Update on Obstetrics

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Curriculum Vitae

Dr CR Nelson graduated from Wits Medical School in 1970 and obtained his MRCOG in London in 1975. From 1974 to 1977 he was registrar in obstetrics and gynaecology at the John Radcliffe Hospital, Oxford. In 1977 he commenced private specialist practice and is part time consultant to Edendale Hospital, Pietermaritzburg and Consultant-in-Charge, Infertility and Endocrine Clinic, Northdale Hospital, Pietermaritzburg.

Summary

This paper is concerned with the foetus that dies of placental insufficiency. It evaluates the high risk pregnancy clinically, with ultrasound, with hormone assays and with a few other dynamic tests of foetal well-being. It discusses the unnecessary Caesarian section and gives guidelines regarding active management in labour.

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THE UNNECESSARY FOETAL DEATH

The death of babies from asphyxia during pregnancy, childbirth, and shortly thereafter, is predominantly due to:

- Immaturity (Prematurity)
- Undernourishment (placental insufficiency)
- Incidental occurrences eg abruptio; cord accidents
- Inappropriate obstetric management.

This discussion concerns the foetus that dies of undernutrition.

Evaluation of the high risk pregnancy can be broken up into the following categories: clinical, ultrasound, hormone assays and dynamic tests of foetal well-being.

Clinical Evaluation

1. a The period of gestation must be known. Factors which can assist one here are:
   (i) Reliable dates
   (ii) Early bimanual uterine size evaluation in the first trimester.
   (iii) When in doubt, carry out a BPD estimation at 20 weeks gestation.
   b Measurements of uterine growth (girth and fundal height)
   c Assessment of oligohydramnios.
   d Serial maternal weight change.

2. **Ultrasound Evaluation**
   The sonographic age must be determined in early pregnancy in order to detect slowing of the foetal growth rate at a later stage. The optimum time for this is between the 20th and 24th week. *ISOLATED MEASUREMENTS OF THE BIPARIETAL DIAMETER IN THE THIRD TRIMESTER CANNOT DETERMINE WHETHER A GIVEN INFANT IS SMALL FOR DATES.* The evaluation of the biparietal diameter growth is then done, in accordance with well accepted graphs. The danger signal here will be a fall-off of the BPD-measurements when compared to the mean over a number of weeks. Often one will be able to detect the presence of an intra-uterine congenital abnormality by the fact that the biparietal diameter seems to run in parallel with the mean growth rate, the so-called symmetrical growth retardation problem.

There is no single test which, in isolation, determines foetal well-being.

Remember that asymmetrical growth retardation, where there is a brain sparing effect, can only be assessed by estimating the biparietal diameter in relation to the abdominal girth. Congenital anomaly has poor growth potential.

3. **Hormone assays**
   In view of the fact that overlap exists between the values of the normal and abnormal pregnancy, it is extremely difficult for these tests to be of value in isolation. In addition, because of the dynamic nature of hormone production and metabolism, the measurement of urinary metabolites will only give a vague, indirect indication as to the true state of the placental condition and therefore the place of hormonal assays in placental monitoring is doubtful.

4. **Dynamic tests of Foetal Wellbeing**
   Two main categories of tests are now in use:-
   a Foetal movement.
   b. Antenatal cardiotocography — the latter requires a foetal monitor which should be available. Practising obstetrics in 1984 without a CTG-machine is like managing the coronary patient without an ECG.

FOETAL ACTIVITY — A MEASURE OF WELLBEING

The foetus has periods of rest and activity commencing very early in the gestation. The rest/activity periods alternate in a 1:2 ratio in cycles of ± one hour. The following factors affect the activity states:

**Gestational age*
Foetal activity is better defined in the third trimester — stretching rolling and isolated limb movements can be observed with real-time ultra sound. As a general rule, no more than 75 minutes should elapse between periods of foetal movements. The respiratory movements become more regular towards term — the heart rate becomes more variable — and there is increased foetal heart rate acceleration in relation to foetal movements.

**Circadian rhythm*
Between 38 and 39 weeks the peak time for foetal activity is around midnight with a scatter from 9pm to 1am. Foetal breathing movements are most prominent between 4am and 7am with the lowest heart rate recordings during the same period.

During labour there may be less foetal heart rate accelerations in response to movements.

**Blood glucose*
Decreased maternal blood glucose is associated with decreased foetal breathing movements and following a meal or an injection of glucose, they increase.

**Labour*
During labour there may be less foetal heart rate accelerations in response to movements.

CLINICAL APPLICATION OF FOETAL ACTIVITY

Foetal movements decrease prior to death and are absent completely for 12-48 hours before the foetal heart stops. The recording of foetal movements is obviously a subjective test, but the important factor is the number of movements felt by the mother which should indicate the need for closer monitoring of the foetus.

**Cardiff Count to 10 Test*
This is the simplest test and merely involves the patient recording her foetal movements starting at 9.00am and counting them until 10 movements have been monitored. The time by which 10 movements are felt is recorded, and the patient then continues her daily business. If less than 10 movements are felt during a twelve hour period, the practitioner must be notified immediately and the patient admitted for closer observation.

CTG MONITORING (NST: NON STRESS TEST)

Thirty minutes of monitoring the foetus are required with recording of continuous foetal heart rate, any Braxton Hicks contractions which may occur and the foetal movements which are noted by the patient herself. The normal picture is a foetal heart rate of 120-160 beats per minute with variability, accelerations in response to movements and no change in the heart rate, during or following a Braxton Hicks contraction.

Danger Signals
1. A lack of acceleration of foetal heart rate in response to movements. This picture is associated with increased perinatal death, increased Caesarean section rate and decreased Apgar scores at birth.
2. Foetal heart rate slowing in response to uterine contraction (if this mimics a Type II deceleration) can be of significance. It can be a reflective of a foetus in imminent danger of dying. False positives — these will be obtained during periods of foetal quiescence. My advice therefore, in the high risk patient, is that the non-stress test be carried out for longer periods of observation or on a number of occasions over one day.

There is no single test which, in isolation, will accurately determine the foetal well-being, and it therefore behoves the clinician to utilize all of the data outlined above in assessing the at risk foetus.

THE UNNECESSARY CAESAREAN SECTION — *The active management of labour*

The partographic control of labour has now outdated an impression of normal labour based on palpation of uterine contractions and occasional vaginal examinations. Progress in labour is now being defined strictly by statisco-graphical means based on progressive cervical dilatation. If one uses the partogram curves which have been defined by Philpott, Stud et al, delay in labour can be detected at an early stage and dysfunctional labour can be corrected before the problems of prolonged labour are compounded. Not only

do we record the rate of cervical dilatation but the foetal head level should be assessed and recorded by bimanual abdominal palpation, and described as the number of fifths of the head palpable above the pelvic brim. The uterine activity is also recorded on the same graph, but although the relative strength of uterine contractions is important, it will give no information regarding the progress of labour. The important factor here is cervical dilatation.

Normal labour

Normal labour has been studied and defined as one which started spontaneously, was not stimulated by oxytocic drugs, did not have a lumbar epidural block, did not require operative delivery, and in which a baby with a mass greater than 2,5Kgs was delivered.

By these criteria 29% of primips and 49% of multigravida are normal.

The active management of labour

A simple protocol to identify and treat dysfunctional labour is as follows:

1. When the patient is admitted in labour, a vaginal examination is performed and the cervical dilatation charted at the zero time on the partogram.
2. The appropriate normogram is drawn on the partogram from zero time to 10cm dilatation.
3. Vaginal examination is performed every three hours to assess cervical dilatation, and the patient's curve of progress is charted.
4. Artificial rupture of the membranes is performed if one is confident that labour is established and the head 4/5 or less palpable abdominally. This would allow assessment of the liquor, stimulate labour and would allow the attachment of a foetal scalp electrode if necessary.
5. If labour then progresses to the left of the normogram or less than two hours to the right, it is considered to be normal and no augmentation is given.
6. If cervical dilatation strays more than two hours to the right of the normogram, labour is judged to be dysfunctional and contractions are stimulated with an intravenous infusion of Syntocinon. The dose is stepped-up incrementally from 2 milli units per minute = one unit of Syntocinon in one litre of 5% dextrose water, running at 30 drops per minute. Before Ocitocin is administered, a vaginal examination must be performed by the doctor to detect any malpresentation or gross pelvic abnormality and to ensure that the membranes are ruptured. This is especially important in multiparous patients.
7. The labour is now high risk and continuous foetal heart rate monitoring is a vital part of the acceleration of labour.

The primigravid uterus is rupture-proof whereas the multigravid uterus is rupture-prone.

This implies that Syntocinon augmentation of multigravid labour must be used judiciously with extreme care and in the absence of cephalo-pelvic disproportion. By the same token a uterus which is not contracting adequately enough to produce cervical dilatation, is probably not contracting adequately enough to rupture itself in the absence of cephalopelvic disproportion.

Abnormal Labour Patterns

a. *Prolonged latent phase:*
The latent phase of labour is the interval from its onset until the cervix is 3cm dilated. A prolonged latent phase is one which lasts more than six hours to a dilatation of 3cm. It is sometimes difficult to distinguish between a prolonged latent phase and spurious pre-labour, and therefore it is better in most cases to be conservative rather than rupture the membranes as this means that you are committed to delivery within twenty four hours. If you are wrong about the diagnosis of labour, and contractions cease with the membranes intact, that is of no consequence.

b. *Primary dysfunctional labour:*
This follows a normal latent phase when the cervix dilates less than one cm an hour within a normal active phase having been established. It is the commonest of abnormal labour patterns and in most cases, is corrected by augmentation.

c. *Secondary arrest of labour:*
Secondary arrest in the active phase describes the cervo-metric progress when the active phase of labour commences normally but stops, or slows, significantly prior to full dilation of the cervix. This time of aberrant labour has always been regarded as indicating a dangerous problem, and may well be suggestive of the presence of cephalo-pelvic disproportion and is particularly so in multigravida with the attendant risk of uterine rupture.

OUTCOME

*Group a —	Normal progress	SVD=80,6% C/S= 1,6% INS=17,8%
*Group b —	Primary Dysfunctional labour	SVD=58,6% C/S= 6,2% INS=35,2%
*Group c —	Secondary Arrest	SVD=53,8% C/S=11,6% INS=34,6%

RESULTS

The improvement in the outcome and the reduction of the Caesarean section rate following augmentation is dramatic. In African patients, Philpott reported a reduction of Caesarean section rate from 9%-2,7% and O'Driscoll achieved a Caesarean section rate of 1,1% in spontaneous labour, in primigravida. There have been other reports of Caesarean rates of 8% in primigravida and 1% in Multigravida who started labour spontaneously.

The ideal way to manage labour is to avoid induction if possible, to recognise and to correct dysfunctional spontaneous labour at an early stage thus avoiding prolonged labour, excessive maternal morbidity and neonatal compromise.

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