STATIC WEIGHT CONTROL IN LATE PREGNANCY — A CASE CONTROL STUDY

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SYNOPSIS

Ninety-one static weight cases were compared with 91 matched controls for incidence of meconium stained liquor, abnormal CTG, Apgar score and birthweight at birth, to determine if static weight is associated with an increase in risk of placental insufficiency. Although all the indices measured occurred more frequently in the static weight group, only abnormality of CTG tracing was the difference statistically significant. It was concluded that static weight is probably associated with higher incidence of placental insufficiency. A larger sample size is needed to confirm such an association.

INTRODUCTION

Static weight or weight loss in late pregnancy continues to be used as a clinical sign for placental insufficiency by many.

Browne and Browne (1) and later Browne (2, 3, 4, 5) thought that loss of maternal weight and diminution in abdominal girth in prolonged pregnancy is indicative of placental insufficiency. However, there are others who do not believe in such an association (6). Others believe that the inaccuracies of weighing makes weight changes an unreliable clinical parameter. Inaccuracy of weighing may result from machine inaccuracy and human error in reading the weights or both. More important are other factors which affect the actual weight of the mother for example - a full bladder, after a full meal or heavy clothings and oedema which are unrelated to the fetal state. Elder et al (7) in a prospective study of the prognostic significance of weight and girth meassurements in apparently normal pregnancies found that there were significantly more dysmature babies in those who had static or weight loss after the 34th week of gestation compared with those who gained weights. There was however no significant difference in incidence of fetal distress in labour.

In Kandang Kerbau Hospital (KKH), recording of maternal weight continues to be a routine in our antenatal clinic. As Kandang Kerbau Hospital has a very large case load of 24,000 deliveries a year, our antenatal clinics are very congested. In view of this, many are skeptical of the accuracy of the weights measured and recorded in our antenatal clinics. However, a sizeable proportion of doctors in KKH induce patients with static weight or weight loss around term believing that these are associated with placental insufficiency. This case control study was designed to determine if there is any justification for inducing patients with a defined static weight in the context of KKH.

		STUDY GROUP	CONTROL GROUP
Age (years)	: Range	18 to 37	17 to 35
	Mean	26.32	26.04
Parity	: Range	0 to 4	0 to 4
	Mean	0.71	0.79
Gestation at onset of labour (week)	: Range Mean	37 to 44 40.35	38 to 44 40.07
Race .	: Chinese	72	72
	Malay	14	14
	Indian	5	5

Table 1 — DATA RELATING TO THE AGE, PARITY, GESTATION AT THE ONSET OF LABOUR AND RACIAL DISTRIBUTION OF PATIENTS

Table 1a — AGE DISTRIBUTION OF PATIENTS IN STUDY AND CONTROL GROUPS

AGE	NUMBER OF PATIENTS			
YEARS	STUDY GROUP	CONTROL GROUP		
17 — 19	5	4		
20 — 24	30	33		
25 — 29	31	33		
30 — 34	24	16		
35 — 37	1	4		
	91	91		

Table 1b — PARITY DISTRIBUTION OF PATIENTS IN STUDY AND CONTROL GROUPS

DADA	NUMBER OF PATIENTS				
PARA	STUDY GROUP	CONTROL GROUP			
0	46	45			
1	28	28			
2	15	14			
3	1	2			
4	1	· 2			
	· 91	91			

Table 1c — GESTATION AT ONSET OF LABOUR IN STUDY AND CONTROL GROUPS PATIENTS

GESTATION AT ONSET OF LABOUR	NUMBER OF PATIENTS			
	STUDY GROUP	CONTROL GROUP		
37 weeks	1	2		
38	4	7		
. 39	23	25		
40	21	29		
41	18	10		
42	15	9		
42 +	9	9		
	91	91		

MATERIAL AND METHOD

Cases: Ninety-one static weight, otherwise normal pregnant patients were collected January 1979 and April 1981. A static weight patient is defined as one who had not gained any weight or who has lost weight over a minimum period of two weeks after the 34th week.

Controls: For each study case, a control was selected matching for age, parity, race and gestation at the onset of labour.

The first fifty cases of controls were selected prospectively at the antenatal clinic. Four control cases were selected for each study case antenatally which were matched for age, parity, race and height. Of the four control cases (for each study case) one control case which matches the study case in gestation at the onset of labour is used as the actual control case for analysis. The remaining forty-one control cases were selected retrospectively. These control cases were selected randomly from the postnatal ward patients which matched in age, parity, race and gestation at the onset of labour as the study cases.

Weighing procedure: All the patients were weighed fully clothed without their shoes and handbags on an Avery counter balance weighing machine, to the nearest 0.1 Kg. The patients had their bladders emptied before weighing. Weighing of a sample of antenatal patient's clothings showed the mean and mode to be 0.31 Kg and 0.2 Kg.

Labour: All patients in the study had spontaneous onset of labour except eight patients in the study group whose labours were induced at past 42 weeks gestation. Labour was augmented with intravenous oxytocin when indicated. The colour of the liquor was noted during vaginal examina-

	STUDY GROUP		CONTROL GROUP		
	Number	Apgar Score	Number	Apgar Score	
Light meconium stained liquor	3	2, 5, and 9	4	All 7	
Moderate meconium stained liquor	7	All 7 except one (Apgar 5)	8	All 7	
Thick meconium stained liquor	12	All 7 except 5 patients (all Apgar 6)	2	8 and 8	
Clear liquor	69	All 7	77	All 7	
TOTAL	91		91		

Table 2 — STATE OF LIQUOR AMNII DURING LABOUR

Table 2a — MATCHED PAIR ANALYSIS IN BOTH STUDY AND CONTROL GROUPS FOR MECONIUM STAINED LIQUOR

	Control Group Meconium Liquor		
Study Group	+ + 5* (3)	- 17* (17)	
Meconium Liquor			
	- 9*	60*	
	(7)	(64)	

n	=	91
*	Ξ	Include light, moderate and thick meconium liquor
X²	Ħ	1.88
Р	=	0.15 (not significant)
Bracket	≕	Include only moderate and thick meconium liquor
X²	=	3.11
Р	=	0.07 (not significant)

	STUDY GROUP		CONT	
	Number	*Apgar Score	Number	*Apgar Score
Early deceleration (Type I)	11	All 7 except two patients (Apgar 5 + 6)	6	All 7
Late deceleration (Type II)	1	7	0	
Basal bradycardia	2	2 and 8	0	
Basal bradycardia with deceleration	2	6 and 7	0	
Basal tachycardia	1	6	0	
Loss of baseline variability	1	8		
	18		6	

Table 3 — CARDIOTOCOGRAPHIC TRACING RESULTS

Appar score at one minute after birth

No sedative given





Matched Pair Analysis X² = 6.86

P = 0.01 (significant)

tion and continuous cardiotocographic monitoring was performed in those patients with meconium stained liquor or fetal heart beat abnormality on auscultation.

Any drug administered during labour was noted and narcotic analgesia was not used if delivery was imminent, within the next two hours. The baby was Apgar scored at minute after birth and the birthweight recorded. The placenta was weighed and noted for any abnormality.

Analysis: Matched pair analysis and paired T test were used to test for differences in variables compared.

RESULTS

The incidence of static weight as defined, excluding those patients with medical and obstetrical complications was 1.3 percent. However, an incidence of 6.8 percent was observed if patients with medical and obstetrical complications were included.

Table 1 to 1C shows that the study cases and controls were fairly well matched for race, age, parity and gestation at onset of labour. There were no perinatal death. Liquor amnii during labour: (Tables 2 and 2A)

In the study group, there were twenty-two patients with meconium stained liquor (eight of these babies had Apgar score of less than seven, one minute after birth) as compared with fourteen patients in the control group (all these babies had Apgar score of seven or more, one minute after birth). The difference is not statistically significant (P=0.07). Cardiotocographic (CTG) monitoring: (Tables 3 and 3A)

Abnormal CTG tracings during labour were more frequently observed in the study group (eighteen patients) as compared with the control group (six patients). This difference is statistically significant (P = 0.01). In the study group, five babies had poor Apgar score at birth (Apgar 6 or less) whereas in the control group, all the babies had statisfactory Apgar scores (Apgar 7 or more) at birth.

Mode of delivery: Nine patients in the study group had their pregnancies terminated by emergency Caesarian section for fetal distress. Of these, three babies had poor Apgar scores - one minute after birth (Apgar two, five and

	STUDY GROUP		CONTROL GROUP		Paired t — Test
	Mean	SE	Mean	SE	
Apgar Score	8.32	0.14	8.58	0.04	P>0.05
Birth Weight	3105 grams	43.35	3177 grams	58,75	P>0.05
Placental Weight	506.9 grams	9.01	505.1 grams	8.49	P>0.05

Table 4 — APGAR SCORE AT ONE MINUTE AFTER BIRTH OF INFANTS BIRTH WEIGHT AND PLACENTAL WEIGHT

SE = Standard Error

six respectively). The remaining three babies had satisfactory Apgar scores one minute after birth (Apgar 7 or more).

In the control group, three patients had emergency Caesarian sections for similar indication. All the three babies had satisfactory Apgar score one minute after birth (Apgar 7 or more).

Assisted outlet forceps deliveries for prolonged second stage of labour were performed in nine patients in the study group and elevan patients in the control group. None of the babies had Apgar score of less than seven.

Apgar Score: (Table 4)

The mean Apgar score at one minute after birth for the study group was lower than the control group (8.32 and 8.58 respectively) which was not statistically significant. Eight of the study group hadd Apgar scores of less than seven, whereas there were none in the control group.

The mean birthweight and placental weight were not significantly different between the two groups. There were two cases of dysmature infants in the study group, whereas there were none in the control group.

DISCUSSION

True static weight during the last six weeks of pregnancy could either be due to (1) failure of the fetus to grow (2) decrease in weight of liquor and placenta more than the weight gained by the fetus, or (3) loss in weight of mother, other than the products of conception. In normal pregnancies, true loss in weight of the mother other than the products of conception is probably uncommon. The situation in (1) and (2) are more likely causes of true static weight. Both these conditions are considered to be effects of placental insufficiency. Theoretically then, true static or loss in weight in normal pregnancies should be a sign of placental insufficiency.

However, in practice, it is difficult to detect true static weight for the following reasons:

- The actual increase in weight attributed to the product of conception during the last four to six weeks of pregnancy is smal amounting to only about 0.36 Kg per week.
- Variation in recorded weight may result from errors made by same nurse or by different nurses reading the weights at different visits.

Other factors are also known to affect fetal growth such as pre-eclampsia, age, parity, fetal malformation, diabetes, gestation, ethnic group and socio-economic status. It is therefore understandable that static weight has not been shown conclusively to be a reliable clinical sign of placental insufficiency. Elder et al (7) in their study showd a higher incidence of dysmaturity in the static weight group. They, however, did not control for variables such as age, parity and gestation which may bias their results. Moreover, many of their static weight cases were induced.

We attempted to find out for ourselves if the static weight pregnancies detected from the weight recorded routinely by our midwives at each antenatal visit, is associated with an increase in incidence of fetal distress in labour, intrauterine growth retardation and fetal loss compared with those without static weight. We have attempted to control for other factors that may bias our results by matching the cases with controls for the relevant factors and excluding any obstetrical or medical complications. We did not induce our patients for static weight unless they go past 42 weeks.

In the study group signs of fetal distress occur more frequently than the control group - meconium stained liquor occurred in 24% in the study group compared with 15% in the control. 8.8% of study group had Apgar score of less than seven compared with none in the control group. Twenty percent of the study group had abnormalities of cardiotocographic tracings compared with 6% in the control group. The mean birthweight was less for the study group. Only two study infants were dysmature. Although the incidence is higher for all these signs, only in abnormality of cardiotocographic tracings was the difference statistically significant. We cannot exclude bias in explaining the difference. The way our controls were selected retrospectively (forty-one cases) could be a source of bias. Static weight cases are considered high-risk pregnancies by many and therefore closer monitoring could have been given to these cases. Apgar scoring is poorly done in our hospital and therefore may not be a reliable index for comparison of fetal condition at birth between the two groups.

Meconium staining rate of 24% is substantially higher than that of the control group (15%). Although the difference is not statistically significant, with our sample size, the chances of detecting a significant difference is only 66%.

As the incidence of all the indices of placental insufficiency measured were higher in the study group, it is probable that the increased incidences are related to static weight. Undoubtedly, the static weight group would have a higher number of patients with true static weight inspite of variations in weight recordings. A larger sample is needed to show conclusively the value of static weight detected in our set up, as an index of placental insufficiency. In the meantime, it is desirable to further investigate these cases by antenatal cardiotocography before inducing them. This should not give rise to any logistic problem as our study showed the incidence of static weight in normal pregnancies to be only 1.3%. If cases with medical and obstetric complications are included the incidence is 6.8%.

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