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A clinician looks at the placenta

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THE PLACENTA has long been the Cinderella organ of the obstetricians. He has learned over the years and taught his midwives to glance at it in cupped hands, stroke the membranes, and pronounce the magic word "complete" before tossing it away into the bin.

The more modern obstetrician has taken it home to fertilise his rose garden, or has arranged for his hospital to sell it to those purveyors of beauty who believe in adding other people's hormones to their beauty creams.

But in fact, a closer study of the placenta is not necessarily something useful just for those interested in the biology of pregnancy or for those whose business it is to perform post-mortem studies. It can be of practical value in the management of the patient. It is therefore our purpose today to explore how useful it is to study the placenta in the management of the mother and child.

Examination of the Placenta – Technique

Let us begin first by looking in more detail at that cursory glance at the placenta. In a significant proportion of patients with postpartum haemorrhage, exploration of the uterus produces placental tissue, sometimes remarkable in amount. How could it be missed? Firstly that cursory glance may have been really cursory. There is no place for a careless examination. Postpartum haemorrhage from retained cotyledons has been known to kill.

Fallacies

Secondly, it is possible to make a genuine mistake. If the placenta is cupped too strongly, the neighbouring cotyledons maybe made to crowd over the area of the missing cotyledon, making the placenta appear complete.

Thirdly, the area of the missing cotyledon may be obscured by blood clot. It is therefore good practice to wash or brush away blood clot adherent to the placenta before examining the placenta for completeness.

There is yet another way in which the lost cotyledon maybe missed: this is when the membranes are not carefully examined and blood vessels leading into them are missed, as also the succenturiate lobe which the vessels supplied.

Finally, in the prevention of post partum haemorrhage, the membranes: parts of these are quite easily missed, and the easiest way to check them is by turning the placental sac "inside out", letting the membranes hang. They have also been examined under water – but that is another story.

Placental Shape

We next come to the shape of the placenta. The circumvallate and circummarginate placenta is more common than is generally recognised (Fox & Sen, 1972), but while its association with several conditions have been noted (for instance, ante-partum haemorrhage), this only "explains" what has happened, and not being of immediate practical importance, does not concern us here.

Placental Size

Of more relevance is placental size. Again, there is a great deal of controversy over the accuracy

and usefulness of weighing the placenta (Fox, 1966). The small placenta may explain a small baby, or it may not. Suffice it to say that a lot is made of feto-placental weight ratios, and useful as this maybe for studying the biology and the pathology of pregnancy, its practical value is strictly limited. If this measure is desired, it should be performed under standard conditions; ideally, within *a few minutes* of delivery, after cutting the cord short of its insertion, and trimming the membranes off.

I will come back later to the trimmed cord and membranes, as there is important information to be gleaned from these.

Going back to placental size, the important placenta is the large placenta. Arbitrarily placed at 1 kg., any placenta that is over 1/3 the weight of the fetus near term, should be looked at with suspicion. The pale, bulky placenta has traditionally been considered as associated with syphilis and with rhesus incompatibility, and is a good warning sign for these conditions if they have been missed during the antenatal period. The rhesus incompatible baby born with this kind of placenta is likely to be critically ill. There are other conditions which may cause a large placenta. These include triploidy (Hecht, 1963), diabetes mellitus (Benirschke & Driscoll, 1967), thalassaemia (Lie-Injo, 1967), congenital nephrosis (Kouvalainen, 1962), toxoplasmosis (Benirschke and Driscoll, 1967), and chorangioma, whether diffuse (Potter, 1961) or localised (Sen, 1970).

In this latter condition the shunting of the fetal circulation and the presence of fetal artery thrombosis is often associated with a neonate in heart failure. The oedematous baby maybe mistaken for a hydropic one, or the cause of cardiac failure assumed to be due to a congenital cardiac lesion, and one may fail to take the necessary steps in therapy which would lead to a rapid recovery of the baby. Hydramnios is common.

Thus it is seen that when a placenta is pale and large, there is good reason to examine the placenta histologically, and to check the mother in detail for a number of conditions which may require either mother or baby to be placed under special care.

Membranes

We have already discussed examining the membranes for completeness. I mentioned that blood vessels running across to end blindly on the membranes indicate retained cotyledons. But if you see the blood vessels ending blindly, or torn, at the edge of the hole through which the baby was delivered, get back to that baby fast, for it is in grave danger of dying of fetal exanguination if it has not died already.

This danger also exists if there is velamentous insertion of the cord and the blood vessels running across the membranes may be torn or compressed. Another not well recognised danger associated with a velamentous insertion of the cord is placental insufficiency causing foetal death, but as this is a diagnosis made only in retrospect it will not be considered here except to note that this is not usually a recurrent cause of stillbirth, and the mother may be reassured accordingly.

Another warning sign of fetal exanguination is the sharing of the circulation between twins. While the suspicion of "twin transfusion syndrome" – with one baby overloaded and the other baby exanguinated should be roused by the appearance of the babies at birth, occasionally injection studies showing a "common villous district" or arterial anastomosis point out to the cause of failure to thrive of a set of twins: the one needs venesection and digitalis, the other a blood transfusion. The association of hydramnios with this has been remarked upon by many workers (Benirschke & Driscoll, 1967).

The membranes should be bright and shiny. Dull membranes should have (i) a strip cut off and put into fixative for histology, and (ii) a swab taken and plated for bacteriology. This is of course for the amnion, lining the fetal side of the placenta and membranes. The chorion is naturally dull.

While the occasional scattering of white cells across the membranes is often seen, this usually indicates that the membranes have been ruptured for some time: even labours induced by artificial rupture of membranes can, to some extent, be spotted in this way. The significant case is one where the membranes are dull and the liquor dirty looking. This associated with an intense polymorphonuclear leucocytic infiltration of the membranes would make it imperative to check the condition of the neonate. If it is not thriving, this maybe due to intrauterine pneumonia, septicaemia, or incipient meningitis.

Umbilical Cord

Another site which can be examined for evidence of infection in the newborn is the umbilical cord. Sections of this showing polymorpho-nuclear leucocytic infiltration indicates intra-uterine infection (Pryse-Davies *et al.*, 1973; Meyer *et al.*, 1968). Unfortunately there is seldom an indication to send a section of cord for histology at birth, and so this usually becomes a post-mortem diagnosis in institutions which do examine placenta and fetus together at postmortem, rather than being a pointer during life to why a baby is not doing well.

The cord is however, often ritually examined macroscopically at birth: while looking for such things as true or false knots, cord round the neck, etc., many units do look at the cut section for the presence of a single umbilical artery. This has for a long time been recognised as being associated with a higher incidence of congenital malformations. Recent studies (Froelich & Fujikura, 1966; Bryan & Kohler, 1974) have indicated a high stillbirth and perinatal loss even where there were no detectable congenital malformations. If babies survive the neonatal period, Froehlich and Fujikura (1973) found only an increased incidence of inguinal herniae of 1 in 20: no other significant defect.

Histology

So far we have called upon the use of histology primarily for confirmation of intrauterine infection and the presence of chorangioma; is there any more *practical* information that can be obtained by histological examination?

To continue the question of intrauterine infection, the presence of endarteritis in villous blood vessels would be a warning sign to look for syphilis. A characteristic histologic picture microabcesses with clusters of polymorphonuclear leucocytes in typical clusters and characteristic small gram positiv organisms seen in the placenta is almost certain to be indicative also of fetal infection – by listeria monocytogenes.

Similar lesions are seen with congenital vaccinia. However, in these, focal villous necrosis is also to be seen, round which the polymorphonuclear leuco cytes aggregate.

Placentitis is also to be seen with rubella, toxoplasmosis, and in the very rare occasions when the fetus is affected by candidiasis.

Diseases and infections which have been diagnosed because the pathologist has examined the maternal cells in the intervillous space include malaria, trypanosomiasis and sickle cell disease.

Benirschke & Driscoll (1967) review extensively this topic and the literature thereof.

Electron Microscopy

As an example of pure speculation, it has been suggested (Sen, 1974) that it may become possible to predict malignant potential of placental trophoblast (especially molar) by examining under the electron microscope.

As I have pointed out, it is not the purpose of this paper to discuss all the knowledge available on the placenta: in the last decade, at least six books published on the subject do not overlap in anyway in the formation offered. Nor is it the purpose of this paper to discuss the information obtainable from the placenta which may explain the mode of death of either a stillborn baby or a neonatal death: I have therefore chosen not to discuss those placental lesions, gross or microscopic, which have been so extensively described elsewhere (Fox, 1971, among others) though some of these changes are being studied in "biopsy" specimens from the pregnant women (Aladjem, 1969), and I have not explained how it is that half the placentae labelled "infarcted" are not infarcted: there are half a dozen other lesions which mimick an "infarct" and need histological confirmation. Nor why placental "calcification" is usually a very healthy sign.

It should however be noted that this kind of information can be of intense practical value: it may indicate whether the cause of death is a recurrent one or not, and thus give the clinician information which is of use either to reassure the sorrowing couple or help in the management of the next pregnancy. It is thus mandatory to realise that when a neonate dies, the postmortem study is not complete without a study of the placenta.

If, after reading this article, when you next cup the placenta in your hands, murmur the magic word "complete" and aim it for the bin labelled "for the roses", you hesitate a moment, the purpose of this paper will have been served.

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