THE PLACENTAL TRANSFER OF GASES AND FIXED ACIDS

BY

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The possibility of obtaining intervillous blood from pregnant women has made it feasible to study the placental exchange of various substances. Those which are perhaps most vital, i.e. oxygen and carbon dioxide, have already been studied to some extent (Prystowsky, 1957, 1958, 1959, 1960; Prystowsky, Hellegers and Bruns, 1960; Walker, 1959; Sjöstedt, Rooth and Caligara, 1960; Rooth, Sjöstedt and Caligara, 1961b). It is the purpose of the present paper to give a more complete picture of the placental transfer by describing both the transfer of the blood gases and the fixed acids.

Material and Methods

In 10 cases the intervillous space was punctured not earlier than three days before onset of labour. The technique has been described earlier by Sjöstedt et al. (1960).

In 22 cases measurements were done on cord blood. All were spontaneous vertex deliveries. In 18 cases the infants showed no sign of asphyxia, while in four cases such signs were slightly present. The cord was clamped in two places as soon as it presented itself and before the onset of respiration. The cord was rapidly brought from the delivery room to our laboratory where the blood was drawn into heparinized syringes, and the analyses were immediately initiated. Haemoglobin, oxygen tension (pO2), pH, carbon dioxide tension (pCO2), buffer base (BB) and base excess (BE) as well as lactic acid were measured in the samples.

Haemoglobin was determined as oxyhaemoglobin (Rooth and Sjöstedt, 1957). pO2 was measured polarographically by a system similar to that described by Rooth, Sjöstedt and Caligara (1959b). However, instead of a lucite cuvette, one of nickel-plated brass was used, which reduced the time for the measurements to 45 seconds. The reliability of the method was as good as before. pH was measured with a radiometer type 22 pH meter and based on N.B.S. standards. pCO2 was measured according to Severinghaus and Bradley (1958).

The lactic acid was determined by a modification of the Barker and Summerson (1941) method which only needs 0.1 ml. of blood (I. H. Kaiser, personal communication, 1959).

In order to obtain the necessary figures for calculating the intervillous oxygen loss and carbon dioxide and fixed acid transportation, data from two further series were used.

In 16 pregnant women without signs of heart disease, pulmonary disease or toxemia, arterial oxygen tension was measured with the technique described by us in 1959 (Rooth et al., 1959). The women were all in the 37th to 42nd week of pregnancy. The same analyses were performed on 11 non-pregnant women of the same age group. The arterial punctures were done in the brachial artery with the women sitting.

On another group of 85 pregnant women between the 37th and 43rd week, arterial pH, pCO2, BB and BE were measured with the Astrup microtechnique using capillary blood (Sjöstedt, 1962).

The oxygen content of whole blood (cO2) was obtained from the amount of dissolved oxygen and from the oxygen saturation, which was calculated from the oxygen tension and the pH using the appropriate oxygen dissociation curves, that of Bartels and Harms (1959) for adult blood and that of Rooth, Sjöstedt and Caligara (1959a) for foetal blood.

The CO2 content of whole blood (cCO2) was obtained in the following way: from the diagram of Siggaard Andersen and Engel (1960), the serum bicarbonate content was calculated. To this was added the dissolved CO2 which gave the total serum CO2. With due attention to the necessary corrections for unsaturated haemoglobin, the cCO2 was obtained from the nomograph of Van Slyke and Sendroy (1928).

The discussion on the metabolic acid base balance will be limited to BE as we, in agreement with Astrup, Jørgensen, Siggaard Andersen and Engel (1960), feel that the metabolic acidosis or alkalosis is best expressed in terms of BE which has the advantage of being independent of haemoglobin concentration.

Results

The mean of the measured or calculated values for pH, Hb, pO2, pCO2, cCO2, BB, BE and lactic acid in the umbilical vessels and from the intervillous
space is given in Table 1. The mean arterial
pO₂M in the pregnant and non-pregnant women is
given in Table 2 and the mean values for arterial
pHₓ, pCO₂M and BEₓM of the pregnant women are
given in Table 3. In the following, pO₂M means
pO₂ in the maternal blood, pO₂F means pO₂ in the
foetal blood, etc.

The mean of the individual differences (Δ)
between the carbon dioxide content of the umbilical
artery and vein as well as the corresponding differ-
ences (Δ) for base excess, total acids and total
oxygen content are given in Table 4.

**Discussion**

The figures here presented make possible a
quantitative description of the interrelation between
the gas and acid transfer across the placenta. Before
this is done it is necessary to discuss the limitations
of the measurements and to compare the present
figures with those of other authors.

**Umbilical Cord Blood and Intervillous Blood.**

pH, pO₂, pCO₂ and Hb are essentially the same as

in our earlier studies (Sjöstedt et al., 1960; Rooth
et al., 1961b) and the reader is referred to these for
comparisons of the relevant literature. To these
should be added the work by Vasicka, Quilligan,
Aznar, Lipsitz and Bloor (1960), who found lower
pO₂ values in the intervillous space (33 mm. Hg)
and consequently also in the umbilical vein (21 mm.
Hg) and in the umbilical artery (10 mm. Hg). This
discrepancy is possibly due to the different treat-
ment of the patient in premedication and anaesthesia.
The BE values in the present study are more reliable
than those in the earlier publications where Hb was
not measured. The mean of our normal cases is
-6.2 mEq/l. in the umbilical vein and -9.4 mEq/l. in
the umbilical artery. The difference of the mean
is 3.2 as against 3.9 in the earlier study. The
difference, however, is not affected by the Hb levels,
and the discrepancy is therefore an indication of the
variations in the material and the errors in the
measurements.

The fact that metabolic acidosis occurs in the
umbilical vessels is well known from Yllpö (1916)
and several subsequent studies (Österlund, 1955). As these authors only gave figures for pH and pCO₂ or at best for CO₂-combining power, their results are not directly comparable with the present material. Only Beer, Bartels and Raczkowski (1955) give sufficient data for a calculation of BE. When this is done BE is found to be -4.8 in the arteries (nine cases) and -3.4 in the veins (11 cases). This discrepancy may be due to the fact that these authors calculated the pH.

James (1959) finds a mean BB of 39 in the umbilical arteries of vigorous infants (26 cases) and of 37 in mildly depressed infants (six cases). In another series James, Wisbrot, Prince, Holaday and Apgar (1958) find a BB of about 41 in the arteries and almost the same value in the vein. The small arteriovenous difference would indicate no passage of fixed acids. This may be because the means used are obtained from different cases, as it has not been possible to measure BB in blood from the arteries and vein of the same cases except in a few instances. However, these BB levels agree with ours.

According to our figures the lactic acid concentration is highest in the umbilical artery, lower in the vein (Δ lact 0.5 mEq/l., p < 0.01) and lower still in the intervillous blood. The studies of lactic acids have been discussed in detail by Vedra (1959). To sum up, our results agree with those of Bell, Cunningham, Jowett, Millet and Brooks (1928), Kaiser and Goodlin (1958) and Hendrichs (1957). Eastman and McLane (1931) and Vedra (1959, 1960) find higher values in the maternal blood than in the foetal blood, but all the authors cited have measured the maternal venous blood and not the intervillous blood. Vedra finds a difference between the umbilical artery and the umbilical vein which is on the 5% level, but which he doubts. Eastman and McLane (1931) only find a difference between the umbilical vessels in cases of asphyxia.

During labour the mother has a higher lactic acid content than before (Kaiser and Goodlin, 1958), and it is possible that in a non-steady state there is a net passage of lactic acid from the mother to the foetus. Friedman, Gray, Grynfogel, Hutchinson, Kelly and Plentl (1960) studying the rhesus monkey have given interesting information about the transfer of lactic acid between the foetus, the amniotic fluid and the mother. In the amniotic fluid we have found a mean value of 8.5 mEq/l. of lactic acid in eight uncomplicated cases punctured before onset of labour. This is the same level as found by Hendrichs (1957), and the high concentration probably derives from the urine of the foetus (see also Rooth, Sjöstedt and Caligara, 1961a; Sjöstedt, 1962). Their results indicate that regardless of the concentrations there is a continuous interchange of lactic acid between the foetus and the mother.

**Arterial Blood of the Mother.** We find no significant difference in arterial oxygen tension between non-pregnant and pregnant women. The small difference observed agrees with that of Rossier and Hotz (1953) who measured oxygen saturation. On the basis of the work of Rossier and Hotz (1953), Beer et al. (1955) calculated that the arterial pO₂ of the pregnant women was 110 mm. Hg. Our mean value of 97 is the same as that of Vasicka et al. (1960).

The pCO₂ of the pregnant woman is known to be low, since the original publication of Hasselbalch (1912). The present figure of 31 mm. Hg taken on capillary blood agrees with that of Rossier and Hotz who found 33 mm. Hg in the brachial artery and Boutouline-Young and Boutouline-Young (1956) who found 30-32 mm. Hg in the inspiratory air.

As the pregnant women have a low pCO₂ and a high pH they have a respiratory alkalosis.

Whether the pregnant woman has a metabolic acidosis or not has been the subject of some discussion. The early literature on this subject is surveyed in detail by Rossier and Hotz (1953) and Österlund (1955). The reduction in the CO₂ combining power observed by Darling, Smith, Asmussen and Cohen (1941) has led to the general opinion that there is a metabolic acidosis. The present figures (Sjöstedt, 1962) indicate only a slight metabolic acid-base change.

**Effect of Non-simultaneous Measurements and of Analyses on Different Groups of Cases.** As complicated deliveries are different and caused by a variety of reasons, it must be extremely hazardous to compare intervillous maternal or foetal umbilical blood from one complicated case with another, but in the case of normal conditions such a comparison should not introduce a major error. It is of greater importance that the intervillous blood samples have always been taken before the onset of labour and the umbilical blood has always been taken after the delivery.

It is likely that were the intervillous blood taken at the time of delivery its lactic acid content would be higher than we have found, but it would hardly be as high as in the umbilical artery. Concomitant with an increased lactic acid content, we would find a lower value for our BE. It would probably be at least 1 mEq/l. lower.

It is also possible that the pCO₂ of the intervillous space during delivery is intermittently lower than
that observed by us because of hyperventilation by
the mother. This, however, will hardly be a constant
finding.

The oxygen in the intervillous space does not
seem to be affected by the delivery. Prystowsky
(1959), who has taken his intervillous samples
immediately before delivery, gives the same mean
pO2 as we have found before the onset of labour.
The net effect, if the samples had all been taken
at the time of delivery, would be a slight decrease in
the BE of the intervillous blood.

The calculation of the ΔO2M for the maternal
blood is not influenced by the fact that the arterial
pO2M was measured on a different group of subjects
from the intervillous pO2M. A small difference in
arterial pO2M would not be noticeable in the results.
The pH, pCO2 and BE measurement of the arterial
blood in the pregnant woman is of more importance.
If the intervillous blood has a lower pCO2M during
delivery because of hyperventilation, this of course
is due to a low arterial pCO2M and the ΔCO2M
would be lower. If the mother has an increased
concentration of lactic acid, the BE M of the arterial
blood will also be lower and the BE M will at least
not be higher than found.

This discussion of the errors has shown that the
analyses of the heterogeneous maternal samples
taken at different times is correct as far as oxygen is
concerned but is probably somewhat inaccurate as
far as pCO2M, cCO2M, and BE M are concerned. The
foetal blood studies which were all done on samples
taken at the same time do not have these errors.

A calculation of the total exchange of the foetus
gives the values shown in Table 4. The ΔO2F is
3·6 mEq/l. If the foetal placental exchange is not
impaired, the total acid excretion should equal this,
and we found a mean value of 3·6 mEq/l. of ΔCO2F
and ΔBE F together. This shows that our normal
foetuses have been in a steady state. The four
distressed cases all show lower values for oxygen,
the acid excretion is normal in three cases and
considerably elevated in one case.

The important point in this connexion is that the
present study has shown that the figures obtained from
cord blood taken immediately after delivery are
representative of the intrauterine conditions, as, for
reasons given earlier (Sjöstedt et al., 1960; Rooth
et al., 1961b), the values cannot be higher for pCO2F
and pHF and not lower for pCO2F, and furthermore
the balance studies show that the foetuses are in
a steady state.

As regards the total acid excretion, the present
study gives rise to interesting speculations, but
because of the indirect way of measuring the CO2
content, we feel that for the moment not too much
attention should be focused on the relative distribu-
tion of the components making up the carbon excre-
tion. As will be seen in Table 4, the ΔCO2P is only
0·6 mEq/l. In fact, in six of the normal cases and
in two of the asphyxiated cases, the CO2P content
was higher in the umbilical vein than in the artery
due to the higher pH and resulting higher CO2P
binding capacity. These results were unexpected,
although Vedra (1959) finds a Δ bicarbonate plasma
of 0·7 mEq/l. which would probably correspond to a
ΔCO2P of about 0·5 mEq/l. Österlund (1955) also gives a Δ alkali reserve of
2·4 mEq/l., which is twice that of Beer et al. (1955)
and probably corresponds to a ΔBE of 3 mEq/l.
These results are opposed to those of, for instance,
Haselhorst and Stromberger (1930) and Beer et al.
(1955). The latter find a mean ΔCO2P of 2·8 mEq/l.
and a ΔBE F of 1·4, i.e. a total Δ acids of
4·2 mEq/l. Thus, although the total Δ acids are of
the same magnitude, the ΔCO2P and ΔBE F differ greatly. This point calls for further investiga-
tion because if our figures are correct they give
important information about the foetal metabolism.
As we obtained the same ΔBE F in both the present
study and in our earlier one (Rooth et al., 1961b) it
will be necessary to do direct measurements of the
CO2 content in future.

In the corresponding maternal balance the ΔO2M
is 1·9 mEq/l., ΔCO2M 1·5 and ΔBE M 2·7. Here
we find an R.Q. of 0·8 which seems not unexpected,
but the total amount of acid is higher than that of
the corresponding amount of oxygen. If indeed
we were to take for comparison figures for BE M
from our earlier study (Rooth et al., 1961b) the
oxygen and carbon dioxide values would be similar
but the BE M would be smaller and a balance
would be obtained. This shows that the studies
have to be done on the same patients, as we have
indicated above.

However, as the oxygen figures will not be affected
by this error, it is interesting to see what information
we get by comparing the maternal and foetal CO2.
If we assume that the maternal CO2M is not affected
by the oxygen consumption of the uterus itself,
the ratio of ΔO2F/ΔO2M equals the ratio of maternal
placental blood flow to foetal placental blood flow
and is 1·8·1. Were the magnitude of either the
foetal or the maternal blood flow known, the other
could then be readily calculated and so too could
the foetal oxygen consumption.

The figures available for the human uterine blood
flow have been reviewed by Metcalfe, Romney,
Swartwout, Pitcairn, Lethin and Barron (1959) and
given as 150 ml./kg./min. The foetal blood flow
would then be 80 ml./kg./min. In a foetus of

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3.5 kg. This would correspond to a foetal placental minute volume of 0.3 l. The oxygen consumption would be 2-4 ml/min.

As the present study has led to figures for the oxygen consumption and carbon dioxide production of the foetus as well as the magnitude of the foetal versus maternal placental circulation, and also gives some idea as to the metabolism of the foetal tissues, it seems that they should be repeated with as many simultaneously performed analyses as possible. This, however, meets with considerable clinical difficulties.

Summary

The blood from the umbilical vein and artery has been studied in 18 normal deliveries and in four cases where the infants were mildly asphyxiated.

Figures are given for the oxygen tension, carbon dioxide tension, oxygen content, carbon dioxide content, pH, base excess and lactic acid. In 10 cases intervillous blood was obtained before delivery and analysed.

The foetus receives 3.6 mEq/l. of oxygen and eliminates 3.6 mEq/l. of total acids.

This balance shows that the foetus, at the time when the samples were taken, i.e. immediately after delivery, was in a steady state. This, together with results drawn from the intervillous measurements, shows that the analyses of cord blood presented by us, here as well as earlier, are representative of the intrauterine conditions during the last period of pregnancy.

The data presented give a physiological picture of the gas exchange levels of the foetus and give figures for the calculation of the ratio between the foetal and maternal placental blood flow. The foetal placental minute volume is about 0.3 litre.

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References


