Editor's choice
Scan to access more
free content

Macrosomic newborns of non-diabetic mothers: anthropometric measurements and neonatal complications

N Linder,^{1,2} Y Lahat,¹ A Kogan,¹ E Fridman,¹ F Kouadio,¹ N Melamed,^{2,3} Y Yogeve,^{2,3} G Klinger^{2,4}

¹Department of Neonatology, Helen Schneider Hospital for Women, Rabin Medical Center, Petach Tikva, Israel

²Sackler School of Medicine, Tel Aviv University, Tel Aviv, Israel

³Departments of Obstetrics and Gynecology, Helen Schneider Hospital for Women, Rabin Medical Center, Petach Tikva, Israel

⁴Neonatal Intensive Care Unit, Schneider Children's Medical Center of Israel, Petach Tikva, Israel

Correspondence to

Professor Nehama Linder, Department of Neonatology, Rabin Medical Center, Beilinson Hospital, Petach Tikva 49100, Israel; nehama.linder@gmail.com

Received 7 August 2013

Revised 3 April 2014

Accepted 4 April 2014

Published Online First

3 May 2014

► <http://dx.doi.org/10.1136/fetalneonatal-2013-305894>

CrossMark

To cite: Linder N, Lahat Y, Kogan A, et al. *Arch Dis Child Fetal Neonatal Ed* 2014;**99**:F353–F358.

ABSTRACT

Objective To assess the association of anthropometric measurements with neonatal complications in macrosomic newborns of non-diabetic mothers.

Design Retrospective cohort study.

Patients All liveborn, singleton, full term newborns with birth weight ≥ 4000 g born to non-diabetic mothers at a tertiary medical centre in 1995–2005 (n=2766, study group) were matched to the next born, healthy, full term infant with a birth weight of 3000–4000 g (control group). Exclusion criteria were multiple birth, congenital infection, major malformations and pregnancy complications.

Intervention Data collection by file review.

Outcome measures Complication rates were compared between study and control groups and between symmetric and asymmetric macrosomic newborns, defined by weight/length ratio (WLR), Body Mass Index and Ponderal Index.

Results The 2766 non-diabetic macrosomic infants identified were matched to 2766 control infants. The macrosomic group had higher rates of hypoglycaemia (1.2% vs 0.5%, $p=0.008$), transient tachypnoea of the newborn (1.5% vs 0.5%, $p<0.001$), hyperthermia (0.6% vs 0.1%, $p=0.012$), and birth trauma (2% vs 0.7%, $p<0.001$), with no cases of symptomatic polycythaemia, and only one case of hypoglycaemia. Hypoglycaemia was positively associated with birth weight. It was significantly higher in the asymmetric than the symmetric macrosomic newborns, defined by WLR (1.7% vs 0.3%, $p<0.001$).

Conclusions Macrosomic infants of non-diabetic mothers are at increased risk of neonatal complications. However, routine measurements of haematocrit and calcium may not be necessary. Symmetric macrosomic infants (by WLR) have a similar rate of hypoglycaemia as normal-weight infants. Thus, repeat glucose measurements in symmetric macrosomic infants are not justified.

INTRODUCTION

There is no consensus regarding the definition of macrosomia, and the term may be applied to newborns with a birth weight greater than 4000, 4500, or 5000 g, irrespective of gestational age.¹ The American College of Obstetricians and Gynaecologists suggests a threshold of 4500 g, as morbidity increases sharply beyond this birth weight.²

The prevalence of macrosomia has been rising in recent decades, with an accompanying increased risk of adverse outcomes for mother and newborn. Risks to the newborn include shoulder dystocia,³

What is already known on this topic

- The prevalence of macrosomia has been rising, with an accompanying increased risk of adverse outcomes.
- Macrosomic newborns are at an increased risk of hypoglycaemia, which is positively associated with birth weight.

What this study adds

- Symptomatic hypocalcaemia and polycythaemia are rare in macrosomic infants of non-diabetic mothers; routine measurements of haematocrit and calcium may not be required.
- Symmetric macrosomic infants (defined by weight/length ratio) have a similar rate of hypoglycaemia as normal weight infants.
- Repeated glucose measurements in symmetric macrosomic infants are not justified.

hypoxia, plexus injuries,⁴ hypoglycaemia, congenital anomalies, and need for intensive care.^{5 6}

The aim of the present study was to quantify the rates of neonatal complications in macrosomic newborns born to non-diabetic mothers; compare these rates between symmetric and asymmetric macrosomic subgroups, defined according to anthropometric indices, and explore possible correlations of neonatal morbidities with anthropometric measurements in macrosomic subgroups.

MATERIALS AND METHODS

Patients and setting

This study was approved by the local institutional research review board. A retrospective cohort study design was used. The computerised discharge records database of Rabin Medical Center, a tertiary university-affiliated hospital, was searched for all liveborn, singleton, full term (37–41 completed gestational weeks) neonates with a birth weight of ≥ 4000 g who were born between January 1995 and December 2005. Exclusion criteria were pregnancy complicated by multiple births, maternal diabetes (one or more abnormal glucose levels),⁷ maternal hypertension and/or toxemia, placental abruption, amnionitis, acute and/or chronic fetal

distress, or impaired fetal growth, and presence of neonatal congenital infection, major congenital malformations, chromosomal aberrations, or congenital syndromes. For each infant in the study group, the next born appropriate-for-gestational age (AGA) (± 1 week) singleton infant with a birth weight of 3000–4000 g was selected for the control group.

Data collection

Maternal and neonatal data were extracted from the medical records of the High-Risk Pregnancy Unit, Delivery Room, and Neonatal Department, as follows: maternal demographic and obstetrical characteristics: age, parity, mode of delivery, date and hour of birth (with risk hours defined as 1600 to 0800 and weekends); neonatal characteristics: gestational age, birth weight, length, head circumference, sex, perinatal mortality, number of days in the neonatal intensive care unit (NICU), hospital length of stay, 5 min Apgar score, umbilical cord blood pH, blood pressure, neurological disorders (hypoxic ischaemic encephalopathy, convulsions), related birth injuries, hypoglycaemia, hypocalcaemia, unexplained hyperbilirubinemia, metabolic acidosis, polycythaemia, meconium aspiration syndrome, respiratory morbidity (including transient tachypnoea of the newborn, respiratory distress syndrome, and the need for oxygen administration or mechanical ventilation), hyperthermia, cyanotic episodes, culture results, neonatal leukocytosis (white blood cell count $\geq 30\,000/\text{mm}^3$), leukopenia (white blood cell count $\leq 5000/\text{mm}^3$) and thrombocytopenia (platelet count $\leq 150\,000/\text{mm}^3$), and loss of $>10\%$ of birth weight.

Measurements and definitions

Weight and length were measured by standard paediatric procedures.^{8–9} Birth weight is routinely measured by trained nurses within 1 h of delivery using electronic scales accurate to 5 g and calibrated before each measurement. Crown-heel length is measured at the same time to the nearest millimeter with an infantometer, with the head placed against the head plate and the knees fully extended.

Weight/length ratio (WLR) (weight/length, kg/m), Body Mass Index (BMI) (weight/length/m²) and Ponderal Index (PI) (weight/length³, g/cm³*100) were calculated for all infants. Asymmetry was defined as an index value above the 90th percentile.^{10–15}

Macrosomia was defined as birth weight ≥ 4000 g irrespective of gestational age.¹⁶ AGA was defined as birth weight between the 10th and 90th percentile.¹⁷ Hypoglycaemia was defined as blood glucose <40 mg/dL (2.2 mmol/L) at any period during the hospital stay, irrespective of gestational age or hour of life.¹⁸ Hyperthermia was defined as temperature $>37.6^\circ\text{C}$, persisting >1 h. Unexplained hyperbilirubinemia was defined as any serum bilirubin value >10 mg/dL (170 mmol/L) before 24 h of age and/or >13 mg/dL after 24 h of age, in the absence of active haemolysis, blood group incompatibility, glucose 6-phosphatase deficiency, or other identifiable cause of hyperbilirubinemia, such as sepsis.¹⁹ Polycythaemia was defined as peripheral venous haematocrit >0.65 (65%).²⁰ Hypoxic ischaemic encephalopathy was defined as a capillary or arterial pH <7.00 and/or base deficit <-16 . Hypocalcaemia was defined as calcium <7.5 mg/dL. Prolonged hospitalisation was defined as an admission for more than 5 days for neonates delivered by caesarean section, and 3 days for vaginal delivery. The composite outcome was defined as the presence of one or more of the following morbidities: 5 min Apgar score <7 , hypoxic ischaemic encephalopathy, convulsions, birth injury, hypoglycaemia, hypocalcaemia, unexplained hyperbilirubinemia, metabolic

acidosis, polycythaemia, meconium aspiration syndrome, transient tachypnoea of the newborn or respiratory distress syndrome, hyperthermia, cyanotic episodes, or loss of $>10\%$ of birth weight.

Departmental policy for macrosomic and non-macrosomic infants

During the study period, it was the departmental policy to routinely measure blood glucose in all macrosomic infants (≥ 4000 g) at 1 h of age, followed by early feeding. Repeated measurements were performed at ages 3, 6, 8 and 24 h. In the control group, glucose was determined in every infant requiring blood test. The majority of these infants were asymptomatic screened for infection due to maternal fever, premature rupture of membranes and so on. However, some of these infants were screened to exclude symptomatic hypoglycaemia.

The department policy is to screen for sepsis at 3–6 h of age, which is similar to the schedule of glucose screening for the macrosomic infants. Newborns found to be hypoglycaemic (glucose <40 mg%) underwent repeated glucose measurement within the hour of the previous measurement. Intravenous glucose was administered to all infants with a glucose level <40 mg% at 1 h after feeding. All infants were followed for a minimum period of 24 h. Infants were screened for polycythaemia at 3 h of age and for hypocalcaemia at 24 h of age.

Glucose screening is performed in non-macrosomic infants only when symptomatic hypoglycaemia is suspected. However, a glucose test is routinely done if blood is drawn for any indication, most commonly to exclude sepsis in infants at risk for sepsis. Repeat blood testing for hypoglycaemic non-macrosomic infants is done similarly to that performed for macrosomic infants.

Statistical analysis

Data were analysed with BMDP Statistical Software.²¹ Univariate analysis was performed to identify differences between two groups. Student t test, Pearson's χ^2 test, and Fisher's exact test were applied as suitable to compare groups. Statistical significance was defined as $p < 0.05$.

RESULTS

Of the 79 031 neonates born in our medical centre during the study period, 60 574 singleton full-term infants were identified and available for analysis (figure 1). They included 2766 macrosomic newborns of non-diabetic mothers who met the inclusion criteria. This group was matched to 2766 control infants. The clinical characteristics of the study and control groups are described in table 1. Median birth weight was 4150 g (range 4000–5370) in the macrosomic group, and 3400 g (range 3000–3995) in the control group. The macrosomic group had a significantly higher percentage of male infants than the control group (67.9% vs 53.2%, $p < 0.001$) and more caesarean deliveries. However, in the macrosomic LGA group vs the control group, there was no difference in gender. Table 2 describes the outcome of the two groups. The rate of prolonged hospitalisation was significantly higher in the macrosomic vs the control infants (OR 2.22, 95% CI 1.96 to 2.51). No difference in the percent of neonates with prolonged hospitalisation was found comparing neonates born by vaginal delivery without complications to all macrosomic infants (4.5% vs 4.7%, $p = 0.8$). A significantly higher number of macrosomic vs control infants were transferred to the NICU (OR 1.78, 95% CI 1.27 to 2.53). The macrosomic versus control group also had significantly more infants with one or more neonatal complications (composite

* Some of the newborns have more than one exclusion criteria

Figure 1 Study group selection.

outcome, 11.7% vs 8.0%, $p < 0.001$) (OR 1.53, 95% CI 1.27 to 1.83). The main complications were hypoglycaemia (OR 2.37, 95% CI 1.23 to 4.81), transient tachypnoea of the newborn (OR 2.83, 95% CI 1.53 to 5.50), hyperthermia, and birth trauma (OR 3.0, 95% CI 1.78 to 5.62) (table 2). There were no between-group differences in rates of low 5 min Apgar score, metabolic acidosis, infection, loss of >10% of body weight, meconium aspiration syndrome, or cyanotic episodes. No infant had symptomatic polycythaemia, and only one had hypocalcaemia.

The composite outcome was calculated for the 325 macrosomic infants with neonatal complications. They did not differ in length or head circumference from the remaining 2441 macrosomic infants, but they had significantly higher birth weights (4232 g vs 4194 g, $p < 0.002$), WLRs (8.07 vs 7.99, $p < 0.001$), BMIs (15.39 vs 15.24, $p = 0.004$), and PI's (2.94 vs 2.91, $p < 0.03$). Among the macrosomic infants with complications,

there was no statistically significant difference in composite score between those who were transferred to the NICU ($n = 100$) and those who were not.

The rate of asymmetry within the macrosomic group (defined as an anthropometric index value above the 90th percentile²²) was 63.2% by WLR, 42.4% by BMI and 22.6% by PI. Regardless of the measure used, infants defined as asymmetric were more likely to be born by caesarean delivery and less likely to be born during high-risk hours. The characteristics and outcomes of symmetric and asymmetric macrosomic infants by WLR are presented in tables 3 and 4.

The incidence of hypoglycaemia in the macrosomic infants was 1.2%, and was positively associated with birth weight (table 5); 42% of the control infants were screened at least once for hypoglycaemia. Of these, only 2.2% were screened to exclude symptomatic hypoglycaemia. The prevalence of hypoglycaemia using as the denominator only neonates who underwent glucose testing showed that 14 infants (1.2%) of the control group had hypoglycaemia compared to 33 infant (1.2%) of the macrosomic infants ($p > 0.9$). Similarly compared, more macrosomic LGA infants had hypoglycaemia (29 infants, 2.15%) vs tested control group infants (14 infants, 1.2%) ($p = 0.07$). Further analysis showed that it was the asymmetric infants within the macrosomic group that accounted for most of the increased rate of hypoglycaemia (table 4). The rate of hypoglycaemia in infants with symmetric macrosomia by WLR was similar to that of the control infants (table 5).

DISCUSSION

This study evaluated the rate of neonatal complications in macrosomic newborns of non-diabetic mothers and the association of symmetry/asymmetry with the risk of neonatal complications. Macrosomic newborns are considered to be at an increased risk of hypoglycaemia, however, maternal diabetes is the underlying cause in most cases. Therefore, we limited the study group to infants of non-diabetic mothers to isolate the possible effect of birth weight. The results showed that macrosomic infants, compared to control infants, have higher rates of hypoglycaemia, transient tachypnoea of the newborn, hyperthermia, and birth trauma.

Table 1 Clinical characteristics of macrosomic and control groups

Clinical characteristics	Control group (n=2766)	Macrosomic group (n=2766)	Macrosomic LGA (n=1347)
Median birth weight, g, (range)	3400 (3000–3995)	4150 (4000–5370)	4270 (4000–5370)
Mean maternal age, years (SD)	29.61 (5.0)	30.66 (5.08)	31.09 (5.02)
Birth parity, median (range)	2.0 (1–12)	2.0 (1–17)	2.0 (1–16)
Mean head circumference, cm (SD)	34.61 (1.05)	35.9 (1.02)	35.98 (1.03)
Mean length, cm (SD)	50.4 (1.3)	52.5 (1.42)	52.67 (1.43)
AGA according to length, n (%)	2766 (100)	1786 (64.6)	684 (50.8)
AGA according to weight, n (%)	2766 (100)	1419 (51.3)	0 (0)
Mean WLR, kg/m (SD)	6.78 (0.44)	8.0 (0.35)	8.18 (0.35)
Mean BMI, kg/m ² (SD)	13.46 (0.89)	15.25 (0.91)	15.54 (0.88)
Mean PI, g/cm ³ ×100 (SD)	2.67 (0.2)	2.91 (0.24)	2.96 (0.23)
Male, n (%)	1472 (53.2)	1877 (67.9)	736 (54.6)*
Vaginal delivery, n (%)	2160 (78.1)	1728 (62.5)	737 (54.7)
Caesarean delivery, n (%)	406 (14.7)	899 (32.5)	561 (41.6)
Instrumental delivery, n (%)	200 (7.2)	139 (5)	49 (3.6)
Birth in high-risk time period, n (%)	2090 (75.7)	1822 (71.6)	851 (68.5)

When comparing the control group to the macrosomic group all p values <0.001. When comparing the control group to the LGA macrosomic group all p values <0.001 except

*p value=0.4 comparing % males between LGA to control group.

AGA, appropriateness for gestational age; BMI, Body Mass Index; LGA, large for gestational age; PI, Ponderal Index; WLR, weight/length ratio.

Table 2 Outcome of macrosomic and control groups

Outcome parameters	Macrosomic group (n=2766)	Control group (n=2766)	p Values*	Macrosomic LGA (n=1347)	p Values†
NICU transfer, n (%)	100 (3.6)	57 (2.1)	<0.001	53 (3.9)	<0.001
Prolonged hospitalisation, n (%)	959 (34.7)	534 (19.3)	<0.001	579 (43)	<0.001
Composite outcome, n (%)‡	325 (11.7)	222 (8.0)	<0.001	185 (13.7)	<0.001
Infants with any birth trauma, n (%)	55 (2)	18 (0.7)	<0.001	28 (2.1)	<0.001
Fracture of clavicle, n (%)	51 (1.8)	15 (0.5)	<0.001	26 (1.9)	<0.001
Respiratory disorders, n (%)	42 (1.5)	15 (0.5)	<0.001	20 (1.5)	0.003
Hypoglycaemia, n (%)	33 (1.2)	14 (0.5)	0.008	29 (2.2)	<0.001
Hyperthermia, n (%)	16 (0.6)	4 (0.1)	0.012	6 (0.4)	0.09
Unexplained jaundice, n (%)	149 (5.4)	132 (4.8)	0.33	90 (6.7)	0.012
Hypoxic ischaemic encephalopathy, n (%)	16 (0.6)	8 (0.3)	0.15	8 (0.6)	0.18
Polycytemia, n (%)	11 (0.4)	6 (0.2)	0.33	7 (0.5)	0.14
Cyanotic event, n (%)	9 (0.3)	8 (0.3)	0.81	6 (0.4)	0.41
Culture-positive sepsis, n (%)	8 (0.3)	6 (0.2)	0.79	5 (0.4)	0.35
Leukocytosis, n (%)	7 (0.3)	1 (0)	0.07	3 (0.2)	0.1
Weight loss >10%, n (%)	6 (0.2)	9 (0.3)	0.61	5 (0.4)	0.78
Thrombocytopenia, n (%)	5 (0.2)	4 (0.1)	0.75	4 (0.3)	0.45
Hypocalcaemia, n (%)	1 (0)	0	0.98	1 (0.1)	0.33
Meconium aspiration, n (%)	1 (0)	0	0.98	0	0.98

*p Values between macrosomic and control groups

†p Values between macrosomic LGA and control groups.

‡Defined as one or more of the following: 5 min Apgar score <7, asphyxia, convulsions, birth injury, hypoglycaemia, hypocalcaemia, unexplained hyperbilirubinemia, metabolic acidosis, polycythaemia, meconium aspiration syndrome, transient tachypnoea of the newborn or respiratory distress syndrome, hyperthermia, cyanotic episodes or loss of >10% of birth weight. LGA, large for gestational age; NICU, neonatal intensive care unit.

The few studies that have assessed the rate of hypoglycaemia in macrosomic newborns of non-diabetic mothers reported rates of 3.8–16%.^{23–26} The wide range was mainly due to differences in the definition of hypoglycaemia, which is still controversial. We selected a definition that provided us a safety margin for prevention and treatment. We sought to maintain plasma glucose concentrations in asymptomatic infants at >40 mg/dL.²⁷ Accordingly, the rate of hypoglycaemia in our study group was 1.2%, which is far lower than previously reported in macrosomic infants.^{23–26} The rate of hypoglycaemia was positively associated with birth weight, ranging from 0.8% in infants with a birth weight of 4000–4499 g to 25% in infants with a birth weight of ≥5000 g.

The macrosomic group was defined by three anthropometric measures. The type of measure used influenced the results, with rates of asymmetry varying from 63.2% when macrosomia was

defined by WLR, to 42.4% when BMI was used, to 22.6% when PI was used. Several similar studies to ours have been conducted, but the different anthropometric parameters applied led to inconsistent results.^{25 28–33} For example, using PI, Ballard *et al*²⁸ reported an asymmetry rate of 12.5%, which is considerably lower than our 22.6%. Persson *et al*^{29 30} reported a rate of 35%, however, infants of diabetic mothers were included in this study.

We found that the tool with the most highly significant results for identifying macrosomic infants at risk for hypoglycaemia was WLR,²⁴ however, BMI was also significant, but less so, while PI was not found to be significant, by contrast with others.^{31–33} The use of WLR, is a simpler concept than BMI or PI, and it is less prone to error because the length measurement is not squared or cubed. Characterisation of the macrosomic infants by low risk (symmetric macrosomia) or high risk

Table 3 Comparison of clinical characteristics of symmetric and asymmetric macrosomic infants by WLR

Clinical characteristics	Symmetric by WLR (n=1020)	Asymmetric by WLR (n=1746)	p Value
Mean maternal age, years (SD)	30.38 (5.0)	30.82 (5.12)	0.027
Birth weight, g, median (range)	4080 (4000–4500)	4210 (4000–5370)	<0.001
Mean head circumference, cm (SD)	35.88 (1.01)	35.92 (1.02)	0.29
Mean length, cm (SD)	53.1 (1.16)	52.16 (1.45)	<0.001
AGA according to length, n (%)	614 (60.2)	1172 (67.1)	<0.001
AGA according to weight, n (%)	923 (90.5)	496 (28.4)	<0.001
Mean WLR, kg/m (SD)	7.73 (0.17)	8.16 (0.32)	<0.001
Male, n (%)	855 (83.8)	1022 (58.5)	<0.001
Vaginal delivery, n (%)	736 (72.2)	992 (56.8)	<0.001
Caesarean delivery, n (%)	213 (20.9)	686 (39.3)	<0.001
Instrumental delivery, n (%)	71 (7)	68 (3.9)	<0.001
Birth during high-risk time period, n (%)	711 (75.4)	1111 (69.4)	0.001

AGA, appropriateness for gestational age; WLR, weight/length ratio.

Table 4 Comparison of outcomes of symmetric and asymmetric macrosomic infants by WLR

Outcome parameters	Symmetric by WLR (n=1020)	Asymmetric by WLR (n=1746)	p Values
Composite outcome, n (%) [*]	94 (9.2)	231 (13.2)	0.001
Unexplained jaundice, n (%)	40 (3.9)	109 (6.2)	0.009
Hypoglycaemia, n (%)	3 (0.3)	30 (1.7)	<0.001
NICU transfer, n (%)	29 (2.8)	71 (4.1)	0.11
Prolonged hospitalisation, n (%)	247 (24.2)	712 (40.8)	<0.001
Respiratory disorders, n (%)	14 (1.4)	28 (1.6)	0.75
Any birth trauma, n (%)	15 (1.5)	40 (2.3)	0.16
Fracture of clavicle, n (%)	15 (1.5)	36 (2.1)	0.31
Hyperthermia, n (%)	6 (0.6)	10 (0.6)	0.99
Hypoxic ischaemic encephalopathy, n (%)	5 (0.5)	11 (0.6)	0.8
Culture-positive sepsis, n (%)	3 (0.3)	5 (0.3)	0.97
Leukocytosis, n (%)	3 (0.3)	4 (0.2)	0.71
Polycythaemia, n (%)	2 (0.2)	9 (0.5)	0.35
Thrombocytopenia, n (%)	2 (0.2)	3 (0.25)	0.98
Cyanotic event, n (%)	2 (0.2)	7 (0.4)	0.5
Hypocalcaemia, n (%)	1 (0.1)	0	0.37
Weight loss >10%, n (%)	0	6 (0.3)	0.09
Meconium aspiration, n (%)	0	1 (0.1)	0.79

^{*}Defined as one or more of the following: 5 min Apgar score <7, hypoxic ischaemic encephalopathy, convulsions, birth injury, hypoglycaemia, hypocalcaemia, unexplained hyperbilirubinemia, metabolic acidosis, polycythaemia, meconium aspiration syndrome, transient tachypnoea of the newborn or respiratory distress syndrome, hyperthermia, cyanotic episodes or loss of >10% of birth weight.
WLR, weight/length ratio; NICU, neonatal intensive care unit.

(asymmetric macrosomia) revealed a significantly higher rate of hypoglycaemia in the asymmetric macrosomic subgroup. Indeed, symmetric macrosomic newborns of non-diabetic mothers, as defined by WLR, had a similar hypoglycaemic rate compared to control infants (table 5). These findings suggest that repeated glucose testing of symmetric macrosomic newborns (defined by WLR) of non-diabetic mothers is not justified. Our results differ from those of Schaefer-Graf *et al*³⁴ who failed to find a correlation between anthropometric parameters and hypoglycaemia, possibly due to an inaccuracy in neonatal length measurement. Previous studies advocated the use of symmetric versus asymmetric growth to assess the risk of hypoglycaemia or other neonatal complications. Ballard *et al*²⁸ and Djelmis *et al*³⁵ studied newborns of diabetic mothers and suggested that hyperglycaemia in utero causes a disproportion in growth, and when this occurs, more neonatal complications are observed, such as hypoglycaemia, hyperbilirubinemia, polycythaemia and metabolic acidosis.^{28 35} This hypothesis was supported by Van Assche *et al*³⁶ who claimed that symmetric growth is explained

by genetic constitutional factors, whereas, asymmetric growth is explained by an abnormal metabolic environment.

A significantly higher percent of the macrosomic group had prolonged hospital stay. This may be partially attributable to the high rate of caesarean deliveries in the macrosomic group, leading to longer maternal hospital stay.³⁷ Furthermore, caesarean delivery is known to be associated with an increased incidence of transient tachypnoea of the newborn,³⁸ which was also true for our macrosomic group. The reason for the slightly increased rate of hyperthermia in the macrosomic group is unclear, as no cases of infection were identified. It is possible that macrosomic infants require less heating, so that the standard placement of infants under a radiant warmer after delivery may be excessive in these cases. Hyperthermia in these neonates could also result from dehydration.

The higher incidence of birth trauma (mainly fracture of the clavicle) was not correlated to any of the anthropometric parameters except head circumference. It is likely that the low incidence of brachial plexus injury relative to reports in the medical literature was also related to the high rate of caesarean deliveries.^{4 25}

In conclusion, macrosomic infants of non-diabetic mothers are at an increased risk of neonatal complications. As symptomatic hypocalcaemia and polycythaemia were not identified in this population, we believe routine measurements of haematocrit and calcium may not be required. Symmetric macrosomic infants (defined by WLR) have a similar rate of hypoglycaemia as normal weight infants. Thus, these infants do not seem to require repeat glucose testing.

Acknowledgements The authors wish to thank Mrs Pearl Lilos for excellent statistical analysis.

Contributors Details of the contribution of individual authors: NL: guarantor, conception and design of study, data interpretation, revision of the article. YL: data analysis, article drafting. AK, NM and YY: intellectual input and design of the study, revision of the article. EF: data analysis and interpretation, intellectual input, revision of the article. FK: data analysis and interpretation, revision of the article. GK: design, analysis, revision of article. All co-authors have approved the final version of the

Table 5 Incidence of hypoglycaemia in macrosomic and control infants

Hypoglycaemia (n/N)	Infant group
33/2766 (1.2)	All macrosomic infants* n (%)
20/2564 (0.8)	Birth weight 4000–4499 g n (%)
11/194 (5.7)	Birth weight 4500–4999 g n (%)
2/8 (25)	Birth weight ≥5000 g n (%)
17/2140 (0.8)	Symmetric macrosomic infants by PI, n (%)
10/1593 (0.6)	Symmetric macrosomic infants by BMI, n (%)
3/1018 (0.3)	Symmetric macrosomic infants by WLR, n (%)
14/2766 (0.5)	Control infants, n (%)

*Birth weight >4000 g.
PI, Ponderal Index; BMI, Body Mass Index; WLR, weight/length ratio.

article, confirm that the work has not been published/submitted elsewhere and agree with submission. All co-authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Competing interests None.

Ethics approval Rabin Medical Center research review board.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- Henriksen T. The macrosomic fetus: a challenge in current obstetrics. *Acta Obstet Gynecol Scand* 2008;87:134–45.
- American College of Obstetricians and Gynecologists. *Fetal macrosomia*. Washington, DC: ACOG Technical Bulletin 1991: No.159.
- Gross TL, Sokol RJ, Williams T, et al. Shoulder dystocia: a fetal-physician risk. *Am J Obstet Gynecol* 1987;156:1408–18.
- Gonen R, Bader D, Ajami M. Effects of a policy of elective cesarean delivery in cases of suspected fetal macrosomia on the incidence of brachial plexus injury and the rate of cesarean delivery. *Am J Obstet Gynecol* 2000;183:1296–300.
- Bromwich P. Big babies. *Br Med J* 1986;293:1387–8.
- Lipscombe KR, Gregory K, Shaw K. The outcome of macrosomic infants weighing at least 4500 grams. Los Angeles County+University of Southern California experience. *Obstet Gynecol* 1995;85:558–64.
- Lavin JP Jr, Lovelace DR, Miodovnik M, et al. Clinical experience with one hundred seven diabetic pregnancies. *Am J Obstet Gynecol* 1983;147:742–52.
- Thomson AM, Billewicz WZ, Hytten FE. The assessment of fetal growth. *J Obstet Gynaecol Br Commonw* 1968;75:903–16.
- Rohrer F. Eine neue Formel zur Bestimmung der Körperfülle, Korrespondenz. *Blatt der Deutschen Gesellschaft für Anthropologie, Ethnologie und Urgeschichte* 1908;39:5–7. [Article in German]
- Cole TJ, Freeman JV, Preece MA. Body mass index reference curves for the UK, 1990. *Arch Dis Child* 1995;73:25–9.
- Dombrowski MP, Berry SM, Johnson MP, et al. Birth weight-length ratios, ponderal indexes, placental weights, and birth weight-placenta ratios in a large population. *Arch Pediatr Adolesc Med* 1994;148:508–12.
- Florey Cdu V. The use and interpretation of ponderal index and other weight-height ratios in epidemiological studies. *J Chronic Dis* 1970;23:93–103.
- Khosla T, Lowe CR. Indices of obesity derived from body weight and height. *Br J Prev Soc Med* 1967;21:122–8.
- Rolland-Cachera MF, Sempé M, Guillaud-Bataille M, et al. Adiposity indices in children. *Am J Clin Nutr* 1982;36:178–84.
- Patterson RM, Pouliot MR. Neonatal morphometrics and perinatal outcome: who is growth retarded? *Am J Obstet Gynecol* 1987;157:691–3.
- Greer FR. Intrauterine growth as estimated from live born birth-weight data at 24 to 42 weeks of gestation, by Lula O. Lubchenko, et al. *Pediatrics* 1963;32:793–800. *Pediatrics* 1998;102:237–9.
- Davidson S, Sokolover N, Erlich A, et al. New and improved Israeli reference of birth weight, birth length and head circumference by gestational age: a hospital based study. *Isr Med Assoc J* 2008;10:130–4.
- Ogata ES. Carbohydrate homeostasis. In: MacDonald MG, Seshia MMK, Mullett MD, eds. *Avery's Neonatology*. 6th edn. Philadelphia: Lippincott Williams & Wilkins, 2005:876–91.
- Maisels MJ. Jaundice in healthy newborns-redefining physiologic jaundice. *West J Med* 1988;149:451.
- Black VD, Lubchenko LO, Luckey DW, et al. Developmental and neurologic sequelae of neonatal hyperviscosity syndrome. *Pediatrics* 1982;69:426–31.
- Dixon WJ. *BMDP statistical software*. Los Angeles, CA: University of California, 1993.
- Davidson S, Natan D, Novikov I, et al. Body mass index and weight-for-length ratio references for infants born at 33–42 weeks gestation: a new tool for anthropometric assessment. *Clin Nutr* 2011;30:634–9.
- Tallarigo L, Giampetro O, Penno G, et al. Relation of glucose tolerance to complications of pregnancy in nondiabetic women. *N Engl J Med* 1986;315:989–92.
- Araz N, Araz M. Frequency of neonatal hypoglycemia in large for gestational age infants of non-diabetic mothers in a community maternity hospital. *Acta Medica* 2006;49:237–9.
- Bertagnon JRD, Segre CAM, DallColletto GM. Weight-for-length relationship at birth to predict neonatal diseases. *São Paulo Med J* 2003;121:149–54.
- Gyurkovits Z, Kálló K, Bakki J, et al. Neonatal outcome of macrosomic infants: an analysis of a two-year period. *Eur J Obstet Gynecol Reprod Biol* 2011;159:289–92.
- Adamkin DH and Committee on Fetus and Newborn. Postnatal glucose homeostasis in late-preterm and term infants. *Pediatrics* 2011;127:575–9.
- Ballard JL, Rosenn B, Khoury JC, et al. Diabetic fetal macrosomia: significance of disproportionate growth. *J Pediatr* 1993;122:115–9.
- Persson M, Pasupathy D, Hanson U, et al. Birth size distribution in 3,705 infants born to mothers with type 1 diabetes. *Diabetes Care* 2011;34:1145–49.
- Persson M, Pasupathy D, Hanson U, et al. Disproportionate body composition and perinatal outcome in large-for-gestational-age infants to mothers with type 1 diabetes. *BJOG* 2012;119:565–72.
- Bollepalli S, Dolan LM, Miodovnik M, et al. Asymmetric large-for-gestational-age infants of type 1 diabetic women: morbidity and abdominal growth. *Am J Perinatol* 2010;27:603–10.
- Rasmussen S, Kiserud T, Albrechtsen S. Foetal size and body proportion at 17–19 weeks of gestation and neonatal size, proportion, and outcome. *Early Hum Dev* 2006;82:683–90.
- Landmann E, Reiss I, Misselwitz B, et al. Ponderal index for discrimination between symmetric and asymmetric growth restriction: percentiles for neonates from 30 weeks to 43 weeks of gestation. *J Matern Fetal Neonatal Med* 2006;19:157–60.
- Schaefer-Graf UM, Rossi R, Bühner C, et al. Rate and risk factors of hypoglycemia in large-for-gestational-age newborn infants of nondiabetic mothers. *Am J Obstet Gynecol* 2002;187:913–7.
- Djelmis J, Bukovic D, Pfeifer D, et al. Ponderal index and disproportionate fetal growth in IDDM pregnancies. *Coll Antropol* 1998;22:491–5.
- Van Assche FA. Symmetric and asymmetric fetal macrosomia in relation to long term consequences. *Am J Obstet Gynecol* 1997;177:1563–4.
- Ju H, Chadha Y, Donovan T, et al. Fetal macrosomia and pregnancy outcomes. *Aust N Z J Obstet Gynaecol* 2009;49:504–9.
- Rawlings JS, Smith FR. Transient tachypnea of the newborn. An analysis of neonatal and obstetric risk factors. *Am J Dis Child* 1984;138:869–71.