

Macrosomia — maternal and fetal risk factors

J. K. Essel, E. T. Opai-Tetteh

Risk factors associated with fetal macrosomia were studied in 348 pregnancies resulting in the delivery of an infant weighing 4 000 g or more in a black population. Identifiable maternal risk factors included a mother in her 3rd decade of life, multiparity, maternal weight of 70 kg or more at the end of pregnancy, prolonged or post-term pregnancy, abnormal glucose tolerance and previous history of a macrosomic infant. Male infants had a higher risk of being macrosomic. Macrosomic infants accounted for 3,4% of all singleton deliveries, with their caesarean section rate of 33,9% being almost three times that of control infants. The importance of antenatal prediction of fetal weight is emphasised and suggestions for reduction of the high perinatal mortality and morbidity rates, as well as maternal morbidity, are discussed.

S Afr Med J 1995; **85**: 43-46.

Little attention has been paid to fetal macrosomia in black African populations, despite the fact that as a high-risk factor in pregnancy and delivery macrosomia probably deserves as much attention as is currently accorded low birth weight, albeit for different reasons. Although perinatal mortality has decreased considerably in recent years,¹ the risks to mother and fetus are serious enough to warrant early detection of macrosomia during labour, and preferably during pregnancy. In fact, Parks and Ziel² suggested that the risks are sufficiently high to justify consideration of elective caesarean section for delivery of such infants. The only study emanating from Africa known to the authors is that by Okpere *et al.*³ from Nigeria.

Our study attempted to identify maternal and fetal risk factors associated with macrosomia in a black African population from another geographical region of Africa. We defined a macrosomic infant as one weighing 4 000 g or more at birth, the currently accepted definition in the developed world.⁴

Patients and methods

Umtata General Hospital, Eastern Cape, serves a population of predominantly low and medium socio-economic status. Between 1 February 1989 and 31 August 1990, 10 507 singleton infants were delivered at the hospital, of whom 360 were macrosomic. Complete documentation was available

Department of Obstetrics and Gynaecology, University of Transkei and Umtata General Hospital, Umtata, Eastern Cape

J. K. Essel, M.B. CH.B., F.W.A.C.S.

E. T. Opai-Tetteh, M.B. CH.B., D.T.M.&H., D.P.H.

for 348 of the 360. An equal number of babies, each delivered within minutes of a macrosomic infant but weighing between 3 000 and 3 200 g, were selected as controls. Mothers and infants in the macrosomic group were compared with those in the control group in respect of age, height, parity, weight at delivery, previous history of macrosomia, glucose tolerance, pregnancy complications, mode of delivery, intrapartum and postpartum complications and neonatal outcome. All pregnancies included in the study had to be at least term by dates (i.e. ≥ 37 completed weeks' gestation), irrespective of booking status or place of prenatal care.

Results were analysed by the simple χ^2 -test. Values of $P < 0,05$ were statistically significant.

Results

The 360 macrosomic infants delivered in the study period constituted 3,43% of all singleton deliveries; 327 of their mothers were booked patients, compared with 329 of the control mothers.

Table I sets out the identified characteristics of the mothers in the two groups. There was a statistically significant increased incidence of macrosomia in the age group 30 - 39 years. Height did not appear to be a significant factor, since about 50% of the macrosomic infants' mothers were less than 160 cm tall. The incidence of macrosomia generally increased with parity and became more obvious in the grand multiparous group. Pre-pregnancy maternal weight and weight gain during pregnancy could not be ascertained because most mothers booked in the second trimester. However, a weight of 70 kg or more at the end of pregnancy was found to be a significant risk factor, as was a previous history of macrosomia (28,4% of mothers in the macrosomic group, compared with 1,1% of controls).

Table I. Maternal characteristics

	Macrosomic infants		Controls	
	No.	%	No.	%
Age (yrs)				
< 20	20	5,7	93	26,7
20 - 29	172	49,4	185	53,2
30 - 39	139	39,9	62	17,8*
≥ 40	17	4,9	8	2,3
Height (cm)				
≤ 160 cm	170	48,9	253	72,7
> 160 cm	178	51,2	95	27,3
Parity				
0	39	11,2	130	37,4
1 - 4	226	64,9	174	50,0†
≥ 5	83	23,9	44	12,6*
Weight at delivery (kg)				
< 70	74	21,3	248	71,3
70 - 90	208	59,8	95	27,3*
≥ 90	66	19,0	5	1,4*
Previous macrosomia	99	28,4	4	1,1*
Abnormal GTT	34	18,5	0	0

* $P < 0,01$.

† $P < 0,05$.

Of the 184 mothers in the macrosomic group (52,9%) who had a glucose tolerance test (GTT) performed, 34 (18,5%) had an abnormal result and 19 (10,3%) were classified as gestational diabetics. The prevalence of gestational diabetes among 51 mothers whose infants weighed 4 500 g or more was 9,8%.

Complications of pregnancy in the two groups are set out in Table II. Post-term delivery occurred in 16,4% of the macrosomic group, compared with 2,9% of controls.

Table II. Complications of pregnancy

	Macrosomic infants		Controls	
	No.	%	No.	%
Post-term pregnancy	57	16,4	10	2,9*
Hypertension/pre-eclampsia	18	5,2	13	3,7
Abruptio placentae	2	0,6	2	0,6
Premature rupture of membranes	4	1,1	5	1,4

* $P < 0,05$.

Table III shows that the caesarean section rate was almost three times higher in the macrosomic group than among the controls. Cephalopelvic disproportion accounted for 90% of abdominal deliveries of macrosomic infants. No forceps deliveries were performed in either group. Thirteen of 22 mothers of macrosomic infants who had a prolonged first stage of labour were given oxytocin and subsequently had a spontaneous uncomplicated vaginal delivery.

Table III. Mode of delivery

	Macrosomic infants		Controls	
	No.	%	No.	%
Spontaneous vaginal delivery	219	62,9	297	85,3
Vacuum extraction	11	3,2	7	2,0
Caesarean section	118	33,9	44	12,6*

* $P < 0,005$.

The incidence of uterine rupture was higher in the macrosomic group (8,6/1 000) than among the controls (2,9/1 000). Of the patients who had vaginal deliveries only 1 mother of a macrosomic infant developed primary postpartum haemorrhage, compared with 4 in the control group; 3 cases of second-degree perineal laceration occurred in the macrosomic group, with 2 in the control group. There was no maternal death in either group.

Table IV shows that 224 of the macrosomic infants were male (male/female ratio 1,8:1,0); the ratio in the control group was 1,07:1. The incidence of birth asphyxia (defined as a 1-minute Apgar score of less than 7) among macrosomic infants delivered vaginally was 4,6%, compared with 5,2% in the control group. There was no difference in the incidence of meconium aspiration. Shoulder dystocia complicated the delivery of 13 (3,7%) of the macrosomic infants: 1 of these developed Erb's palsy in the neonatal period, while another had clavicular fracture. When fetuses who were dead when the mother arrived at the hospital were excluded, the neonatal mortality rate for the macrosomic infants was 23/1 000, compared with 11,5/1 000 for the

control group. Six of 9 perinatal deaths in the macrosomic group were due to avoidable causes, viz. 3 cases of ruptured uterus, 2 cases of obstructed labour, and 1 case of eclampsia. All of these mothers had been referred from peripheral district hospitals.

Table IV. Outcome

	Macrosomic infants		Controls	
	No.	%	No.	%
Male infants	224	64,4	180	51,7
Female infants	124	35,6	168	48,3
Birth asphyxia	16	4,6	18	5,2
Meconium aspiration	4	1,1	3	0,9
Shoulder dystocia	13	3,7	—	—
Brachial plexus palsy	1	0,3	—	—
Clavicular fracture	1	0,3	—	—
Facial palsy	1	0,3	—	—
Perinatal deaths	9	2,6	6	1,7

Discussion

Comparison of the incidence of macrosomia among different racial groups is fraught with difficulties because of the different birth weights and denominators used by various authors.^{3,5-7} In recent years a birth weight of 4 000 g and above has been used by most workers. Using this definition, Modanlou *et al.*⁸ observed an increase in the incidence of macrosomia from 7% in 1960 to 10,7% in 1980. Our incidence of 3,42% is similar to the 3% reported by Marinho⁷ from Nigeria but lower than the 5,5% reported by Gross *et al.*⁶ among 7 123 infants in the USA. While the lower incidence in Africa is difficult to explain, it may be related to the generally lower pre-pregnant weights of black African women. The preponderance of male infants in our study is in accord with findings from other studies.^{1,3}

In order to make the diagnosis of fetal macrosomia antenatally, it is vital to be aware of the predisposing factors. In our environment these were found to be as follows: a mother in her 3rd decade of life who is multiparous or grand multiparous, weighs over 70 kg at term, and has a previous history of a macrosomic infant, a current post-term pregnancy, and gestational diabetes. In fact our 16,4% incidence of post-term or prolonged pregnancy is similar to the 16% reported by Boyd *et al.*¹ The above risk factors are all similar to those identified by other workers.^{3,8} Like Modanlou *et al.*,⁸ our study also confirmed the significance of a previous history of macrosomia.

The role of maternal height remains controversial. Although our study failed to establish any association between maternal height and macrosomia, Boyd *et al.*¹ and Okpere *et al.*³ found height above 169 cm and 160 cm, respectively, to be significant risk factors. Although other studies^{1,3} have reported a weight gain in pregnancy exceeding 13 kg at term as an important risk factor, we could not be certain that it applied in our study population, because most of our patients booked late for antenatal care. The incidence of gestational diabetes of 9,8% among mothers of infants weighing more than 4 500 g is much higher than the 0,7% reported by Oats *et al.*⁹ in a similar

group of mothers. We believe that this finding warrants routine screening for diabetes in mothers with risk factors for macrosomia. Parks and Ziel² reported that a symphysis-fundus height of 40 cm or more may be an important finding. We have yet to determine its significance in our environment.

In the final analysis, ability to estimate fetal weight appears to be of the utmost importance in identification of the macrosomic infant. Clinical estimation can serve as a useful guide in experienced hands, but predictions are often falsely positive or negative.¹⁰ We agree with others¹¹⁻¹³ that real-time ultrasonography gives the best estimate of fetal weight if available and should be used routinely. Although a slow active phase of labour may forewarn of a large baby,¹⁴ our study demonstrates that this *per se* does not exclude judicious use of oxytocin to augment labour.

Despite the fact that macrosomic infants constitute a small percentage of all singleton deliveries, macrosomia is associated with considerable maternal morbidity and high neonatal mortality and morbidity. The high caesarean section rate recorded in this study is similar to rates reported by others.^{1,3,6,10} Although our incidence of shoulder dystocia (3,7%) is similar to others,^{1,15} we experienced fairly low incidences of brachial plexus injury, facial palsy, clavicular fractures, birth asphyxia and meconium aspiration. We believe that this may be because we do not perform midforceps deliveries in our institution. The recent use of ultrasonography for antenatal prediction of shoulder dystocia^{5,16} should help minimise the above risks. The high perinatal mortality rate recorded in our study is grounds for concern, in that most of the causes of death were avoidable.

While awareness of antenatal risk factors is certainly important in the prediction and subsequent management of macrosomia, fetal and maternal outcome depends largely on how well labour is managed. This assertion is supported by the fact that the majority of perinatal deaths in our series were due to obstructed labour and ruptured uterus. It is pertinent to emphasise that these mothers were referred to our institution from peripheral hospitals and that in no case was uterine rupture associated with the presence of a scar from a previous caesarean section or injudicious use of oxytocin in a multigravida. The major underlying cause of these perinatal deaths was late diagnosis of obstructed labour due to macrosomia.

We believe that the unacceptably high perinatal mortality rate and maternal morbidity can be avoided if midwives and labour room doctors are properly trained in the concept of active management of labour, and hence early diagnosis of failure to progress. Clinical suspicion of a large baby coupled with a slow active phase of labour, especially arrest of cervical dilatation over a 2-hour period in the presence of adequate uterine contractions, constitutes an early sign of failure to progress which should not be ignored. In the second stage of labour, intervention is required if there is no descent of the presenting part after 30 minutes of bearing down, or if the patient is undelivered after 45 minutes of pushing.

We express our gratitude to all consultants in the Department of Obstetrics and Gynaecology, Umtata General Hospital, for permission to include their patients in this study. Our thanks also go to the professional nurses in the labour ward for their co-operation.

REFERENCES

1. Boyd ME, Usher R, McLean FH. Fetal macrosomia: prediction, risks and proposed management. *Obstet Gynecol* 1983; **61**: 715-722.
2. Parks DG, Ziel HK. Macrosomia: a proposed indication for primary caesarean section. *Obstet Gynecol* 1978; **52**: 407-409.
3. Okpere EE, Ezimohai M, Agbopuonwu I. Maternal and fetal risk factors associated with macrosomic babies in Benin City, Nigeria. *Nigerian J Obstet Gynecol* 1984; **4**(2): 51-55.
4. Treharne I. Obesity in pregnancy. In: Studd JWW, ed. *Progress in Obstetrics and Gynaecology*. Vol. 4. Edinburgh: Churchill Livingstone, 1984: 127-138.
5. Modanlou HD, Komatsu G, Dorchester WG, Freeman RK, Bosu SK. Large-for-gestational age neonates: anthropometric reasons for shoulder dystocia. *Obstet Gynecol* 1982; **60**: 417-422.
6. Gross TL, Sokol BJ, Williams J, Thompson T. Shoulder dystocia: a fetal-physician risk. *Am J Obstet Gynecol* 1987; **156**: 1408-1418.
7. Marinho AO. Birthweight and low birthweight among live singleton Nigerian infants. *Trop J Obstet Gynecol* 1980; **55**: 420-424.
8. Modanlou HD, Dorchester WL, Thorosian A, Freeman RK. Macrosomia — maternal, fetal and neonatal implications. *Obstet Gynecol* 1980; **55**: 420-424.
9. Oats JN, Abell DA, Beischer NA, Broomhall GR. Maternal glucose tolerance during pregnancy with excessive size infants. *Obstet Gynecol* 1980; **55**: 184-186.
10. Svigos JM. The macrosomic infant: a high risk complication. *Med J Aust* 1981; **1**: 245-246.
11. Campbell S, Wilkin D. Ultrasonic measurements of fetal abdominal circumference in estimation of fetal weight. *Br J Obstet Gynecol* 1975; **82**: 689-697.
12. Ogata ES, Sabbagha R, Metzger BE, Phelps RJ, Depp R, Freinkel N. Serial ultrasonography to assess evolving fetal macrosomia. *JAMA* 1980; **243**: 2405-2408.
13. Sampson MB, Thomason JL, Kelly SL, Work BA. Prediction of intrauterine fetal weight using real time ultrasound. *Am J Obstet Gynecol* 1982; **42**: 554-556.
14. Khatree MHD, Gamsu HR, Rudd P, Studd JWW. Features predictive of brachial plexus injury during labour. *S Afr Med J* 1982; **61**: 232-233.
15. Golditch IM, Kirkman K. The large fetus: management and outcome. *Obstet Gynecol* 1978; **52**: 26-30.
16. Hopwood HG. Shoulder dystocia: fifteen years' experience in a community hospital. *Am J Obstet Gynecol* 1982; **144**: 162-166.

Accepted 18 Apr 1994.
