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Abstract

Based on an Address delivered to the Royal Medical Society, on Friday, 13th January. 1961.

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THE UNBORN CHILD

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Society, on Friday, 13th January, 1961.

Just as the clinical picture of any pregnancy changes from week to week so the picture of obstetric practice as a whole is changing. As childbearing and childbirth have become safer for the mother more and more attention has been directed to her unborn child. The foetus and its placenta, however, are remote when compared with the mother and therefore can often only be studied indirectly.

The Examination of the Foetus

The methods of examination of the foetus in late pregnancy are mentioned first. We can palpate it, estimate its size however roughly, recognise movements, look at its shadow on an X-ray film and over a period of time estimate its growth. Also we can listen to the foetal heart sounds, but listening is one thing and understanding what we hear is another. It is difficult to interpret foetal heart irregularities and observed changes may mean different things in different cases. The ordinary methods of listening to the foetal heart are good enough in most cases, but for some, rather more precise techniques should be employed. In cases of special difficulty the foetal heart can be monitored with a cardiophone. This apparatus is expensive but provides a clear and continuous record of the foetal heart rate, though the sounds differ from those heard with the ordinary stethoscope.

As a result of this monitoring of the foetal heart in special cases we have noted changes in the rate, not otherwise detectable, which we have interpreted as meaning that the child was in danger. Some of these changes have been translated into graphic records. Figure 1 demonstrates intermittent slowing of the foetal heart rate in a case of severe placental insufficiency. The routine interrupted listening had revealed no abnormality but continuous recording over many hours showed that from time to time the heart rate dropped alarmingly. After observation for 24 hours it was decided to deliver the baby by Caesarean Section despite its tiny size because it was thought that otherwise it would die in utero. This was done and as we suspected, the placenta was small and extensively infarcted. The child did not survive.

In another case, illustrated in Figure 2, a patient who had lost her previous child was admitted because she was a few days beyond the expected date of delivery. Induction of labour was decided on and an attempt was made to rupture the forewaters, but no liquor was obtained. The child was small and the foetal heart in this case was also monitored. For nearly an hour after the

attempted artificial rupture of the membranes there was no suggestion of foetal heart irregularity. Then quite suddenly and rather unexpectedly the rate became grossly irregular, occasionally falling to 80 and 90. Believing the foetus to be in danger, we performed Caesarean Section; this child survived. A particular question which we were not able to answer was why the foetal heart should have shown this gross slowing and irregularity, and why in a case which was otherwise perfectly normal at 41 weeks was there no liquor in the uterus.

Attention to the foetus is very important and so is attention to the placenta but this remarkable structure is even more difficult to study; as there is no direct method of examination, indirect methods have to be relied on.

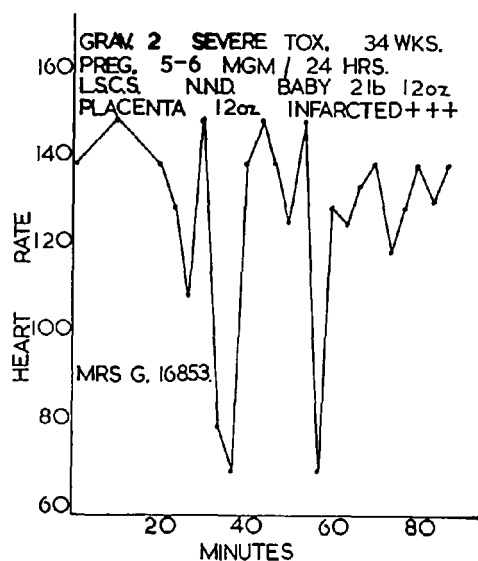


FIG. 1. Intermittent slowing of the foetal heart in a case of severe placental insufficiency.

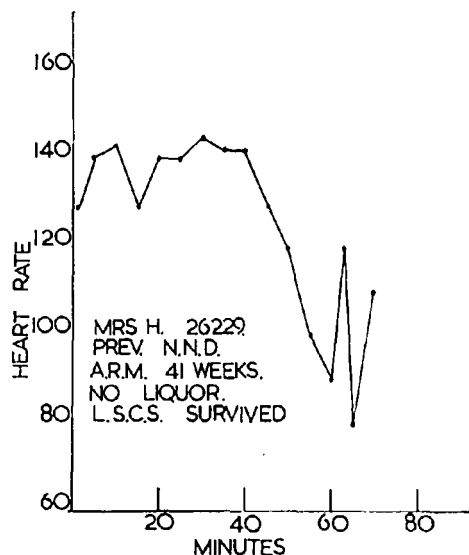


FIG. 2. Slowing of the foetal heart in a case with absent liquor.

Placental Reserve

The placenta normally has ample reserve for supplying the needs of the growing foetus. Sometimes, however, diminishing vascularity and increasing fibrosis can materially reduce this reserve until the amount of functioning tissue is barely sufficient or is insufficient for the foetal requirements; interference with nutrition will then retard foetal growth and the child may just survive, or succumb during labour, or in cases of severe placental insufficiency, die in utero some time before term. Sometimes the placenta is too small to nourish a normal sized foetus and the placental reserve gets used up before term.

Placental Insufficiency

Placental insufficiency though as old as midwifery itself is rather a new expression and like other new expressions has different shades of meaning. The failure of function can occur quickly, e.g. in accidental haemorrhage or more slowly as in pregnancy toxæmia; also the failure may occur at different stages of pregnancy. Placental insufficiency is most often seen in association with hypertension or toxæmia of pregnancy or both, but it is not an invariable complication of even the more severe examples of these diseases and it may

occur in the absence of these conditions. The problem is how to recognise in any particular case if there is sufficient placental damage to affect the foetus. In the Ante-natal period, the recognition of a small child which fails to grow shows that it is in danger, and the estimate of the degree to which its growth is affected is the first measure of how great this danger is.

Hormonal Excretion in Pregnancy

Additional information enabling a better estimate of placental function would be of undoubted benefit in such cases. To this end we have in Sheffield been studying the hormonal excretion in pregnancy, and particularly the excretion of pregnanediol, the principal excretion product of progesterone. During pregnancy there is a steady increase in the urinary output of pregnanediol until about the 35th or 36th week when the rising curve of excretion flattens off and falls slightly as term approaches (Fig. 3).

Our early work (Russell, Paine, Coyle and Dewhurst 1957) indicated that in cases in which there was placental damage, the levels of excretion were below normal and therefore that knowledge of the levels of excretion should prove helpful in practice.

More recently we reported (Russell, Dewhurst and Blakey 1960) a study of the pregnanediol excretion in a group of 58 patients in whom we had diagnosed on clinical evidence placental insufficiency or in whom there was the possibility that it might be present. Most of the patients either suffered from hypertension or pre-eclampsia or gave a history of a previous still-birth believed to be due to placental insufficiency; a few patients had no such associated abnormalities.

We based our clinical diagnosis of placental insufficiency on the finding

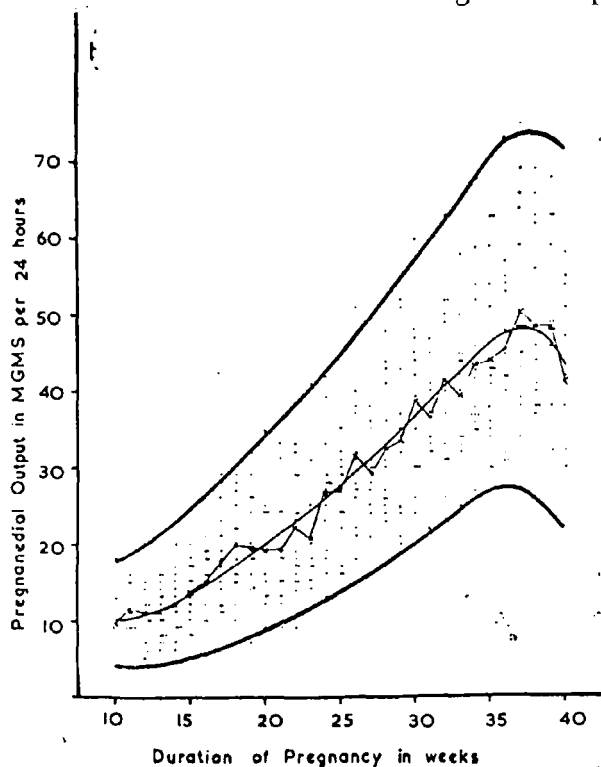


FIG. 3. Pregnanediol excretion in pregnancy: normal distribution.

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of a small child which did not grow normally; on this basis we made a confident diagnosis of placental insufficiency in 16 cases (Group 1); in another 14 (Group 2) we considered placental damage was not significant; and in the

remaining 28 cases (Group 3) we could not decide on clinical grounds alone whether there was placental insufficiency or not.

Pregnanediol studies were carried out in all cases. The method of assay was that described by Klopper (1955). In Group 1 low readings of pregnanediol confirmed our diagnosis of placental insufficiency in 14 of the 16 cases; in the remaining 2 cases normal readings indicating good placental function led to a revised diagnosis (Fig. 4). In Group 2, normal readings were obtained in

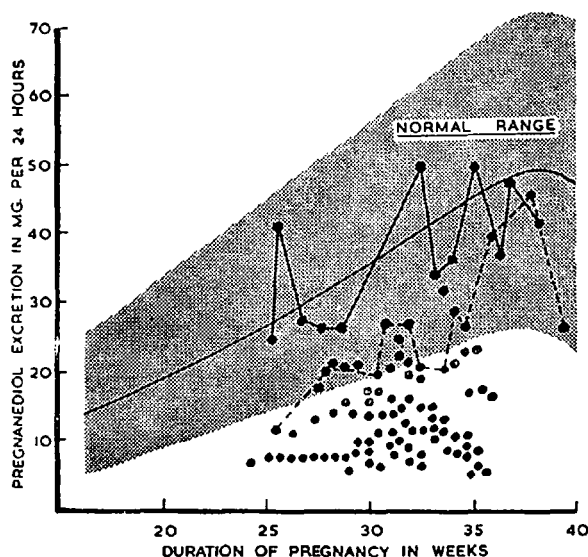


FIG. 4. Pregnanediol excretion in the 16 cases comprising Group 1.

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13 cases supporting our clinical impression; in one case low readings suggested some placental damage. In Group 3, readings within the normal range but below the normal average showed reasonable placental function in 10 cases; low readings showed placental insufficiency in 15 cases, while in 3 cases equivocal results were obtained.

Table 1.
CLINICAL GROUPING OF CASES

Total No. of cases in the survey	58
Group 1 (Placental insufficiency)	16
Group 2 (No placental insufficiency)	14
Group 3 (Uncertain)	28

Taking all 3 groups together there were 30 cases with low readings. In all of these there was evidence of enough placental damage to affect the child; 11 babies were still-born and 2 died in the neo-natal period; all the babies were below average weight for the stage of pregnancy. The placentae were small and in addition showed evidence of infarction which was sometimes very severe. By contrast, in the 25 cases with normal values, apart from one unexplained death, the weights of the babies and of the placentae were normal.

The pregnanediol assay provided us therefore with confirmation or correction of our clinical diagnosis, or the only reliable evidence of the state of the

placenta when a clinical diagnosis could not be made ; moreover, the amount by which the readings fell below the normal range indicated the severity of the placental damage and the urgency of the problem as it affected the child.

The treatment of an individual case was not based on the pregnanediol readings alone, but on all aspects of the case. The assay was helpful none the less, for although the mode of the delivery was decided almost exclusively on clinical grounds, the timing of induction or operation was simplified by our greater knowledge of the degree of placental damage present.

In a number of cases, the knowledge that there was a severe degree of placental damage made it clear that if the child was to be saved delivery was necessary even although the child was tiny and the duration of pregnancy perhaps not more than 32 weeks ; in such cases without this additional knowledge our inclination would certainly have been to delay interference with the likely death of the foetus in utero. In other instances we were encouraged by good readings to withhold interference until the child was more mature or even altogether : in still others, a number of late pregnancy readings falling just below the normal range suggested that it would be unwise to allow the patient to go overdue.

The assay requires accuracy and precision if reliance is to be placed on the result obtained. A 24 hour specimen of urine is needed and must be collected exactly, and the assay carefully performed. There is some uncontrollable error so second or third specimens should often be assayed. Reliance on the average of more than one assay divides the error of the single assay by the square root of the number of assays.

The Delivery of the Child

Having decided that the foetus is in danger in utero either labour must be induced prematurely or Caesarean Section performed. This choice though superficially straightforward can be most difficult because practice has shown that there is no guarantee that the child will survive its delivery however this is arranged. There is much still to be learnt about normal labour. What aspects of natural vaginal delivery are good for the child? The means by which the baby negotiates the birth canal may be passed over quickly, the descent and flexion of the baby's head, the internal rotation, the extension, the restitution and so on are all important but the slow squeezing of the baby's head and thorax in labour has also to be considered. Is this a hazard separating the robust from the fragile baby? Perhaps the squeezing of the baby's head is a way of showing the baby that life may be hard and that it had better get on with it. The squeezing of the child's thorax in the course of a normal delivery may be an important means by which the chest is cleared of any debris or liquor that may have been inhaled during the practice respirations that we know go on in utero.

These considerations are important because as an alternative to vaginal delivery we may perform Caesarean Section and in this operation there can be no comparable pressure on head and thorax. There is seldom any serious difficulty with the robust foetus of good size but the minutiae of the delivery may be of prime importance when the foetus only weighs 3 - 4 lbs. or even less. My impression is that normal labour is preferable to Caesarean Section for the very small foetus but the labour must be normal otherwise different hazards operate ; the closest watch must be maintained at the end of the first and beginning of the second stages because it is then that the foetus is in danger of asphyxia.

Other Hazards to the Unborn Child.

The unborn child may die in utero or it may die after its birth but there are

other hazards. It may be born handicapped, be mentally or physically retarded, or suffer from epilepsy or cerebral palsy, etc. These hazards are also being studied by us and I should like to record our gratitude to the National Spastics Society for their most generous financial support; it is to be hoped that we will be able to throw some further light on these distressing diseases. More needs to be known about the relationship between the foetus and its placenta. What decides the size of the placenta? Is placental growth dependent on the uterine blood flow? Can the foetus make its own placenta grow? These and other questions await answer.

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