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EFFECT OF UTERINE CONTRACTIONS ON MATERNAL BLOOD FLOW THROUGH THE PLACENTA[1]

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The maintenance of normal fetal homeostasis is basic to cellular growth and development. Fetal homeostasis is largely dependent on metabolic exchanges with the mother through the placenta. A reduction in these exchanges causes a diminution in the supply of anabolites to the fetus and also a retention of catabolites, with several harmful consequences such as acidosis.

Insufficient feto-maternal exchanges may be produced by several factors. Uterine contractions are the most important cause in reducing the blood flow through the intervillous space of the placenta (IVS) because they are always present in labor (other causes, if present, add their effects to that of the contractions), because they may in themselves produce fetal distress, because they act through different mechanisms that potentiate each other, and because they are often iatrogenically augmented by the administration of oxytocic drugs.

Mechanisms of action

Uterine contractions reduce the maternal blood flow through the placenta by means of at least two mechanisms: the compression of the intramyometrial vessels and the compression of the aorta and iliac arteries by the contracting uterus.

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[2] Presented by Dr. Poseiro.

Compression of the intramyometrial vessels

The placental blood flow is a function of the difference between the mean arterial blood pressure and the intramyometrial pressure. It is directly proportional to the mean arterial pressure and indirectly proportional to the resistance of flow. This relationship can be expressed by the equation

$$\text{Placental blood flow} = K\frac{\text{perfusion pressure}}{\text{resistance opposed to blood circulation}}$$

Figure 1 illustrates the pressure system when the uterus is relaxed. Under these conditions

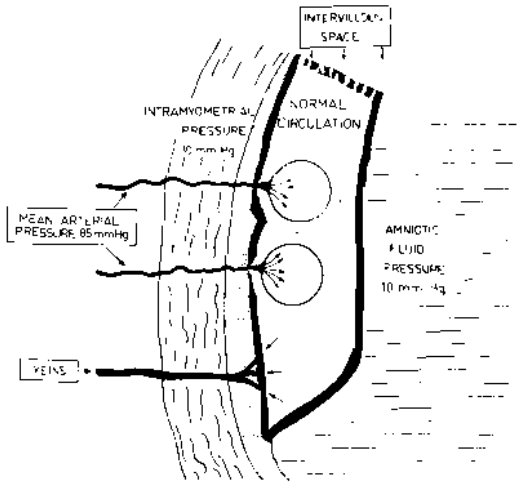

FIGURE 1. Schematic representation of circulatory conditions when uterus is relaxed. Blood circulates freely through IVS (*17*).

the amniotic pressure is around 10 mm Hg; the intramyometrial pressure is also low, 8–10 mm Hg (see also Figure 2); the blood in the arteries crossing the myometrium has a mean normal pressure of 85–100 mm Hg and has no difficulty in entering the IVS, circulating through it, and leaving via veins that have a pressure of 8 mm Hg.

When the uterus contracts, the circulatory conditions are completely different. Caldeyro-Barcia *et al.* reported in 1952 (*12*) that during contractions, even in normal conditions, the intramyometrial pressure reaches values two to three times higher than the amniotic pressure (Figure 2). The muscular fibers act like sphincters completely surrounding the intramyometrial vessels and may even occlude them.

The following equation may be used instead of the one above:

$$\text{Placental blood flow} = K_1 \frac{\text{perfusion pressure}}{\text{intramyometrial pressure}}$$

From these results Caldeyro-Barcia postulated in 1956 (*15*) that each contraction apparently reduced or even suppressed the maternal blood flow through the IVS (Figure 3). When the contraction starts, on its ascending branch, the first vessels to undergo this compression are the veins that drain the IVS; the blood accumulates backward, and the arteries (having a higher pressure) continue to supply blood to the IVS. Later, when the contraction exerts a greater pressure, the arteries are also compressed. The compression pressure exceeds the mean arterial pressure; at this stage the blood neither enters or leaves the IVS. As a consequence of this circulatory stasis, the blood in the IVS lacks oxygen and anabolites, which are continuously being used by the fetus, and at the same time accumulates catabolites, which are continuously being produced by the fetus. Hypoxemia, hypercapnia, and acidosis are produced both in the IVS and in the fetal blood (*17*) (Figure 3).

Figure 4 schematizes the relationship between the pressures. It has been assumed that the mean arterial pressure is 100 mm Hg and does not change during contractions (this is not entirely true). Each uterine contraction is represented by the value of its corresponding intramyometrial pressure. The placental flow at a given time (in the figure, periods of 10 minutes) is proportional to the dotted area between the two pressures. Five situations are represented. Even in normal conditions there are about 15 seconds at the top of the contraction during

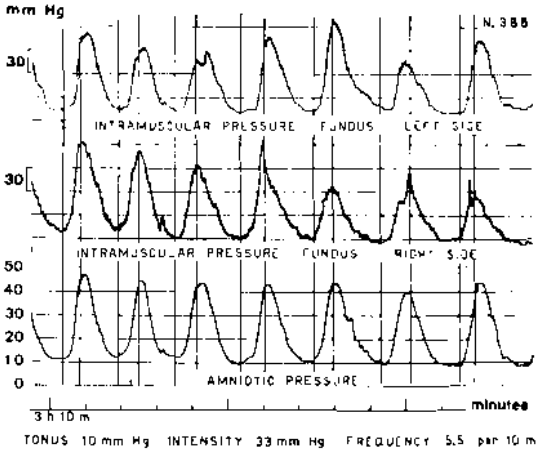

FIGURE 2. Records of intramyometrial pressure obtained with microballoons inserted into thickness of uterine wall. Contractions cause much greater rise in intramyometrial pressure (60–120 mm Hg) than in amniotic pressure (35–40 mm Hg). Intramyometrial aspect of vessels supplying IVS is exposed to intramyometrial pressure, which during contractions becomes high enough to occlude them (*16*).

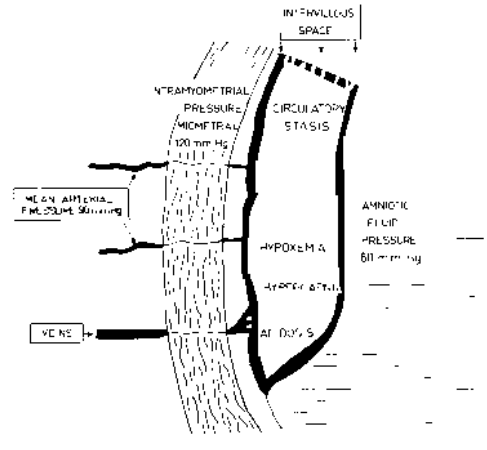

FIGURE 3. Circulatory stasis when the uterus is contracted (*17*).

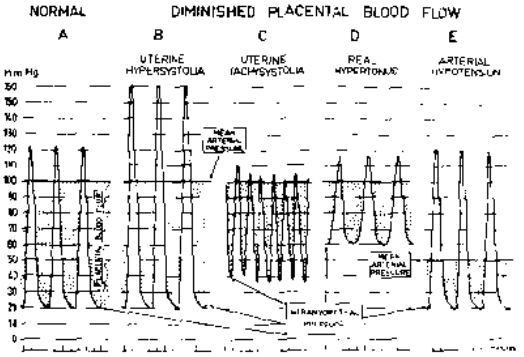

FIGURE 4. Placental blood flow (dotted area) is function of difference between mean arterial blood pressure and intramyometrial pressure. Dotted area is markedly reduced in B to D because of abnormal uterine contractility and in E because of maternal arterial hypotension. (Highly schematic.)

which the intramyometrial pressure is higher than the mean arterial pressure and thus no blood is circulating. We postulate that this is the mechanism by which each uterine contraction causes a transient episode of fetal hypoxia (see below). When abnormally high uterine contractility exists (Figure 4B, C, and D) the circulatory stasis is more marked and its consequences are more serious. These abnormalities in contractility can in themselves be a definite cause of fetal distress (*53*). It must be borne in mind that while here they are represented singly, they can occur in combination and thus worsen the condition of the fetus (*23*). An additional cause of fetal distress, under certain conditions, may be maternal arterial hypotension (*51*) (Figure 4E).

The intramyometrial vessel compression has been demonstrated by several methods:

1. *Clearance of radioisotopes injected in the IVS.* In 1953 Browne *et al.* (*11*) reported the first results injecting ^{24}Na. A lot of work has been done up to now (*39, 40, 41, 43, 45, 66*), but little evidence of the effect of uterine contractions has been reported. In a recently published thesis Lagorce (*42*) made a good review of the literature and documented very well the study of uterine and placental blood flow with Xenon 133. He did not study the effects of uterine contractions but promised to do so in the near future.

Caldeyro-Barcia in 1956 (*14, 17*) and Poseiro in 1958 (*50*) studied the influence of uterine contractility on the flow of maternal blood through the IVS. Three phenomena were recorded: maternal arterial pressure, amniotic pressure, and the clearance of a radioisotope, ^{131}I, injected in the IVS. This experiment has been performed in cases of intrauterine fetal death because uterine hypercontractility was induced and in order to avoid any passage of radioisotopic material into the fetal circulation that would interfere with the results.

Figure 5 shows a typical result of this study. In the upper part of the figure the arterial pressure was normal and uterine contractility had values corresponding to those observed in the days preceding labor. Under these conditions 2 microcuries of ^{131}I were injected into the IVS. Placental radioactivity increased rapidly and then decreased, following an exponential curve that reached the background line in one and a half minutes. The half period (T ½) was 30 seconds. In the lower part of the figure, ¼ unit of pituitrin injected intravenously produced a great increase in uterine contractility. A second injection of the same amount of ^{131}I

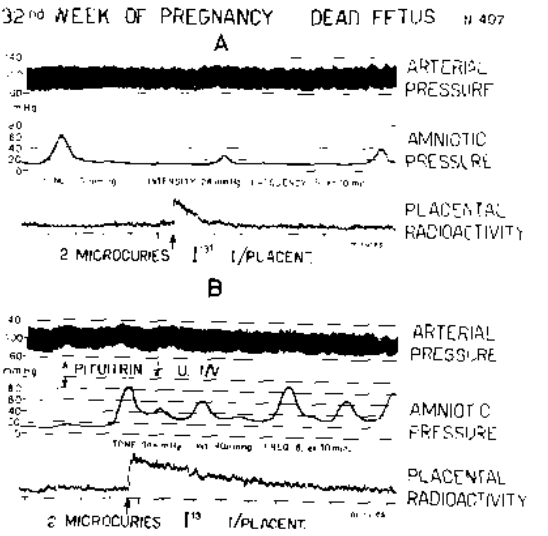

FIGURE 5. Influence of uterine contractility on flow of maternal blood through IVS, studied by clearance curve of radioactive ^{131}I injected in IVS. In A, contractions are normal; in B, hypercontractility was induced by injection of Pitocin (*14*).

disappeared much more slowly than in normal conditions, taking more than 9 minutes to reach the baseline. The T ½ was 144 seconds. Maternal blood flow through the placenta was about five times less during hypercontractility than in normal conditions. We can conclude that uterine hyperactivity markedly reduced the blood flow through the IVS.

2. *Angiographic studies.* Ramsey *et al.* (*56, 57*), using radioangiocinematographic methods in the rhesus monkey, found that during uterine contraction the IVS blood flow was arrested and only restarted when the uterus relaxed.

Using similar methods in pregnant women, Borell *et al.* (*8, 9, 10*) found a suppression or slowing of utero-placental circulation during uterine contractions. Figure 6 shows schematically the results of an angiogram obtained when the uterus was relaxed (*9*). The spurts into the IVS appear in large number—25. When the uterus is contracted the intramyometrial vessels are compressed, and a similar study (*9*) shows a great diminution in the number of spurts; there are 6 completely tinged and 6 incomplete (Figure 7).

FIGURE 6. Schematized results of angiogram with relaxed uterus. Thickness of lines is proportional to diameter of vessels. Number of spurts to IVS (black circles) is high—25 (*8*).

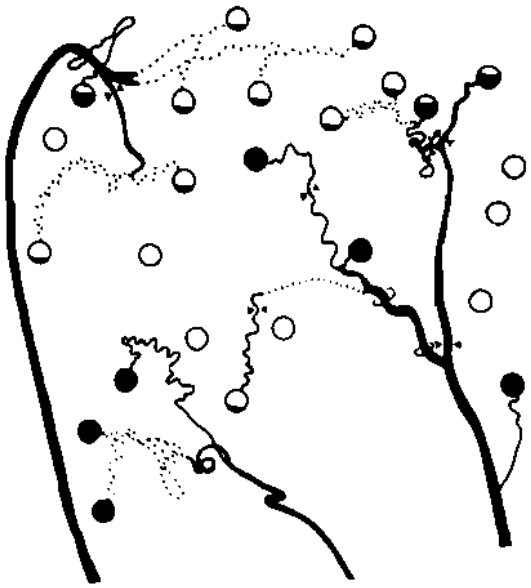

FIGURE 7. When uterus is contracted, number of spurts is markedly smaller than in Figure 6. There are 6 incompletely tinged spurts (incomplete black circles) (*8*).

Bieniarz *et al.* (*6, 7*) used serial abdominopelvic arteriography to visualize the aorta as well as the utero-placental blood vessels in more than 150 women whose pregnancies were past the twenty-seventh week. A catheter was inserted into the aorta through the femoral artery using the Seldinger technique (*61*), similar to that used by Borell *et al.*, Fernström (*29*), and Ramsey *et al.* Four exposures were made in six seconds. Figures 8 and 9 summarize schematically the findings shown by X-ray films taken at different times. The film taken at the sixth second shows (Figure 8, relaxed uterus) a good quantity of the opaque substance in the IVS, whereas that taken at the same time during a uterine contraction (Figure 9) shows none.

3. *Measurements of uterine blood flow.* In 1947 Ahlquist *et al.* (*1*) reported the first direct observation of uterine blood flow during labor in the pregnant bitch. They observed that the flow diminished when the uterus contracted. In 1958 Assali *et al.* (*3, 4*) found in the ewe that each contraction caused a transient decrease of blood flow in the uterine artery, which was recorded with electromagnetic flow-meters. They observed that uterine blood flow decreased

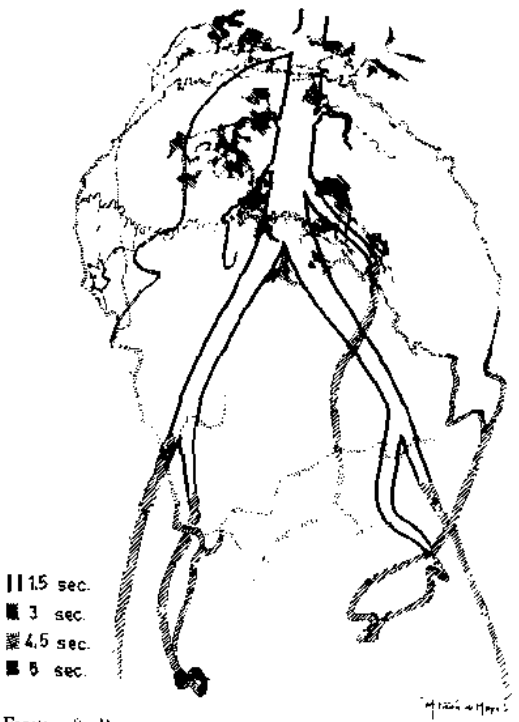

FIGURE 8. Between contractions. Schema of anteriogram in a normal full-term pregnant woman. Superposition of pictures demonstrates progress of dye clearance in sequential exposures of series. IVS shadow is dense, showing good blood circulation (*6*).

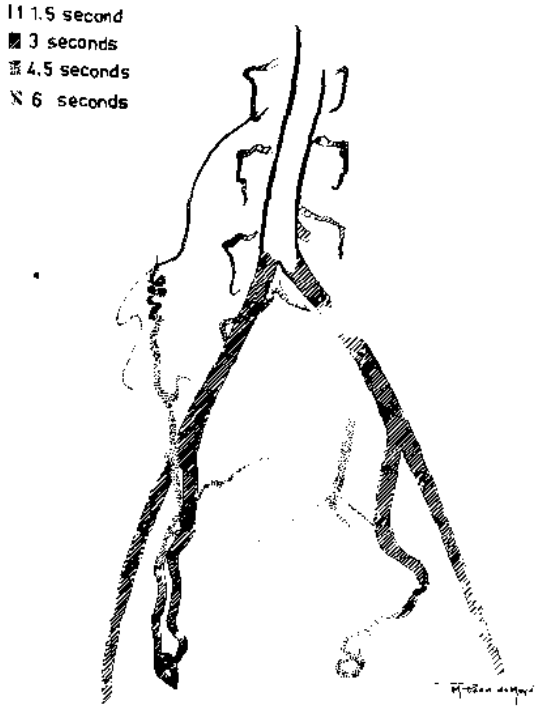

FIGURE 9. During contraction. Same case as in Figure 8. Right uterine artery, which mainly irrigates IVS, is occluded by uterus. IVS does not appear (*6*).

with each contraction and that the degree of flow reduction correlated with the intensity of palpable contractions.

Most recently, Greiss *et al* (*32, 33, 34*) implanted electromagnetic flow probes and occlusion loops around the uterine arteries and the descending aorta. Intrauterine pressure and maternal arterial pressure were measured simultaneously. Figure 10 (*34*) illustrates the effect of a single contraction. The uterine blood flow diminishes gradually as intrauterine pressure rises. The recovery of the baseline of the blood flow is slow, related to the very prolonged descending limb of the contraction wave. The basal flow was reached only when the contraction descended to the normal level of uterine tonus.

Figure 11 (*34*) illustrates the effect of an excessive dose of oxytocin. When intrauterine pressure reached high hypertonus the uterine blood flow decreased to the zero level. Hypersystolic and tachysystolic contractions produced falls in the flow, giving a mirror image of the tracing of the contractions.

Greiss and Anderson (*34*) conclude that "the uterine blood flow varies inversely with the intensity, frequency and duration of uterine contractions and with the level of the tonus." Maternal hypotension due either to hemorrhagic

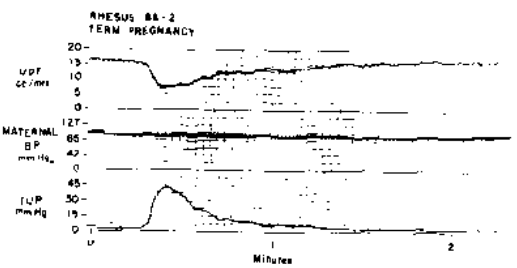

FIGURE 10. Relationship between uterine blood flow and uterine contractions during spontaneous labor in rhesus monkey (*34*).

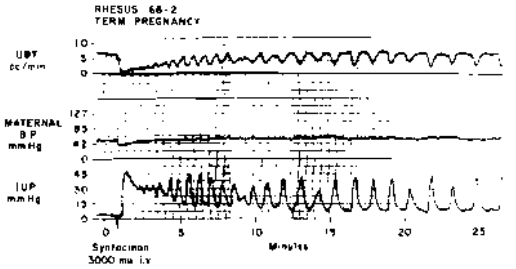

FIGURE 11. Hypercontractility due to excessive administration of oxytocin (lower tracing). Tracing of maternal blood pressure shows small lowering, coinciding with injection of Pitocin. Marked fall in uterine blood flow coincides with hypertonic uterine contractions (*34*).

shock or to sympatholytic mechanisms diminishes the uterine blood flow (*32*).

Compression of the aorta and iliac arteries by the contracting uterus

During labor, when the mother is in the supine (dorsal recumbent) position, the contracting uterus may compress the aorta against the spine, reducing or suppressing the arterial circulation of blood through the occluded vessels.

In 1955 this effect was described (*35*) in about 20 per cent of the records of maternal femoral arterial pressure. Figure 12 shows how the relaxed uterus rests on the main vessels. When the uterus contracts it tends to take a more spherical shape; its antero-posterior diameter augments, compressing the inferior vena cava and the aorta against the spine. Usually this compression occurs at the end of the aorta or at one of its divisions, right or left iliac arteries. The compressive effect was found in 28 per cent of the blood pressures recorded in one femoral artery (*44, 54*). It is possible that this percentage would be greater if the pressure were recorded in both femoral arteries. Each uterine contraction produces a marked fall in the systolic and a less marked one in the diastolic pressure of the femoral artery (Figure 13). The systolic fall is so marked that the pulse pressure disappears and the tracing appears as a line around the diastolic values.

The effect described is strictly local. The tracing of the brachial artery in Figure 13 (*54*) shows no fall in pressure during uterine contractions. The same fact can be observed in aortic tracings, as shown in Figure 16. The compression of the main abdominopelvic vessels can be demonstrated by several methods:

1. *Clinical findings.* As the pulse pressure disappears, clinical palpation of the femoral region can easily detect this phenomenon during contractions and note how the pulse reappears when the contraction ends. It can also be observed by performing the oscillometry in the legs of the parturient.

2. *Angiographic studies.* Figure 14 shows schematically the findings obtained with X-ray films taken at different times in the intervals

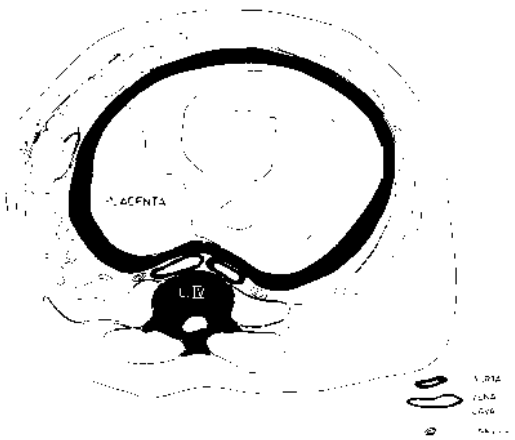

FIGURE 12. Transverse section, modified from Couvelaire's, of 32-week-pregnant woman, illustrating how weight of pregnant uterus may compress aorta against vertebral column just as it obstructs inferior vena cava.

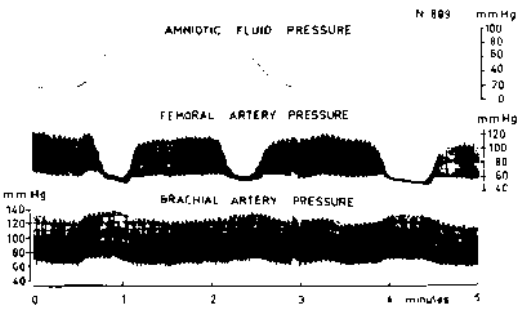

FIGURE 13. Responses to uterine contraction: each coincides with transient fall in systolic and pulse pressure in femoral artery and rise in brachial artery pressure, proving complete separation of the two vascular regions.

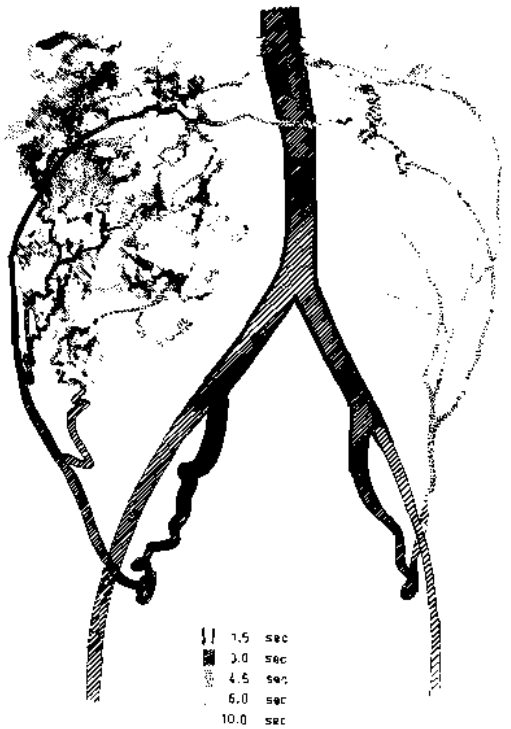

FIGURE 14. Superposition of pictures taken between contractions in angiographic study demonstrates progressive dye clearance through all supplying arteries into IVS.

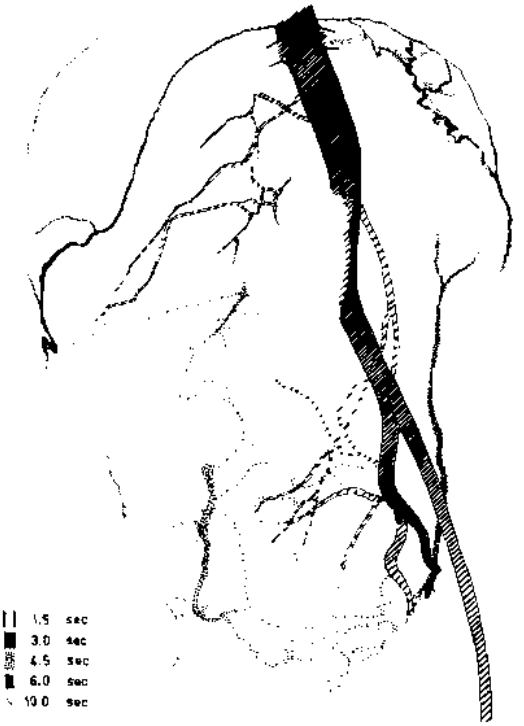

FIGURE 15. During contraction, superposition of pictures in series similar to Figure 14 shows marked obstruction of the aorta and occlusion of right common iliac artery by contracting uterus. Retro-uterine fulcrum (L_{4-5}) is completely ischemic; placental irrigation is deficient despite compensatory retrograde irrigation.

between contractions (*52*). As can be seen, the contrast medium circulates more rapidly through the right artery than through the left. Multiple spurts and good visualization of cotyledonary pools can be seen at the right upper uterine quadrant (films taken at the sixth and tenth seconds). The contraction displaces the aorta to the left (Figure 15), partially obstructing its flow and completely occluding the flow of the right common iliac and consequently of the hypogastric and uterine arteries (*51, 52*). Only the left iliac and uterine arteries maintain the uterine blood supply. Furthermore, the entrance of the vessels into the contracted myometrium is occluded. The effects of the contraction both on the main arteries (X-ray film taken at 1.5 seconds in Figure 15) and on their intramural branches (4.5 to 10 seconds) are additive and no spurts are seen in the placental location.

3. *Hemodynamic studies* (*52, 54, 55*). The same occlusive effect of each contraction that

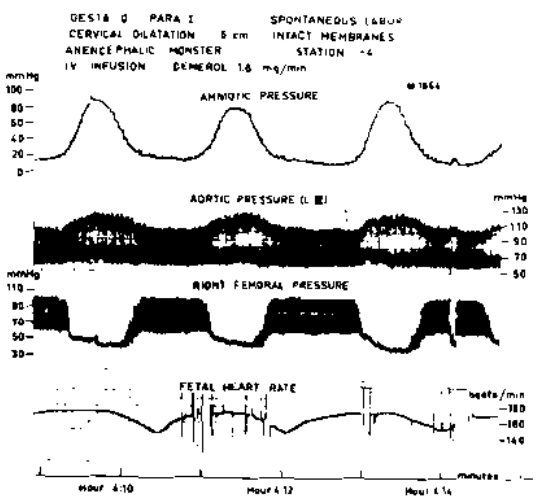

FIGURE 16. Occlusive effect of each contraction recorded in same woman as in Figure 15: transient hypotension in right femoral artery, coinciding rise in aortic pressure, proving complete separation of these vascular regions. Descent in fetal heart rate follows (dip II), related to tidal hypoxia due to interference with placental circulation during contraction.

was visualized in the angiograms of Figures 14 and 15 was observed in the simultaneous recording of aortic and femoral pressures and amniotic pressure (Figure 16). Each contraction is accompanied by a marked fall in systolic blood pressure in the femoral artery, whereas in the aorta a small increase in both systolic and diastolic values is noted (*12, 35*). This demonstrates that the effect is exclusively local. The effect does not appear in the lateral position. In Figure 17, when the patient is in the supine position each contraction causes a fall in femoral pressure and also a dip II (see below); when she turns to the lateral position, similar contractions produce neither of these effects, because the uterus does not compress the main vessels. The utero-placental blood flow is assumed to have increased.

Effect of contractions on fetal oxygenation

Caldeyro-Barcia *et al.* (*22*) postulated in 1961 that the above demonstrated effects of uterine contractions on maternal blood flow through the placenta caused fetal asphyxia. This has been demonstrated by Pose *et al.* (*28, 47, 48, 49*), who performed simultaneous recordings of the amniotic pressure and the partial pressure of oxygen (pO_2) in fetal tissues. They concluded (*48, 49*) that each uterine contraction caused a slow and transient fall in fetal pO_2

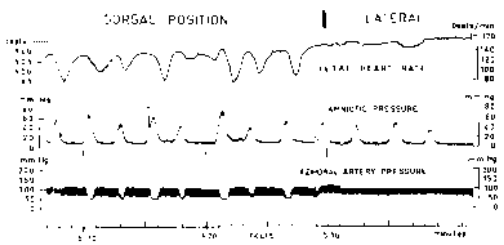

FIGURE 17. With mother in dorsal position, each contraction causes marked Poseiro effect in femoral artery pressure (pressure in uterine artery assumed to have fallen similarly) and dip II of large amplitude in FHR; in lateral position, Poseiro effect disappears and amplitude of dips II diminishes markedly. Record obtained in a severe pre-eclampsia with arterial hypotension, after arterial pressure had fallen because of infusion of Demerol and Chlorpromazine. Loop of cord around neck. Cervical dilatation 3 cm. Intact membranes.

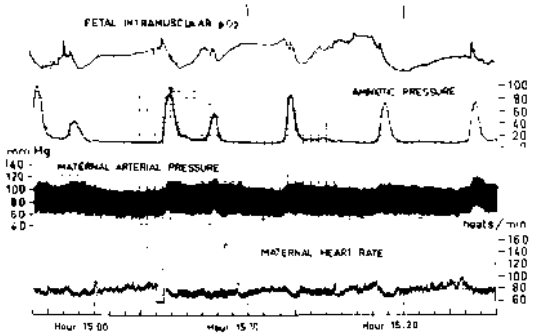

FIGURE 18. Record of fetal muscular pO_2 ($FMpO_2$) obtained with polarographic electrode inserted into buttock muscle of fetus. Each uterine contraction causes a marked fall in $FMpO_2$, starting a few seconds after onset of contraction and reaching bottom 30–45 seconds after its peak. Recovery to initial level after each fall is very slow—2–5 minutes (*49*).

(Figures 18 and 19), which reached bottom about 30 to 45 seconds after the peak of the contraction—a lag-time similar to that of a

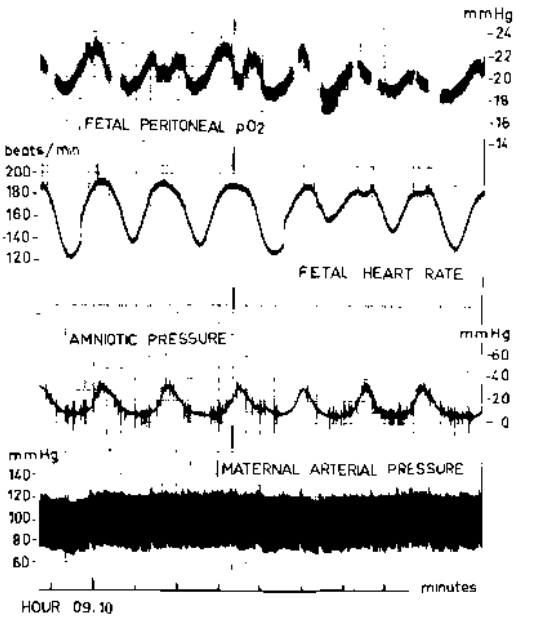

FIGURE 19. Records obtained in full-term (159 days) monkey. Each uterine contraction is followed by slow drop in fetal peritoneal pO_2 and this, after about 10 seconds, by dip II. Intravenous infusion of Demerol at 0.02 mg/kg/min and of oxytocin at 0.1 mU/kg/min. Three doses of 0.2 mg of atropine were given i/m to mother at hours 05:13, 06:33, and 08:40. Artificial respiration with pure oxygen. At time of this record, arterial pressure of the mother was much lower than at beginning of experiment (180/120 mm Hg) (*46, 48, 49*).

dip II (*13, 15, 22, 23, 37, 38*), which is a transient fall in fetal heart rate produced by one uterine contraction (Figures 16 and 19), mainly by hypoxic mechanisms (*22, 23*). These results agree with those of Dawes *et al.* (*27*) obtained in the rhesus monkey. These authors reported that 30 seconds after each contraction, a transient fall occurred in the oxygen saturation of fetal arterial blood; similar effects have been found by Paul (*46*) in the fetal lamb. Saling (*59, 60*) found that a fall in oxygen saturation of hemoglobin with O_2 in fetal capillary blood follows the uterine contraction.

Conclusions

It is concluded that each uterine contraction causes a reduction of maternal blood flow through the IVS and that this is reflected in the internal milieu of the fetus, causing a lowering of the pO_2 of the fetal tissues and a fall in the saturation percentage of hemoglobin with O_2 in fetal capillary blood.

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