturations <50% in FiO_2 1.00), circulatory shock and hypothermia (temperature 30.0°C). In 2006, case 2 was a 3.9-kg term infant also born in a tertiary unit in Sydney. The infant developed severe cyanosis at 20 min of life; echocardiogram confirmed the presence of TGA with an intact septum. Despite mechanical ventilation, PGE_1 infusion and inotropic support, the baby remained profoundly acidotic (pH 6.5 base excess-29), shocked and hypoxic (O_2 saturations <50%). The baby developed fixed and dilated pupils and proceeded to cardiorespiratory arrest. In 2009, case 3 was a 3.5-kg term infant born in a remote rural area of NSW, and the diagnosis of TGA was confirmed only at post-mortem. The infant developed marked respiratory distress and cyanosis following birth, with a recorded PaO_2 of 11 mmHg (in FiO_2 1.0), pH 6.9 and temperature of 35.1°C at NETS first look. The infant was mechanically ventilated and received inotropic support but did not receive PGE_1 infusion. The infant remained severely acidotic and deteriorated with cardiorespiratory arrest, fixed and dilated pupils and died despite extensive resuscitation attempts.

A quarter of cases transported had complications or an adverse transport-related event (39/157, 24.8%) (Table 2). Fifteen cases reported significant clinical incidents, including one case where the infusion of 10% dextrose was administered through a peripheral arterial cannula instead of peripheral venous cannula, and another case where prostacyclin infusion was commenced instead of PGE_1. Both incidents were identified without adverse consequence. The incidence of hypoglycaemia (15%) and temperature instability (9%) highlighted in Table 3 are clinically significant. Fifteen per cent of those ventilated (13/84) had evidence of receiving excessive ventilation, with a recorded pH of >7.46 (nine cases at the point of NETS stabilisation and five at the point of admission to the accepting unit, with one case at both time points).

Table 2 Complications/adverse events of TGA transports

Complications	Frequency
Foregoing of life-sustaining treatment	3
Equipment failure	4
Delay: NETS team availability	5
Delay: transport availability	3
Clinical incidents	15
Drug error	1
Access	1
Excessive ventilation	13
Clinical instability	9
Desaturations	4
Hypotension	3
Bradycardia	1
Seizures/hypotension	1

NETS, Newborn and Paediatric Emergency Transport Service; TGA, transposition of the great arteries.

Nine cases were reported to be clinically unstable during the transport process, requiring further management, including desaturations (four), hypotension (three), bradycardia (one) and seizures with hypotension (one). One baby required bilateral drainage of pneumothoraces following vigorous bag mask ventilation in the face of severe cyanosis at the referring hospital.

Discussion

NETS transported an average of eight infants with TGA per year between 1991 and 2010. A further 19 cases during this time period were transported by other retrieval modalities; therefore, the true rate of outborn infants with TGA requiring transport is

Table 3 Patients with abnormal clinical and physiological status

Parameter	Pre-transport	Transport	Post-transport	*P*-value
	% (*n*/total)	% (*n*/total)	% (*n*/total)	
pH ≤7.25	20 (27/130)	17 (11/62)	9 (6/67)	0.110
PaO_2 ≤30	61 (75/122)	59 (36/61)	47 (31/65)	0.183
BE ≥–10	13 (16/120)	19 (12/61)	6 (4/65)	0.077
Temperature ≤36 C	9 (14/154)	7 (11/143)	2 (2/144)	0.013
Heart rate >160	26 (40/154)	22 (34/152)	25 (38/149)	0.729
Respiratory rate >60	20 (30/152)	16 (21/152)	16 (23/147)	0.361
O_2 saturation ≤70%	45 (71/156)	29 (44/107)	22 (33/149)	<0.001
Mean BP <40 mmHg	26 (25/94)	20 (15/91)	25 (15/59)	0.640
Blood glucose <2.6	12 (13/110)	15 (15/85)	14 (9/64)	0.816
CRT >3	3 (5/148)	<1 (1/134)	<1 (1/128)	0.145

BE, base excess; BP, blood pressure; CRT, capillary refill time.

slightly higher. The rate of antenatal diagnosis of TGA among outborn infants (4%, $n = 7/157$) is high, given that antenatal diagnosis is an indication for *in utero* referral and delivery in an appropriate tertiary perinatal centre adjacent to paediatric cardiac and surgical facilities.[19] Forty-six per cent (72) of the babies had a diagnosis of TGA prior to or during the retrieval, including two using a laptop-sized ultrasound by the retrieval team. In sixty-three babies (40%), the diagnosis was clinical until arrival at the specialist paediatric cardiology service. On 8% of the occasions, the baby was initially transferred to a tertiary perinatal centre and then required a second retrieval to a hospital with a paediatric cardiology service. This doubles the clinical risk to the babies transported and also has a financial impact on the health service.

Table 3 shows the proportion of babies in each time point of the retrieval process with abnormal physiological and clinical parameters. There are limitations in the power analysis and interpretation of comparisons due to a reduction in the number of babies in which they are recorded across the time points, which may reflect the expedited transport process undertaken via NETS to specialist receiving institutions.

Yates reported 17% of TGA babies exhibited deterioration in one or more metabolic measurements during transport and a post-natal diagnosis of TGA was associated with a higher incidence of pre-operative collapse.[20] Bonnet *et al*. reported 12% (prenatal) versus 22% (post-natal group) of TGA babies had a metabolic acidosis (defined as pH <7.10).[21] In this study, the incidence of acidosis improved during retrieval, with less than 10% of cases with pH <7.25 upon admission to the specialist unit. The physiological and clinical parameters of the transported babies demonstrated overall stability. Nearly half had PaO_2 of <30 mmHg, and approximately one-fifth had oxygen saturations of <70%, emphasising the need to rapidly transfer these babies to a tertiary care centre where BAS can be performed urgently. A PaO_2 value of <50 mmHg despite an FiO_2 of 1.0 can be expected in infants with TGA. There are currently no published data available for comparison on the PaO_2 status of TGA babies during transport.[16,22,23] The main differential diagnosis for this clinical presentation with low PaO_2 values in the absence of echocardiography is persistent pulmonary hypertension of the newborn.

The mean oxygen saturation values approximate 75%, with no significant difference across the retrieval process $P = 0.082$, and are comparable with those reported by Raboisson *et al*.[24] The proportion of babies with oxygen saturations of ≤70% improved significantly over the successive time points (pre-transport 45% (71/156), transport 29% (44/107), post-transport 22% (33/149), $P < 0.001$). Improving oxygen delivery to such infants facilitates the relaxation of pulmonary vascular resistance (which may be elevated secondary to the hypoxia).

The incidence of hypoglycaemia (15%) and temperature instability (9%) are comparable with rates reported by Lang *et al*. in their study of neonatal CHD transports (13% and 11%, respectively)[22] and remain significant with scope for future clinical improvements when transporting this group of infants identified at particular risk.

Approximately, half (54%) of the babies were mechanically ventilated for transport, and over half received PGE_1 infusion. This is comparable with rates reported by other studies (47–66%).[23,25]

Fuchs *et al*. demonstrated 18% of babies with post-natal diagnosis of TGA had evidence of ductal closure prior to commencement of PGE_1 infusion.[26] PGE_1 infusion may not have been commenced by NETS teams in some instances of diagnostic uncertainty, not considered as a treatment option or to avoid further delay in transportation. One death occurred in a severely cyanotic baby, where PGE_1 infusion was not considered, and subsequently, post-mortem examination revealed the diagnosis. This reinforces the need to commence PGE_1 infusion in cases of diagnostic uncertainty in the face of profound perinatal cyanosis.

Adverse effects of PGE_1 infusion include apnoea, hypotension, vasodilatation, arrhythmias and pyrexia. Given the risks of apnoea and clinical instability in the transport environment, traditionally, babies have been electively mechanically ventilated; however, this does carry the risk of endotracheal tube

occlusion, displacement and equipment failure. Meckler and Lowe found that elective intubation is a significant predictor of major transport complications odds ratio 20.56, 95% CI 3.34–113.09.[25] Fifteen per cent of the babies developed pyrexia of ≥38.0°C after commencement of PGE_1. Meckler and Lowe reported higher rates of adverse effects (total 38%, specifically 18% apnoea and 13% hypotension), particularly at PGE_1 infusions of >50 ng/kg/min (odds ratio 3.72 95%, CI 1.10–12.63).[26] Much lower doses of PGE_1 (10 ng/kg/min) have been shown to be just as successful with DA patency and with a safer side effect profile, specifically in relation to risk of apnoea.[27,28]

Almost 40% of cases underwent BAS upon admission to the specialist unit, reinforcing the necessity for expedited transfer of these infants with confirmed or suspected TGA. BAS has been reported to have a higher success rate in cases with an antenatal diagnosis of TGA (81% vs. 51%, $P < 0.001$).[27] In contrast, Bonnet *et al*. reported no significant difference between groups and only 12% of babies with an antenatal diagnosis of TGA required urgent BAS.[21]

The median stabilisation time for all TGA transports was 1 h 05 min, and this is consistent with other international dedicated transport services.[17,29] This is less than the median time for all NETS NSW neonatal retrievals (1 h 25 min) and reflects the urgency of transportation to a specialist unit for definitive care. The NETS policy is to obtain expert clinical advice from the point of referral and during stabilisation of an infant via telephone conference calls. This ensures delivery of optimal care and assists the preparation of the receiving specialist unit.

In the three TGA cases where life-sustaining treatment was foregone, there were no attempts of performing BAS at the referring institution. This was due to the lack of interventional cardiology skills. Each case differed in the timing of diagnosis: case 1 antenatally, case 2 post-natally and case 3 only at post-mortem. Our transport-related mortality rate of 1.9% for TGA infants is comparable with the report by Hellström-Westas *et al*. of 0.7% for long-distance transports for infants with CHD.[23] Although our mortality rates are low, three deaths occurring in a condition that is potentially treatable remain significant.

Conclusions

NETS NSW transports an average of eight babies with TGA each year. The majority demonstrate overall clinical stability during transport, despite a low mean PaO_2 of 30 mmHg and O_2 saturations of 75%. Post-natal transport is an undesirable alternative, given that one-quarter experience adverse events and one in 50 die. Until fetal detection rates improve, allowing optimal place of birth decisions, NETS will continue to focus on clinical review to improve post-natal transport of these babies.

Acknowledgements

Thank you to the dedicated NETS teams for their service.

References

1 Johnson TR. Conotruncal cardiac defects: a clinical imaging perspective. *Pediatr. Cardiol.* 2010; **31**: 430–7.
2 Centre for Epidemiology and Research. NSW Department of Health. New South Wales mothers and babies 2008. *Public Health Bull. Suppl.* 2010; **21** (S2): 94.
3 Bu'Lock FA. Transporting babies with known heart disease; who, what and where? *Arch. Dis. Child. Fetal Neonatal Ed.* 2007; **92**: F80–1.
4 Khoshnood B, De Vigan C, Vodovar V *et al*. Trends in prenatal diagnosis, pregnancy termination, and perinatal mortality of newborns with congenital heart disease in France, 1983–2000: a population-based evaluation. *Pediatrics* 2005; **115**: 95–101.
5 Soongswang J, Adatia I, Newman C, Smallhorn JF, Williams WG, Freedom RM. Mortality in potential arterial switch candidates with transposition of the great arteries. *J. Am. Coll. Cardiol.* 1998; **32**: 753–7.
6 Jouannic JM, Gavard L, Fermont L *et al*. Sensitivity and specificity of prenatal features of physiological shunts to predict neonatal clinical status in transposition of the great arteries. *Circulation* 2004; **110**: 1743–6.
7 Huhta JC. Evaluating the fetus with transposition. *Cardiol. Young* 2005; **15** (Suppl 1): 88–92.
8 Grandjean H, Larroque D, Levi S. The performance of routine ultrasonographic screening of pregnancies in the Eurofetus Study. *Am. J. Obstet. Gynecol.* 1999; **181**: 446–54.
9 Jaeggi ET, Sholler GF, Jones OD, Cooper SG. Comparative analysis of pattern, management and outcome of pre- versus postnatally diagnosed major congenital heart disease: a population-based study. *Ultrasound Obstet. Gynecol.* 2001; **17**: 380–5.
10 Chew C, Stone S, Donath SM, Penny DJ. Impact of antenatal screening on the presentation of infants with congenital heart disease to a cardiology unit. *J. Paediatr. Child Health* 2006; **42**: 704–8.
11 Blyth M, Howe D, Gnanapragasam J, Wellesley D. The hidden mortality of transposition of the great arteries and survival advantage provided by prenatal diagnosis. *BJOG* 2008; **115**: 1096–100.
12 Sholler GF, Kasparian NA, Pye VE, Cole AD, Winlaw DS. Fetal and post-natal diagnosis of major congenital heart disease: Implications for medical and psychological care in the current era. *J. Paediatr. Child Health* 2011; **47**: 717–22.
13 Randall P, Brealey S, Hahn S, Khan KS, Parsons JM. Accuracy of fetal echocardiography in the routine detection of congenital heart disease among unselected and low risk populations: a systematic review. *BJOG* 2005; **112**: 24–30.
14 Maeno YV, Kamenir SA, Sinclair B, van der Velde M, Smallhorn F, Hornberger LK. Prenatal features of ductus arteriosus constriction and restrictive foramen ovale in d-transposition of the great arteries. *Circulation* 1999; **99**: 1209–14.
15 Yeager SB, Horbar JD, Greco KM, Duff J, Thiagarajan RR, Laussen PC. Pretransport and posttransport characteristics and outcomes of neonates who were admitted to a cardiac intensive care unit. *Pediatrics* 2006; **118**: 1070–7.
16 Kanter RK, Boeing NM, Hannan WP, Kanter DL. Excess morbidity associated with interhospital transport. *Pediatrics* 1992; **90**: 893–8.
17 Britto J, Nadel S, Maconochie I, Levin M, Habibi P. Morbidity and severity of illness during interhospital transfer: impact of a specialised paediatric retrieval team. *BMJ* 1995; **311**: 836–9.
18 Pejovic B, Peco-Antic A, Marinkovic-Eric J. Blood pressure in non-critically ill preterm and full-term neonates. *Pediatr. Nephrol.* 2007; **22**: 249–57.
19 New South Wales Department of Health, Statewide Services Development. *NSW Health Policy Directive Antenatal Maternal Referral/Transfer: Known Congenital Anomalies Likely to Require Surgery*. 2005. Available from: http://www.health.nsw.gov.au [accessed 18 December 2012].
20 Yates RS. The influence of prenatal diagnosis on postnatal outcome in patients with structural congenital heart disease. *Prenat. Diagn.* 2004; **24**: 1143–9.

21 Bonnet D, Coltri A, Butera G *et al*. Detection of transposition of the great arteries in fetuses reduces neonatal morbidity and mortality. *Circulation* 1999; **99**: 916–8.
22 Lang A, Brun H, Kaaresen PI, Klingenberg C. A population based 10-year study of neonatal air transport in North Norway. *Acta Paediatr.* 2007; **96**: 995–9.
23 Hellström-Westas L, Hanséus K, Jögi P, Lundström NR, Svenningsen N. Long-distance transports of newborn infants with congenital heart disease. *Pediatr. Cardiol.* 2001; **22**: 380–4.
24 Raboisson MJ, Samson C, Ducreux C *et al*. Impact of prenatal diagnosis of transposition of the great arteries on obstetric and early postnatal management. *Eur. J. Obstet. Gynecol. Reprod. Biol.* 2009; **142**: 18–22.
25 Meckler GD, Lowe C. To intubate or not to intubate? Transporting infants on prostaglandin E1. *Pediatrics* 2009; **123**: e25–30.
26 Fuchs IB, Müller H, Abdul-Khaliq H, Harder T, Dudenhausen JW, Henrich W. Immediate and long-term outcomes in children with prenatal diagnosis of selected isolated congenital heart defects. *Ultrasound Obstet. Gynecol.* 2007; **29**: 38–43.
27 Kramer HH, Sommer M, Rammos S, Krogmann O. Evaluation of low dose prostaglandin E1 treatment for ductus dependent congenital heart disease. *Eur. J. Pediatr.* 1995; **154**: 700–7.
28 Browning Carmo KA, Barr P, West M, Hopper NW, White JP, Badawi N. Transporting newborn infants with suspected duct dependent congenital heart disease on low-dose prostaglandin E1 without routine mechanical ventilation. *Arch. Dis. Child. Fetal Neonatal. Ed.* 2007; **92**: F117–9.
29 Whitfield JM, Buser MK. Transport stabilization times for neonatal and pediatric patients prior to interfacility transfer. *Pediatr. Emerg. Care* 1993; **9**: 69–71.