Journal of Perinatology (2006) 26, 170–175

www.nature.com/jp

ORIGINAL ARTICLE

Monochorionic monoamniotic twins: neonatal outcome

L Cordero[1], A Franco[2] and SD Joy[2]

[1]Department of Pediatrics and Obstetrics, Division of Neonatal-Perinatal Medicine, College of Medicine and Public Health, The Ohio State University Medical Center, The Ohio State University, Columbus, OH, USA and [2]Department of Obstetrics and Gynecology, College of Medicine and Public Health, The Ohio State University, Columbus, OH, USA

Background: Monochorionic monoamniotic twins (MoMo) occur in one of 10 000 pregnancies. Cord entanglement, malformations, twin-to-twin transfusion syndrome (TTS) and prematurity are responsible for their high perinatal morbidity and mortality.

Objective: To report our experience with 36 sets of MoMo twins (1990 to 2005) and to provide updated information for counseling.

Methods: Chorionicity was determined by placental examination, gestational age and TTS clinically and by sonography. Intrauterine growth restriction (IUGR) was diagnosed with a twin-specific nomogram.

Results: Cord entanglement was observed in 15 pregnancies, but only one twin with entanglement and a true knot, experienced related morbidity. Four of 71 live births were IUGR. Malformations were diagnosed prenatally (one hypoplastic left heart and one body stalk) and postnatally (one vertebral anomalies-anal atresia-tracheoesophageal fistula-renal defect (VATER) and two lung hypoplasias). Twin-to-twin transfusion syndrome affected three sets of twins. Five twin sets delivered before 31, 19 sets at 31 to 32 and 12 sets at 33 to 34 weeks. Six of 71 (8%) twins died (four malformations, one TTS and one 26 weeks premature). Head ultrasounds in 59 of 65 survivors showed two (3%) periventricular leukomalacia, five (9%) Grade I–II intraventricular hemorrhage and 52 (88%) normal.

Conclusions: Monochorionic monoamniotic twins remain a group at risk for cord entanglement, congenital malformations, TTS and prematurity. Although their neonatal mortality and morbidity is high, outcomes for survival are better than anticipated.
Journal of Perinatology (2006) **26,** 170–175. doi:10.1038/sj.jp.7211457; published online 9 February 2006

Keywords: monochorionic; monoamniotic; twins; perinatal; neonatal; outcome

Introduction

Every year 4 million infants are born in the United States and approximately 130 000 of them are twins.[1] The prevalence of twin deliveries has increased 48% between 1990 and 1998.[1] Of great concern is that although twins represented only 2.5% of the entire group, they accounted for 12.6% of the total perinatal mortality.[2]

Only one-third of all twins are monozygotic, but 75% of the monozygotic twins are monochorionic. Among monochorionic twins, about 2% are monochorionic monoamniotic (MoMo), 18% monochorionic diamniotic (MoDi) with twin-to-twin transfusion syndrome (TTS) and 80% MoDi without TTS.[3] Monochorionic monoamniotic twins have the highest incidence of perinatal losses, congenital malformations, perinatal mortality and perinatal morbidity.[3–5]

The purpose of the present investigation is to report the clinical outcome of 36 sets of MoMo twins consecutively born in our Institution. We also provide practicing neonatologists with updated information for counseling parents.

Methods

Demographic and clinical data were obtained from medical records of women with MoMo twin pregnancies and their infants born at The Ohio State University Medical Center between 1990 and 2005. This study was approved by the Institutional Review Board. Conjoined twins and gestations other than twins were excluded. Gestational age (GA) was determined by sonography or by examination of the newborn infant. Within each set, twins were classified according to their birth weight into either large or small size. Inter-twin growth discordance was calculated as the difference between the birth weight of the large and the small twin expressed as a percentage of the large twin birth weight.[6] Twin pregnancies were considered discordant if the inter-pair differences in birth weights were $\geqslant 20\%$.

Chorionicity was established by histological examination of the placenta and zygosity was determined by chorionicity, infant's gender and blood group.[7] Based on sonographic and pathologic evidence, all monochorionic gestations were classified as MoMo or MoDi. Twin-to-twin transfusion syndrome was diagnosed by the

Correspondence: Dr L Cordero, Department of Pediatrics and Obstetrics, Division of Neonatal-Perinatal Medicine, The Ohio State University Medical Center, The Ohio State University, N118 Doan Hall, 410 W. 10th Avenue, Columbus, OH 43210-1228, USA.
E-mail: leandro.cordero@osumc.edu
Received 3 October 2005; revised 8 December 2005; accepted 19 December 2005; published online 9 February 2006

recognition of a single placenta, same gender, twins weight discordance, intrauterine growth restriction in the small (donor), normal or large size of the recipient, hemodynamic compromise of the recipient and polyhydramnios.[8,9] Twin-to-twin transfusion syndrome severity was staged sonographically.[10]

Acute fetal distress was diagnosed by fetal heart rate abnormalities (late decelerations or decreased beat to beat variability) or by decreases in the biophysical profile score.[11] Chronic fetal distress was defined by growth restriction in combination with abnormal fetal Doppler values, oligohydramnios or with both.[11] Neonatal depression was considered severe when 1- and 5-min Apgar scores were $\leqslant 3$. Depending on maternal and fetal conditions, MoMo pregnancies were monitored as in-patient or as outpatient. Fetal management goal was to deliver by elective cesarean at 32 to 34 weeks of gestation.

Birth weight and GA for each individual twin were plotted on a growth nomogram specific for twins.[12] Infants who plotted at the 10th percentile or lower were considered intrauterine growth restricted. Umbilical cord hemoglobin (Hgb) was studied. For the few instances when cord blood was not available, the first arterial or venous Hgb recorded was used.

Respiratory distress syndrome was diagnosed using clinical and radiographic parameters. The need for mechanical ventilation and for exogenous surfactant was determined by the attending neonatologist. Severe bronchopulmonary dysplasia was diagnosed in infants receiving supplemental oxygen at 36 weeks postconceptional age. Intraventricular hemorrhages were documented by head ultrasound and were graded.[13]

Statistical analysis

Comparisons between groups and subgroups were made using the Student's *t*-test for interval and χ^2 analysis and Fisher's exact test for categorical data. Values were reported as mean, standard deviation (s.d.), range and median.

Results

The study population consisted of 36 women with MoMo pregnancies and their seventy-one live born infants (Table 1). None of the pregnancies were conceived with artificial reproductive techniques. Twenty-five (69%) of the mothers were Caucasian and 11 (31%) were African American. The median age was 25 years (range 16 to 35) and one-third of the mothers were primigravida. Twenty-six of the 36 (72%) twin sets were females.

Antepartum complications noted in 36 pregnancies included discordant fetal growth in four (11%), fetal growth restriction in one or both twins in four (11%), preeclampsia in three (9%), congenital malformations in four (11%) and TTS in three (9%). Two of the three pregnancies complicated by TTS were treated with serial amnioreductions. Acute or chronic fetal distress affected 10 (28%) of the pregnancies and preterm labor was noted in 19 (53%) of the cases.

Table 1 Monochorionic monoamniotic pregnancies

Pregnant patients no.	36
Maternal age (years)	25 (16–34)
Primigravida no. (%)	11 (31)
Discordant fetal growth no. (%)	4 (11)
Fetal growth restriction no. (%)	4 (11)
Preeclampsia no. (%)	3 (9)
Preterm labor no. (%)	19 (53)
Congenital malformations no. (%)	4 (11)
Twin-to-twin transfusion no. (%)	3 (9)
Acute/chronic fetal distress no. (%)	10 (29)
Antepartum steroids no. (%)	33 (92)
Umbilical cord entanglement no. (%)	15 (42)
Umbilical cord true knot no. (%)	5 (14)
Cesarean delivery no. (%)	34 (94)
Live births no. (%)	71 (99)
$\leqslant$30 weeks gestational age at delivery no. (%)	5 (14)
31–32 weeks gestational age at delivery no. (%)	19 (53)
33–34 weeks gestational age at delivery no. (%)	12 (33)

Antepartum steroids were given to 33 (92%) of the 36 women who delivered vaginally (two cases) or by cesarean (34 cases). All 36 mothers delivered 71 live infants and one stillbirth. Although this delivery occurred at 32 weeks, an autopsy failed to determine the cause for fetal demise that occurred at approximately at 22 weeks. Five (14%) twin sets were delivered at $\leqslant$30 weeks, 19 (53%) sets at 31 to 32 weeks and 12 (33%) sets at 33 to 34 weeks.

Umbilical cord entanglement

Entanglement was diagnosed at the time of delivery in 15 of the 36 (42%) pregnancies. In 10 of these cases (67%), entanglement occurred without true knots and none of these twins showed complications attributable to umbilical cord accidents. In the remaining five (33%) pregnancies, there were entanglements of the cord with true knots. Eight twins experienced no complications. In the remaining case, twin 'A', whose true knot was very tight, had Apgar scores of 2 and 3, was anemic (10 g/dl cord Hgb), required packed red blood cells for treatment of hypotension, but survived without complications. Twin 'B' had Apgar scores 6 and 6, was polycythemic (20 g/Hgb) and had an uncomplicated hospital course.

Only one of the 30 (3%) infants with entangled cord was considered to be intrauterine growth restricted.

Congenital malformations

There were four pregnancies complicated by congenital malformations. In one, both twins were affected, and in the remaining three pregnancies, only one twin was affected (Table 2).

Table 2 Major morbidities in seven monoamniotic monochorionic pregnancies

	Pregnancy	*Complication*	*Diagnosed at*	*At delivery*		*Hospital course*	*Outcome*
				GA (w)	*Bwgt (g)*		
1	A female	Normal		29	1772	Uncomplicated	Survived
	B female	Body stalk anomally	20 weeks		1600	Resp. failure	Died 5 min
2	A female	Hypoplastic L heart[a]	25 weeks	34	2002	Stage I Norwood	Survived
	B female	Normal			2200	Uncomplicated	Survived
3	A male	Normal		31	1490	RDS NEC	Survived
	B male	VATER[a]	After birth		1340	Renal failure	Died 6 days
4	A female	Lung hypoplasia, R[a]	After birth	30	1170	RDS PIE	Died 11 days
	B female	Lung hypoplasia, L[a]	After birth		1040	RDS PIE	Died 31 days
5	A male	TTS (recipient)	22 weeks	27	870	RDS BPD PVL	Survived
	B male	TTS (donor)			618	RDS BPD PVL	Survived
6	A male	TTS (recipient)	24 weeks	27	894	RDS nl head U/S	Survived
	B male	TTS (donor)			747	RDS nl head U/S	Survived
7	A male	TTS (recipient)	32 weeks	33	2158	nl head U/S	Survived
	B male	TTS (donor)			1600	nl head U/S	Survived

[a]Normal phenotype and normal karyotype.
Abbreviations: BPD: bronchopulmonary dysplasia; L: left; nl: normal; PIE: pulmonary interstitial emphysema; PVL: periventricular leukomalacia; R: right; RDS: respiratory distress syndrome; TTS: twin-to-twin transfusion syndrome; U/S: ultrasound; VATER: vertebral anomalies-anal atresia-tracheoesophageal fistula-renal defect.

Case 1. MoMo pregnancy uncomplicated until 22 weeks of gestation when one twin was diagnosed with body stalk anomally, whereas her sibling was normal. This pregnancy was monitored as in-patient until 29 weeks when intractable preterm labor started. Following antepartum steroids, an elective cesarean section was performed. Twin A whose Apgar scores were 9 and 9 had an uneventful hospital course. Twin B with a confirmed body stalk anomally without craniofacial defects expired 5 min after birth.

Case 2. Following the diagnosis of MoMo pregnancy, fetal echocardiography at 25 weeks showed one twin to be normal and the other to have hypoplastic left heart syndrome. As both twins continued to grow appropriately, this pregnancy was monitored as an outpatient until 34 weeks when an amniocentesis was performed for assessment of lung maturity. Shortly thereafter, the patient went into preterm labor, received steroids and was delivered by cesarean. Twin A whose Apgar scores were 7 and 8 developed respiratory distress and severe metabolic acidosis. She was intubated, given prostacyclin, placed on a mechanical ventilator and transferred to the Columbus Children's Hospital Heart Center. Following the diagnosis of aortic valve atresia, aberrant subclavian artery and ventricular septal defect, she underwent a stage I Norwood procedure and later a left diaphragmatic plication. She is doing well at four years of life.

Case 3. An uncomplicated MoMo pregnancy was monitored as an outpatient until 31 weeks when preterm labor occurred. Following steroid administration she delivered by cesarean. Twin A weighed 1490 g, had Apgar scores of 6 and 7 and developed mild respiratory distress. At 16 days of life, he became acutely ill, with abdominal distension and radiological signs of pneumatosis intestinalis. In the absence of intestinal perforation, he was managed with parenteral nutrition and multiple antibiotics. Oral feedings were restarted 14 days later and he was discharged in good health at 56 days of life.

Twin B whose Apgar scores were 6 and 7 developed mild respiratory distress. He was noted to have T4 and T10 hemivertebra, imperforated anus, rectoperineal fistula, normal external genitalia and bilateral dysplastic kidneys (vertebral anomalies-anal atresia-tracheoesophageal fistula-renal defect (VATER) sequence). He died of renal failure at 6 days of life.

Case 4. This MoMo pregnancy was complicated by limited prenatal care, tobacco and marijuana use and preterm labor at 27 weeks of gestation. She responded promptly to hospitalization, bed rest and antibiotics. At 30 weeks of gestation, a significant decrease in amniotic fluid and fetal distress indicated a cesarean delivery. Twin A weighed 1170 g and twin B 1040 g. Both twins developed respiratory distress, respiratory failure, received mechanical ventilation and exogenous surfactant. Owing to tracheal stenosis, their endotracheal intubations were difficult. Computerized axial tomography demonstrated hypoplasia of the left lung in Twin A and hypoplasia and lobar sequestration of the right lung in Twin B. Both infants developed pulmonary interstitial emphysema, bronchopulmonary dysplasia and irreversible respiratory failure.

Twin A died at 11 and Twin B at 31 days of life. Their phenotypes and karyotypes were normal.

Twin-to-twin transfusion syndrome

Case 5. This MoMo twin pregnancy was diagnosed at 22 weeks. Over the next 4 weeks, persistent polyhydramnios, fetal growth discordance, ascites and pleural effusions in the recipient twin suggested TTS. Serial amnioreductions were followed by cesarean delivery at 27 weeks. Birth weights were 870 and 618 g and umbilical cord Hgbs were 20 and 11 g/dl for the recipient and the donor, respectively. The donor received one blood transfusion. Both twins developed severe bronchopulmonary dysplasia and periventricular leukomalacia, but only one had retinopathy. After prolonged hospitalizations, both twins were discharged home.

Case 6. This MoMo twin pregnancy was diagnosed at 24 weeks. Twin-to-twin transfusion syndrome was suspected 2 weeks later owing to the presence of polyhydramnios, fetal growth discordance and abnormal Doppler studies in the intrauterine growth-restricted twin. Two amnioreductions provided some improvement, but the absence of end diastolic flow in the 'donor' prompted cesarean delivery at 27 weeks of gestation. Birth weights were 894 and 747 g and umbilical cord Hgbs were 19 and 11 g/dl, for the recipient and the 'donor', respectively. Both infants developed respiratory distress, received exogenous surfactant and total parenteral nutrition for a few days. The donor received one blood transfusion. Both infants had normal head ultrasounds, uneventful hospital courses and were discharged home at 45 days of life.

Case 7. MoMo pregnancy diagnosed at about 28 weeks of gestation. Polyhydramnios and growth discordance were noted at 32 weeks. Amniocentesis carried out for fetal lung maturity was followed by fetal distress, preterm labor and cesarean delivery. Birth weights were 1980 and 1610 g and umbilical cord Hgb were 18 and 12 g/dl, for the recipient and donor, respectively. The donor did not require blood transfusion. Both infants had normal head ultrasounds, uneventful hospital courses and were discharged home at 11 and 15 days of life, respectively.

Prematurity

Fifteen of the 36 (42%) women in this series started preterm labor between 24 and 34 weeks of gestation. Five patients (one who failed tocolysis at 24 weeks and four others who were at 32 to 34 weeks) were given steroids and promptly delivered. The 10 remaining women were successfully tocolysed and electively delivered at 32 to 34 weeks.

Excluding the stillbirth, five twins with congenital malformations and six twins with TTS, there were 60 neonates left whose problems were mainly related to prematurity (Table 3). One of the twins born at 26 weeks died of respiratory failure, but the remaining 59 survived. The sibling of the twin with VATER sequence and another twin, whose sibling was normal, developed necrotizing enterocolitis, but both recovered fully. Very low 1- and 5-min Apgar scores were noted in one infant born at 26 weeks GA and in another infant who had a true tight umbilical cord knot.

Table 3 Neonatal outcomes for monochorionic monoamniotic twins without congenital malformations or twin-to-twin transfusion syndrome

Infants no.	60
Gestational age (weeks)	32±2[a]
Birth weight (g) of 29 large twins	1780±335
Birth weight (g) of 28 small twins	1646±387
Cord hemoglobin (g/dl) of 29 large twins	16±3
Cord hemoglobin (g/dl) of 28 small twins	15±3
1-min Apgar ⩽3 no. (%)	2 (3)
5-min Apgar ⩽3 no. (%)	2 (3)
Infants on mechanical ventilation no. (%)	23 (38)
Hospital stay (days)	25±17
Neonatal death no. (%)	1 (2)
Infants with head ultrasound no. (%)	55 (92)
Head ultrasound normal no. (%)	50 (91)
Grade I–II intracranial hemorrhage no. (%)	5 (9)

[a]Mean±s.d.

Twenty-three of the 60 (38%) infants in this subgroup had respiratory distress requiring mechanical ventilation and treatment with exogenous surfactant. All except one of these infants were extubated in less than 1 week. Only one twin born at 28 weeks of GA developed severe bronchopulmonary dysplasia and was discharged home on oxygen at 37 weeks of postconceptional age.

Paired umbilical cord Hgbs were available from 32 of the 36 sets of twins. Cord Hgb differences of ⩾5 gm/dl were noted in the three sets affected by TTS, in the set with congenital heart disease and in five sets of otherwise uncomplicated premature twins. Anemia requiring transfusion was seen in two of the three donor twins and in one twin described above who had entanglement and tight umbilical cord knot.

Discussion

Monoamniotic placentation is the result of late splitting of the developing embryo around 8 to 9 days after fertilization.[3,14,15] MoMo pregnancies although rare (about one in every 10 000 pregnancies) have been reported to have a perinatal mortality as high as 68%.[16,17] Traditionally, these high perinatal losses have been attributed to umbilical cord entanglements, congenital malformations, TTS and prematurity.[4,5,16,17] Before 1990, the antenatal diagnosis of MoMo pregnancies was made in not more than half of the patients and for whom the majority of the deliveries were vaginal.[4] Recent reviews of the literature indicate

significant reductions in perinatal mortality, 40 to 60% in 1961 to 1962, 28% in 1961 to 1982, 20% in 1997 to 1999 and 12% in 2001 to 2004.[4,5,16,19] Factors that may explain these changes in perinatal mortality are antenatal diagnosis of twinning, recognition of MoMo placentation, close fetal surveillance, cesarean delivery and advances in neonatal care.

Cord entanglement, a condition unique to MoMo pregnancies, occurs in 42 to 80% of the cases and it has been traditionally related to high perinatal mortality.[3,14–19] Although entanglement has been blamed for many antepartum losses, the majority of the fetuses involved in this and in other studies did not experience fatal cord accidents.[20] There have been reports of successful vaginal deliveries with entangled cords and with true umbilical cord knots.[21] How often entanglement associates with true umbilical cord knots is unclear. In our series, we observed true knots in five of 15 entangled cords, an incidence similar to that of Bajoria *et al*.[20] (eight of 13), Rodis *et al*.[16] (five of six) and Suzuki *et al*.[22] (three of nine). Although entanglement with true knots represents a significant risk, actual umbilical cord vascular accidents may be less common than anticipated. The risk that a tightening knot may compromise the umbilical circulation may be ameliorated by the low friction surfaces of the cords, the cushioning effect of Wharton's jelly and the resistance of the vessels to compression.[14]

Congenital malformations are associated with high perinatal mortality in premature singletons as well as in twins.[23] Analysis of a large database from nine European and Latin American registries confirmed that malformations are more common in twins than in singletons and that all anatomical sites could be involved.[24] Malformations are more frequent in monozygotic than dizygotic twins and they are 2.5-fold higher in monochorionic than in dichorionic placentations.[23] Although no specific malformation or group of malformations are unique to multiples, recent data suggest that lung hypoplasia may occur more often in twins.[23,24] What makes our cases peculiar is its occurrence in both twins as well as the different locations of the hypoplastic lung.

Some malformations may be recognized antenatally (i.e. body stalk anomaly, congenital heart defect), whereas others may not (i.e. VATER sequence, lung hypoplasia). Like in the cases reported here, most malformations affect only one twin and the majority are not associated with chromosomal abnormalities.[23,24] Body stalk anomaly is a rare and uniformly fatal malformation known to affect singletons as well as multiples.[25] This condition can be diagnosed early in pregnancy because of the large thoracoabdominal anterior body wall defect, small thorax, protruding heart, liver and other viscera. Differential diagnosis should include other anterior wall defects such as gastroschisis or amniotic band sequence. The absence of craniofacial malformations and normal chromosomes in body stalk anomaly has been reported before. Although selective feticide of affected twins has been proposed, our case supports expectant management.

Twin-to-twin transfusion syndrome is not as common among MoMo as is in MoDi pregnancies because MoMo placentas have more arterioarterial and fewer deep arteriovenous anastomoses than MoDi placentas.[8,20] We report TTS in three of the 36 MoMo pregnancies with an incidence of 10%, quite similar to that noted by others.[5,16,19,21,22]

The presence of polyhydramnios, discordant fetal growth, hydrops, congestive heart failure, tricuspid regurgitation and discordant bladder fillings make the prenatal diagnosis of TTS possible.[8–10] Postnatal angiograms of the placenta demonstrating deep arteriovenous anastomoses with cotyledon sharing may confirm the diagnosis.[15] Sebire *et al*.[26] suggested that acute TTS may be responsible for antepartum fetal demise in MoMo twins, speculating that imbalances between the two fetal circulations cannot be sustained for any prolonged time. The differences in cord Hgbs $\geqslant 5$ g/dl in cases other than those three TTS pregnancies suggests that acute transfer of fetal blood from one twin to the other at the time of delivery is possible.

It is known that uncomplicated twin pregnancies have a higher incidence of premature birth than singletons and that MoMo twins are at an even greater risk of being born before 32 weeks of gestation.[1,2,7] Our data showed that preterm labor occurred in one-half of all MoMo pregnancies. In these cases efforts were made to control it, allowing time for steroid administration and to determine the most favorable time for delivery.

Concerns about entanglement of the cord, late fetal death and risks for the surviving twin are some of the considerations involved in timing for delivery. Although optimal time is still controversial, most management strategies including ours favor delivery no later than 32 to 34 weeks of gestation.[17–19] Premature infants, especially those born before 32 weeks of gestation, have a high incidence of perinatal depression, respiratory distress, early- and late-onset sepsis, patent ductus arteriosus, necrotizing enterocolitis, intracranial hemorrhage, prolonged hospitalization and poor neurological outcomes.[27] Data provided here (Table 3) highlight low mortality, but still document the significant morbidities associated with premature deliveries.

During the last 20 years, most clinicians have opted for cesarean delivery in MoMo pregnancies.[17–19] The absence of umbilical cord accidents and the low incidence of perinatal depression among our twins may be a positive reflection of the mode of delivery.

In summary, our study documents that MoMo twins remain a group at risk for cord entanglement, congenital malformations, TTS and prematurity. Although their perinatal mortality and morbidity remain high, current fetal and neonatal management resulted in vastly improved outcomes. Our findings provide updated information for counseling parents with MoMo pregnancies and may be helpful for planning fetal surveillance and neonatal follow-up.

References

1 Martin JA, Hamilton BE, Sutton PD, Ventura SJ, Menacker F, Munson M. Birth: final data for 2002. *Natl Vital Stat Rep* 2003; **52**: 1–27.
2 Powers WF, Kiely JL. The risks confronting twins: a national perspective. *Am J Obstet Gynecol* 1994; **170**: 456–461.
3 Benirschke K. The biology of the twinning process: how placentation influences outcome. *Semin Perinatol* 1995; **19**: 342–350.
4 Carr SR, Aronson MP, Coustan DR. Survival rates of monoamniotic twins do not decrease after 30 weeks' gestation. *Am J Obstet Gynecol* 1990; **163**: 719–722.
5 Tessen JA, Zlatnik FJ. Monoamniotic twins: a retrospective controlled study. *Obstet Gynecol* 1991; **77**: 832–834.
6 Victoria A, Mora G, Arias F. Perinatal outcome, placental pathology, and severity of discordance in monochorionic and dichorionic twins. *Obstet Gynecol* 2001; **97**: 310–315.
7 Dube J, Dodds L, Armson BA. Does chorionicity or zygosity predict adverse perinatal outcomes in twins? *Am J Obstet Gynecol* 2002; **186**: 579–583.
8 Umur A, Van Gemert M, Nikkels P. Monoamniotic-versus diamniotic-monochorionic twin placentas: anastomoses and twin–twin transfusion syndrome. *Am J Obstet Gynecol* 2003; **189**: 1325–1329.
9 Gallot D, Saulnier JR, Savary D, Laurichesse-Delmas H, Lemery D. Ultrasonographic signs of twin-twin transfusion syndrome in a monoamniotic twin pregnancy. *Ultrasound Obstet Gynecol* 2005; **25**: 307–311.
10 Quintero RA, Morales WJ, Allen MH, Bornick PW, Johnson PK, Kruger M. Staging of twin–twin transfusion syndrome. *J Perinatol* 1999; **19**: 550–555.
11 Harman CR. Assessment of fetal health. In: Creasy RK, Resnik R, Iams JD (eds). *Maternal–Fetal Medicine: Principles and Practice*. Saunders: Philadelphia, PA, 2004, pp 357–401.
12 Ananth CV, Vintzileo AM, Shen-Schwarz S, Smulian JC, Lai YL. Standards of birth weight in twin gestations stratified by placental chorionicity. *Obstet Gynecol* 1998; **91**: 917–924.
13 Papile LA, Burstein J, Burstein R, Koffler H. Incidence and evolution of the subependymal intraventricular hemorrhage: a study of infants with weights less than 1500 g. *J Pediatr* 1978; **92**: 529–534.
14 Baldwin V. The pathology of monochorionic monozygosity. In: Baldwin V (ed). *Pathology of Multiple Pregnancy*. Springer-Verlag: New York, 1994, pp 199–213.
15 Bilardo CM, Arabin B. Monoamniotic twins. In: Blickstein I, Keith LG (eds). *Multiple Pregnancy*. Taylor & Francis: London and New York, 2005, pp 574–582.
16 Rodis JF, McIlveen PF, Eagen JF, Borgida AF, Turner GW, Campbell WA. Monoamniotic twins: improved perinatal survival with accurate prenatal diagnosis and antenatal fetal surveillance. *Am J Obstet Gynecol* 1997; **177**: 1046–1049.
17 Allen VM, Windrim R, Barrett J, Ohlsson A. Management of monoamniotic twin pregnancies: a case series and systematic review of the literature. *Br J Obstet Gynecol* 2001; **108**: 931–936.
18 Roque H, Gillen-Goldstein J, Funai E, Young BK, Lockwood CJ. Perinatal outcomes in monoamniotic gestations. *J Matern-Fetal Neonat Med* 2003; **13**: 414–421.
19 Ezra Y, Shveiky D, Ophir E, Nadjari M, Eisenberg VH, Samueloff A *et al*. Intensive management and early delivery reduce antenatal mortality in monoamniotic twin pregnancies. *Acta Obstet Gynecol Scand* 2005; **84**: 432–435.
20 Bajoria R. Vascular anatomy of monochorionic placenta in relation to discordant growth and amniotic fluid volume. *Hum Reprod* 1998; **13**: 2933–2940.
21 Demaria F, Goffinet F, Kayem G, Tsatsaris V, Hessabi M, Cabrol D. Monoamniotic twin pregnancies: Antenatal management and perinatal results of 19 consecutive cases. *Int J Obstet Gynecol* 2004; **111**: 22–26.
22 Suzuki S, Kaneko K, Shin S, Araki T. Incidence of intrauterine complications in monoamniotic twin gestation. *Arch Gynecol Obstet* 2001; **265**: 57–59.
23 Ananth CV, Smulian JC. Trends in congenital malformations, chromosomal anomalies and infant mortality among twin birth. In: Blickstein I, Keith LG (eds). *Multiple Pregnancy*. Taylor & Francis: London and New York, 2005, pp 246–251.
24 Mastroiacovo P, Castilla EE, Arpino C, Botting B, Cocchi G, Goujard J *et al*. Congenital malformations in twins: an international study. *Am J Med Genet* 1999; **83**: 117–124.
25 Smrcek JM, Germer U, Krokowski M, Berg C, Krapp M, Geipel A *et al*. Prenatal ultrasound diagnosis and management of body stalk anomaly: analysis of 9 singleton and 2 multiple pregnancies. *Ultrasound Obstet Gynecol* 2003; **21**: 322–328.
26 Sebire NJ, Souka A, Skentou H, Geerts L, Nicolaides KH. First trimester diagnosis of monoamniotic twin pregnancies. *Ultrasound Obstet Gynecol* 2000; **16**: 223–225.
27 McIntire D, Bloom SL, Casey BM, Leveno KJ. birth weight in relation to morbidity and mortality among newborn infants. *N Engl J Med* 1999; **340**: 1234–1238.