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# PRESSURE EXERTED BY UTERINE CONTRACTIONS ON THE HEAD OF THE HUMAN FETUS DURING LABOR<sup>1</sup>

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Ingelman-Sundberg *et al.* (12) and Lindgren (14, 15, 16) have reported that under certain conditions during labor the pressure exerted by uterine contractions on the fetal head may be two to four times higher than that exerted on the amniotic cavity at the same time. Schwarcz and Salaber (18) reported that manual compression of the fetal head through the anterior abdominal wall against the promontorium caused a sudden and marked fall in fetal heart rate (FHR), which was perceived by clinical auscultation—a finding confirmed by many authors and recently studied with greater precision by means of the electronic record of FHR by Hon (10), Chung and Hon (7), and Arellano-Hernández *et al.* (1). These falls in FHR are mediated by the vagus nerve, since they are completely blocked by atropinization of the fetus (17).

During advanced labor, particularly after rupture of the membranes, each uterine contraction may cause a transient fall in FHR (dip I), which is simultaneous with the contraction in such a way that the bottom of the dip I coincides with the peak of the contraction (5). It has been postulated (5, 6) that each dip I is caused

by a strong compression exerted on the fetal head by the corresponding uterine contraction. Cephalic compression would produce vagal stimulation. Dips I have similar characteristics to the "early decelerations" described by Hon and Quilligan (11), who have postulated the same pathogenic mechanism.

The purpose of the present paper is to record and measure the compression received by the fetal head during each uterine contraction, correlating it with the rise in amniotic pressure and the amplitude of the dip I (if present) caused by the same contraction.

## Methods

This study was made in 18 normal, term pregnant women during labor, with vertex presentation. The pressure received by the fetal head during labor is recorded by means of three flat pressure receptors, which are introduced between the uterine cervix and the fetal head (Figure 1) outside the ovular membranes (12, 18, 20).

Each pressure receptor is a flat tambour (Figure 2) formed by two latex membranes, which are glued to the edges of a circular hole on a thin plastic blade. The diameter of each receptor is 15 mm, and the distance between the centers of two consecutive receptors is 30 mm. Each receptor is filled with water and connected to a recording pressure transducer by a thin polyethylene tube. The three re-

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<sup>2</sup>Presented by Dr. Schwarcz.

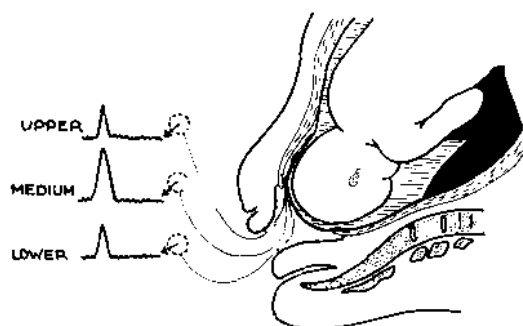


FIGURE 1. Method of recording pressure between fetal head and birth canal with three receptors.

ceptors are centered in line on the blade; they are known as "upper," "medium," and "lower" according to their position in relation to the uterus. The plastic blade—100 mm long, 30 mm wide, and 1 mm thick—is very flexible.

The blade is introduced between the membranes and the uterus toward the fundus (Figure 2). One surface of the receptor touches the lower pole of the amniotic sac (eventually the fetal head) and the other faces the uterine

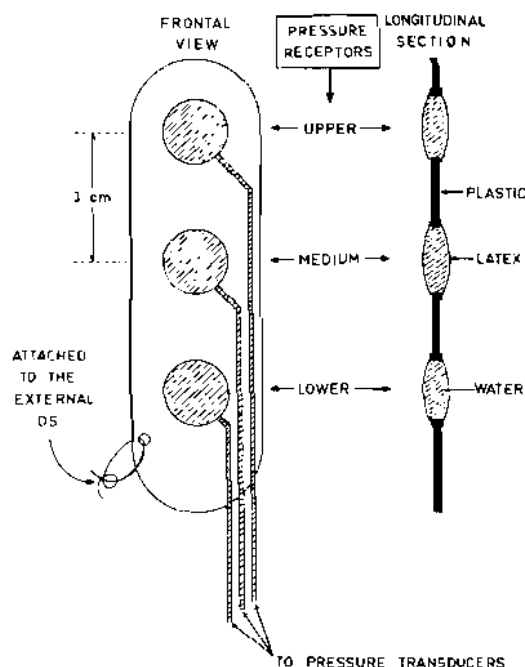


FIGURE 2. Detailed diagram of pressure receptors inserted between fetal head and uterus (see also Figure 1).

wall. The lower end of the blade is sutured to the uterine cervix at the external os (Figures 1 and 2).

The position of the receptors relative to the fetal head is determined by radiology; it changes with the progress of cervical dilatation or with the station of the fetal head. As labor progresses, there is a relative displacement of the blade and receptors from the vertex toward the base of the fetal head (Figures 5 and 7).

The intrauterine (amniotic) pressure is recorded by means of a catheter introduced through the abdominal and uterine walls into the amniotic sac (2) and connected to a pressure transducer. The FHR is recorded by means of an instantaneous cardiometer triggered by the fetal electrocardiogram (3). The ECG is obtained almost free of maternal interference by means of electrodes inserted under the skin of the fetus in either the buttock (3), the scalp, or both (8). The amniotic pressure, the FHR, and the pressures between the fetal head and birth canal are all inscribed on the same recording paper (Figures 3, 4, 6, and 8) to facilitate the study of their interrelations. The fetal EEG is also recorded, as will be reported at this meeting by García-Austt *et al.* (9).

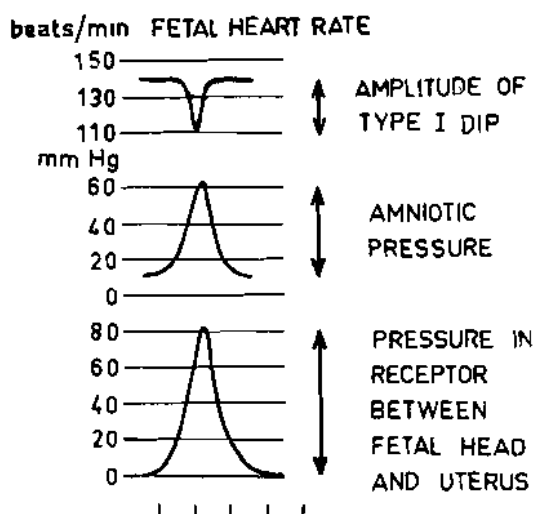


FIGURE 3. Method of measuring amplitude of type I dips and rise in pressure caused by each uterine contraction in several simultaneous tracings.

## Results

### *Correlation between cephalic compression and amniotic pressure*

During each uterine contraction, the pressure recorded by the cephalic receptors rises almost simultaneously with the amniotic pressure (Figures 3, 4, 6, and 8). The amplitude of the rise is measured in every tracing for each contraction (Figure 3). The pressure rise in each cephalic receptor is plotted against the corresponding rise in the amniotic pressure (Figures 5 and 7). For any given period of labor in which the cervical dilatation and station remain unchanged, a given direct linear relationship is found between the pressure in each cephalic receptor and the amniotic pressure (Figures 5 and 7). The correlation coefficients ( $r$ ) are very high—0.95 or more.

When the receptors are displaced and change their position relative to the fetal head (as a consequence of the progress of cervical dilatation or of the station), a new linear correlation is established between the pressure in each cephalic receptor and the amniotic pressure, the value of the intercept and of the regression coefficient changes depending on the new relative position of the receptor (Figures 5 and 7).

When a cephalic receptor is more than 2 cm above or below the equator of the fetal head, the pressure it records at the peak of each uterine contraction is very similar to that in the amniotic cavity (Figures 5 and 7). When the receptor is displaced and comes closer to

the equator of the fetal head, the pressure it records at the peak of the contraction becomes higher than that in the amniotic sac. The ratio (pressure in cephalic receptor/amniotic pressure) is higher than 1 and is significantly different ( $p < 0.001$ ) from that for receptors distant from the equator (Table 1). This is clearly shown in Figures 5 and 7, where the regression coefficients corresponding to receptors near the cephalic equator are higher than those for receptors distant from it. For example, in Figure 5C the lower receptor (at the equator) records much higher pressures than the other two.

The difference in pressure between the receptor near the equator and those distant from it has been found both when the membranes are intact (Figures 5A) and when they are ruptured (Figures 5B and 5C). However, the greatest pressure differences have been observed after rupture of the membranes (Figure 5C). In Figure 8, the pressure at both receptors (medium and lower) is always greater than the amniotic pressure, but the exact relationship between each receptor and the amniotic pressure varies according to the stage considered. In section A, the pressure at the medium receptor is approximately 2.6 times greater. In section B, the pressure at the lower receptor is approximately 2.5 times greater than the amniotic pressure; at the medium receptor it is a little less than at the lower receptor, though still greater than the amniotic pressure. In section C (ending in delivery of the newborn), the pressures at the two receptors are practically identical; both are much lower than those recorded

TABLE 1. Analysis of variance of the ratio between pressure in cephalic receptor\* and amniotic pressure

SOURCE OF VARIATION	SUM OF SQUARES	DEGREES OF FREEDOM	MEAN SQUARES	$F_{(1, 165)} = 7.71$
Between groups	17.7551	1	17.7551	
Within groups				$F_{(1, 165)} = 21.2942$
Between periods	3.3351	4	0.8338	
Within periods	9.6726	164	0.0590	
	30.7628	165		

\* Comparison between data obtained from the receptor near the cephalic equator and those distant from it in record No. 1475, illustrated in Figures 4 and 5.

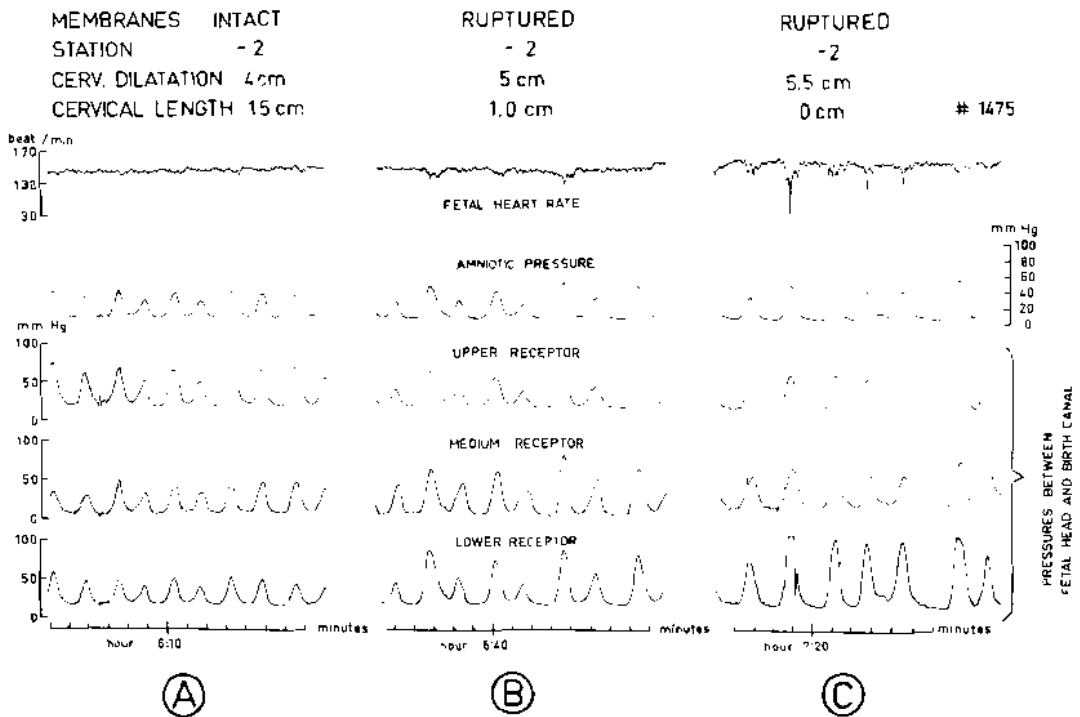


FIGURE 4. Three segments of record obtained during spontaneous labor at term pregnancy (newborn weight 3,320 g, Apgar score 9). Multipara in supine position. Vertex presentation. In A, pressure tracings recorded from receptors placed between fetal head and birth canal are not much different from tracing of amniotic pressure. In B and C, after rupture of membranes, uterine contractions exert much higher pressures on receptors near equator of fetal head (see Figure 5) than on amniotic cavity. Type I dips appear in FHR tracing. Record is quantitatively analyzed in Figure 5.

in section B and only 1.3 times greater than the corresponding amniotic pressure.

These results are interpreted as follows: In the three sections (A, B and C), both receptors are near the cephalic equator and thus record higher pressures than the amniotic. In section A, the cephalic equator is nearer the medium receptor. Upon the descent of the presentation (section B), the cephalic equator gets nearer to the lower receptor, which thus records the highest pressures. When the descent of the head is completed (section C), both receptors are above the cephalic equator; this explains the relative reduction in the pressure they record.

#### *Correlation between cephalic compression and dips I*

If the fetus is not suffering from systemic hypoxia and acidosis, uterine contractions cause

no changes in FHR as long as the pressure received by the fetal head during each uterine contraction is not much higher than the amniotic pressure (Figure 4A). Under these conditions the FHR tracing shows the normal "rapid" oscillations, the baseline is close to 140 beats/min, and there are no dips I.

When the compression exerted by one uterine contraction on the fetal head is much higher than that on the amniotic cavity, a transient fall (dip I) occurs in FHR (Figure 4, sections B and C; Figure 6, sections A and B; and Figure 8). In Figure 6C, uterine contractions cause dips I even though there are no differences between the amniotic pressure and that recorded by the cephalic receptors. However, these receptors are above and distant from the equator (Figure 7C) and thus do not report the maximal pressure received by the fetal head.

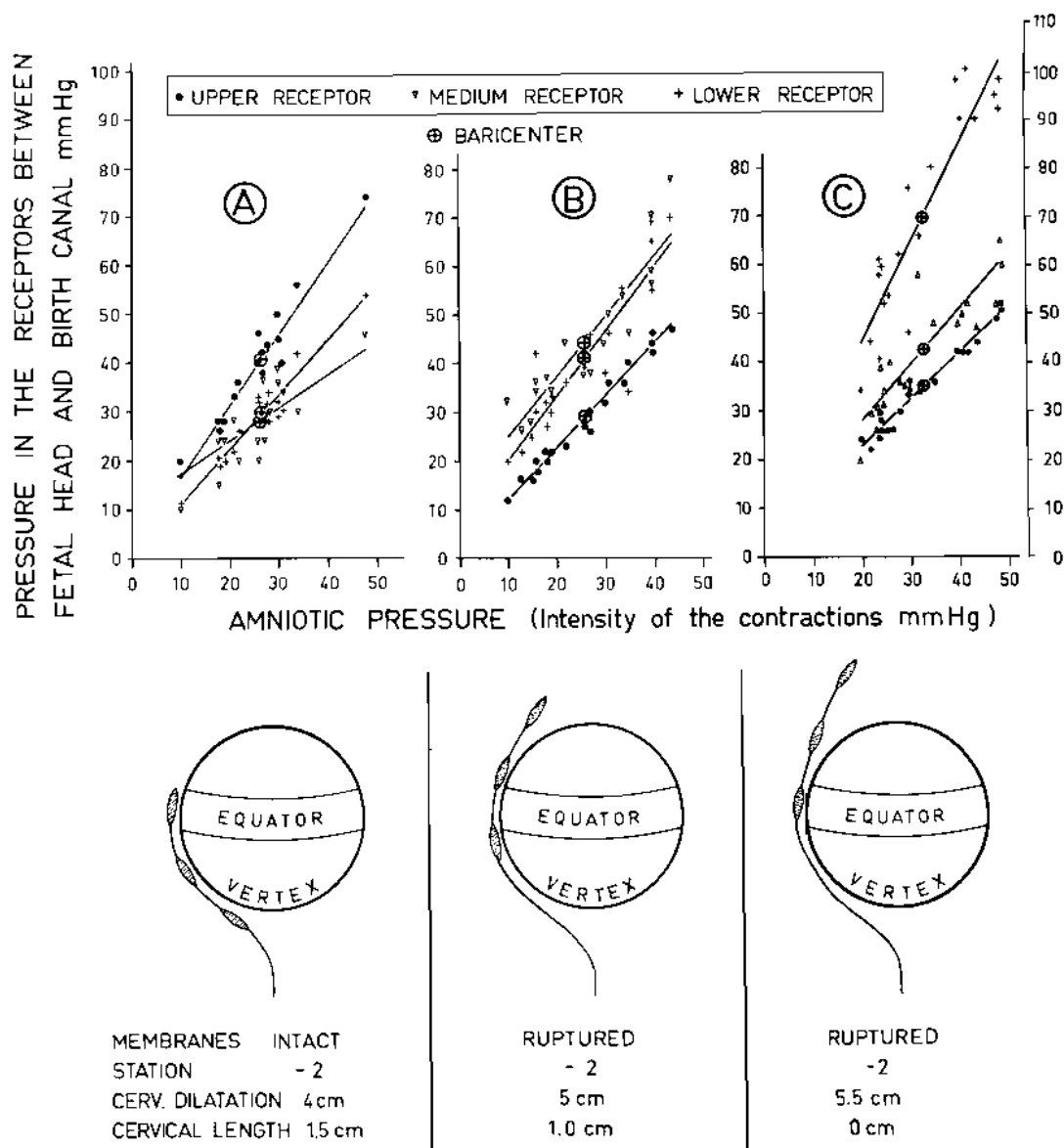


FIGURE 5. Upper half: correlation between pressure on cephalic receptors and in amniotic cavity measured at peak of each uterine contraction (Figure 3). A, B, and C refer to three periods illustrated in Figure 4. Diagrams in lower half of figure show, for each period, position of receptors in relation to fetal head.

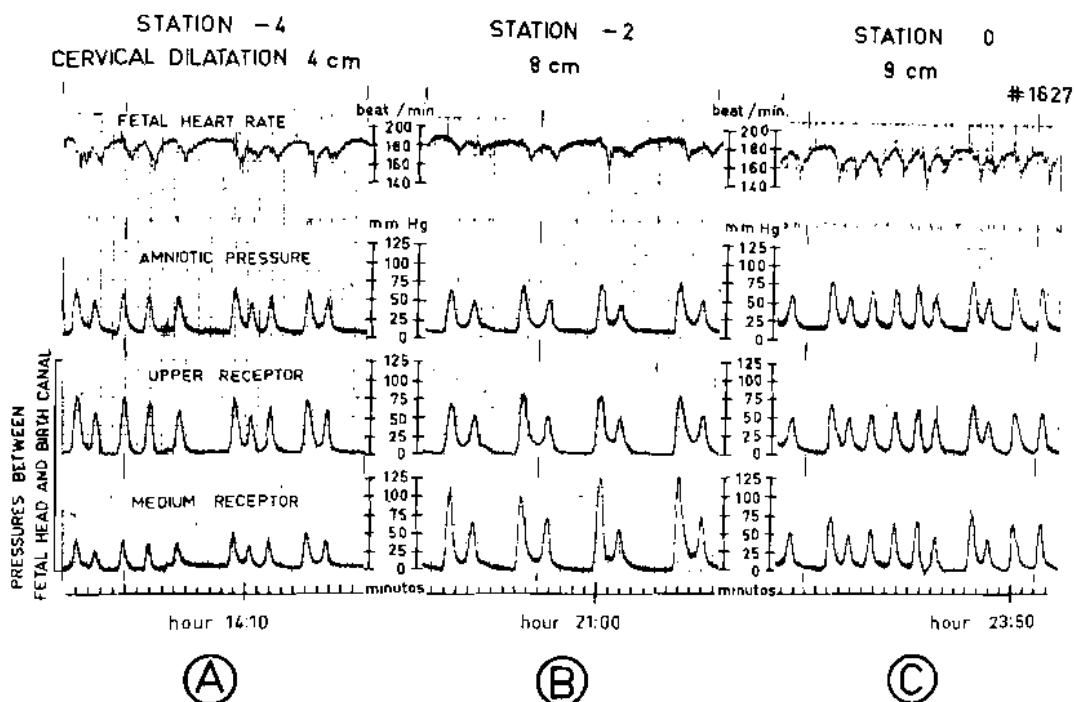


FIGURE 6. Three segments of record obtained during labor induced with oxytocin infusion at term pregnancy (newborn weight 3,460 g, Apgar score 6). Membranes were ruptured at hour 12:30. Receptor placed near equator of fetal head (upper receptor in Section A and lower receptor in section B) record pressure higher than that in amniotic cavity. In section C both receptors are distant from cephalic equator, and pressures recorded are similar to those in amniotic sac. Uterine contractions cause dips I in all 3 sections. This record is quantitatively analyzed in Figure 7.

The greater the pressure exerted by the uterine contraction on the fetal head, the larger the amplitude of the corresponding dip I in FHR (compare sections B and C in Figures 4 and 5). In Figure 8 almost every uterine contraction causes a dip I. Several contractions cause, in addition, a dip II—that is, a second transient fall in FHR recorded immediately after the dip I. Dips II will not be discussed in this paper. The amplitudes of dips I are larger in section B of Figure 8 than in sections A and C, probably because in section B the fetal head was receiving a stronger compression than in sections A and C, as is shown by the corresponding cephalic pressure receptors.

Figure 9 shows the direct linear relationship present in record No. 1475 between the amplitudes of dips I and the pressures recorded in the cephalic receptors at the peaks of the correspond-

ing uterine contractions. When this pressure was higher than 50 mm Hg, almost every contraction caused a dip I in the FHR tracing.

## Discussion

### *Relation between amniotic pressure and cephalic compression*

Our results confirm Lindgren's report (14, 15, 16) that under given conditions the pressure exerted by uterine contractions on receptors placed between the fetal head and the uterine wall may be much higher than the pressure in the amniotic cavity. They also confirm Lindgren's statement (14, 15) that the receptor placed at the equator of the fetal head is the one that records the highest pressure.

The ratio (pressure in cephalic receptor/amniotic pressure) is much higher in Lindgren's

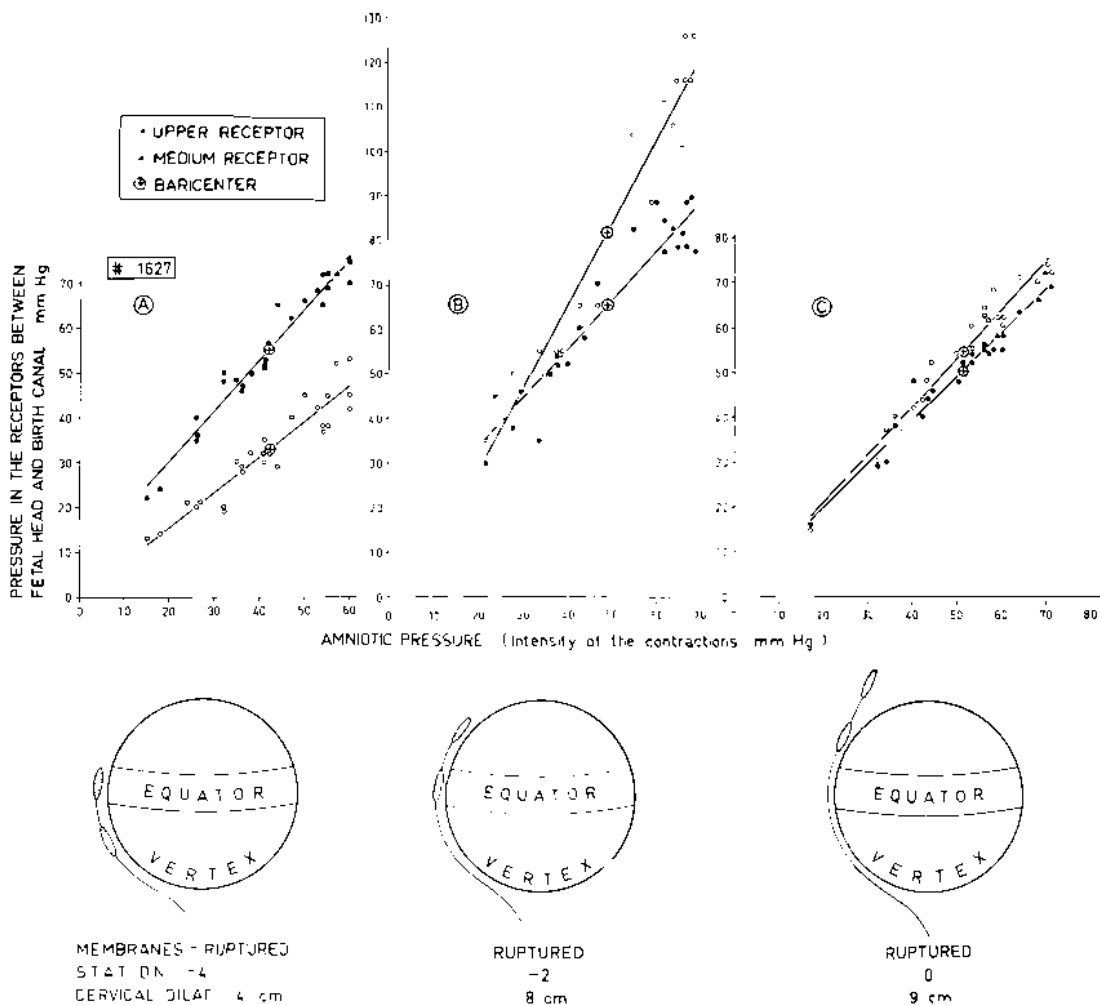


FIGURE 7. Upper half: correlation between pressure on cephalic receptors and in amniotic cavity measured at peak of each uterine contraction (Figure 3). A, B, and C refer to three periods illustrated in Figure 6. Diagrams in lower half of figure show, for each period, position of receptors in relation to fetal head.



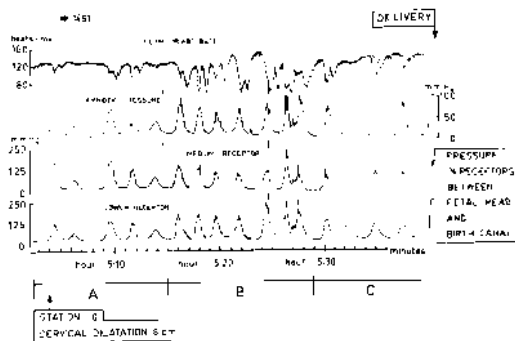
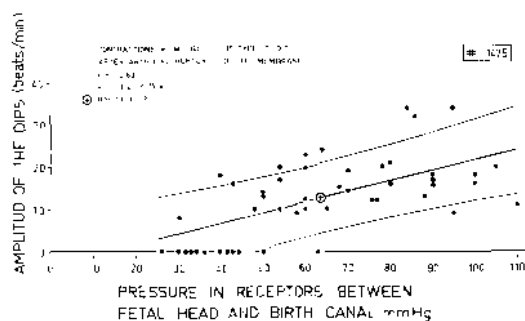


FIGURE 8. Record obtained during last 40 minutes of normal spontaneous labor at term pregnancy (newborn weight 3,375 g, Apgar score 8). Membranes were ruptured at hour 2:45. In section B, uterine contractions cause very strong compressions of cephalic receptors (up to 200 mm Hg) and dips I of very large amplitude. In sections A and C, pressure recorded by cephalic receptors and amplitude of dips I are smaller than in section B.

results (16) than in those reported here, even for similar obstetrical conditions (Figure 10). Whereas we found a linear relationship between the pressure in the cephalic receptors and the amniotic pressure, Lindgren finds (16) that when the amniotic pressure increases beyond a certain limit, the linear relation is lost, since the pressure in the cephalic receptor does not rise accordingly. These discrepancies may be due to differences in the type of cephalic pressure receptors employed.

### Pressure required to produce dips I

The correlation found between the pressure in



dominal manual compression was applied to the fetal head, the cephalic receptor indicated a pressure higher than 50–60 mm Hg before the fall in FHR was produced (Figure 11). Somewhat lower values (30–40 mm Hg) were obtained by Chung and Hon (7) with direct compression of the fetal head by the vaginal route. Kelly (13) estimated that when the amniotic pressure is 40 mm Hg the fetal head would be supporting a force of 11 pounds, assuming the head to be spherical and to have a diameter of 10 cm.

### Consequences of cephalic compression

**Cranial hypertension and cerebral ischemia.** Since the bones of the fetal skull are not fused, it is natural to assume that compression on the fetal head will produce cranial hypertension (Figure 12). The correlation between intracranial pressure and pressure in the receptor outside the fetal head was studied by simultaneous recording in the same fetus. Intracranial pressure was recorded in a dead fetus by introducing a catheter into the fetal head, which was punctured through the sagittal suture. Figure 13 shows that there is an acceptable correlation between direct intracranial pressure and the record obtained with the cephalic pressure receptors. In a living fetus, increased intracranial pressure may result in reduced blood flow through the brain. The resulting hypoxia and hypercapnia of the central nervous system would stimulate

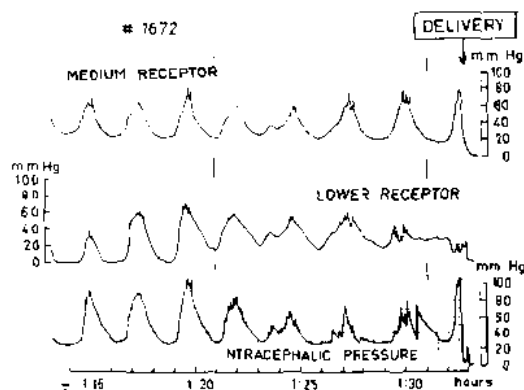


FIGURE 12. Pathophysiology of fetal disturbances resulting from cephalic compression produced by uterine contractions (working hypothesis).

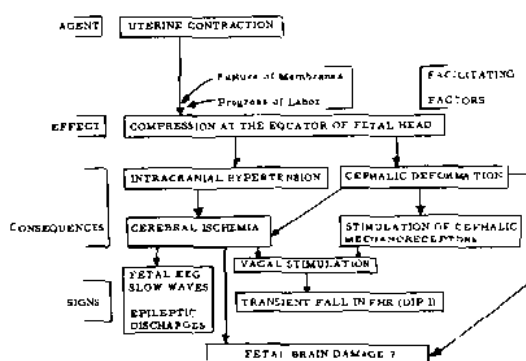


FIGURE 13. Lower tracing is record of intracephalic pressure obtained in dead fetus at 36th week of pregnancy by means of catheter introduced into head by puncturing cranium through sagittal suture. Elevations of intracephalic pressure caused by uterine contractions are similar to those recorded by pressure receptor placed between fetal head and uterine wall.

the vagus center and contribute to dips I (Figure 12). The complete disappearance of dips I after fetal atropinization is in accord with the hypothesis (5, 6) postulating that increased vagal tone is the mechanism involved in their pathogenesis. A transient cerebral ischemia can also explain the changes observed in the fetal electroencephalogram (slow waves of high voltage) (9) occurring during the peaks of strong uterine contractions that also produce dips I (Figure 12).

**Cephalic deformation.** Even in normal conditions, the compression of the equatorial zone of the fetal head is stronger than in other areas (16) and may cause a deformation (molding) of the head (Figure 12). Borell and Fernström (2) have shown radiologically that during labor the parietal bones are usually "disaligned"—that is, more prominent than the occipital and frontal bones (Figure 14A). It should be recalled that the larger portion of the parietal bones are in a zone distant from the equator—that is, one that receives a lower pressure. The frontal and occipital bones are in the equatorial zone, receive a stronger compression, and are relatively depressed inward (Figure 14).

The deformation of the head may stimulate cephalic mechanoreceptors, which may elicit a reflex vagal stimulation and thus contribute to

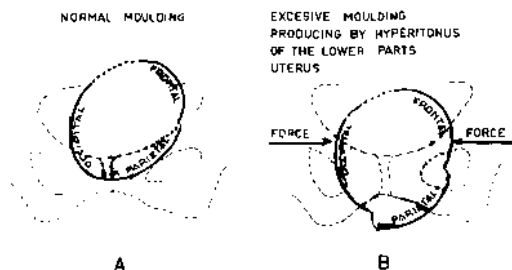


FIGURE 14. Molding of fetal head during labor. In normal conditions (A), parietal bone is moderately displaced outward and disaligned in relation to occipital and frontal bones. In abnormal conditions (B), parietal displacement becomes more pronounced, with great bony disalignment more marked at lambdoid suture. (Drawn after radiological pictures from Borell and Fernström.)

the pathogenesis of dips I (5, 17). By distorting cerebral vessels, cephalic deformation may additionally aggravate cerebral ischemia caused by increased intracerebral pressure (Figure 12).

#### *Factors facilitating equatorial compression and deformation of the fetal head*

**Hypertonus of the lower uterine segment.** The abnormal increase in the tone of the lower uterine segment (hypertonic lower segment, "spasm," inversion of contractile gradient) (4)

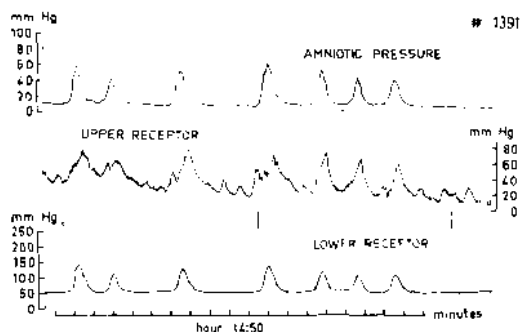


FIGURE 15. Hypertonus of lower uterine segment. Record obtained during labor at term pregnancy. Cervical dilatation is 5 cm. Vertex presentation in LOA, station -1. Upper cephalic receptor shows augmented contractility and tonus of lower part of uterus.

augments the compression on the equatorial zone (15) and magnifies the deformation of the fetal head (2) (Figure 14B). One example of increased tone and contractility of the lower part of the uterus is shown in Figure 15. The record obtained by the upper cephalic receptor shows much more contractile activity and higher pressures than the lower cephalic receptor or the amniotic pressure record.

**Rupture of the membranes.** When the membranes are intact and the fetal head is completely surrounded by amniotic fluid (Figure 16A),

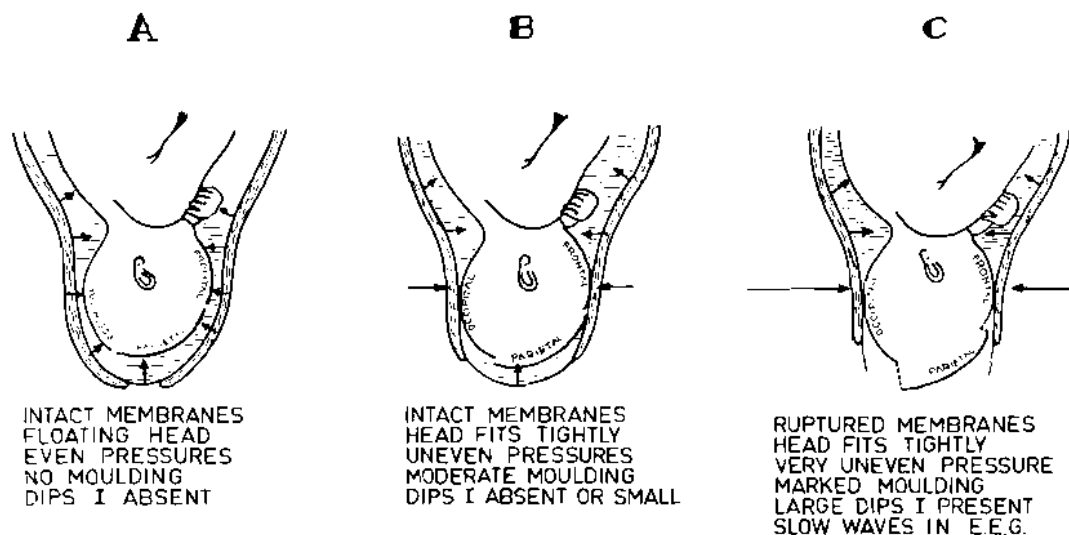


FIGURE 16. The three hydrostatic conditions in which fetal head may be during labor. In A there is no deformation of the fetal head; in B, only moderate molding; in C, marked deformation.

the pressure received is the same in all areas of the head. During uterine contractions there is no cephalic deformation. Blood flow through the brain is not disturbed, because the rise in cephalic pressure is similar to that occurring in other body fluids of the fetus, including the arterial pressure. Dips I are not produced.

When the equatorial zone of the head is in contact with the uterine wall (Figure 16B), this zone receives a higher pressure than the remaining areas of the cephalic pole. However, if the membranes are intact, some counterpressure will be exerted by the forewaters on the parietal bone, preventing excessive disalignment and molding.

This counterpressure markedly diminishes after the rupture of the membranes (Figure 16C), facilitating the bulging of the parietal bone. Molding is increased even further because the pressure on the equatorial zone augments after rupture of the membranes (14). The rise in intracranial pressure will be higher than that in amniotic pressure (and in fetal arterial pressure), with a consequent reduction in blood flow through the fetal brain. It follows that the rupture of membranes may facilitate cerebral ischemia and deformation of the fetal head by uterine contractions and thus the production of dips I.

#### *Fetal brain damage*

It is logical to assume that a repetition of successive episodes of cerebral ischemia and also the deformation of the brain may lead to permanent damage of the central nervous system of the fetus. This subject has not yet been properly investigated, although it deserves high priority. If cerebral damage may result from this mechanism, obstetricians should be very cautious before deciding to perform the rupture of membranes.

#### **Summary**

The pressure exerted by uterine contractions on the fetal head was recorded during labor

by means of flat pressure receptors introduced between the uterine wall and the fetal head, outside the membranes. Simultaneous records of amniotic pressure and fetal heart rate were obtained.

Each uterine contraction produced a compression of the fetal head equal to or greater than the pressure rise caused in the amniotic cavity, depending on the obstetrical conditions.

For each period of labor and for each given receptor, there is a direct linear relationship between the pressure recorded by that receptor and the amniotic pressure. The receptors placed near the equator of the fetal head record higher pressures than those at a greater distance. In the latter the pressure is equal to the amniotic pressure, whereas in the former the pressure may be up to 2.5 times higher than the amniotic pressure.

The stronger compression exerted by uterine contractions on the equatorial zone causes a deformation of the fetal head. This molding is usually characterized by bulging of the parietal bone because it receives less pressure than the occipital and frontal bones at the equatorial zone.

During each uterine contraction, the intracranial pressure increases and the cerebral blood flow is consequently reduced. The transient cerebral ischemia stimulates vagal tone and causes a temporary fall in fetal heart rate (dip I), which is simultaneous with the contraction.

Rupture of the ovular membranes increases the compression at the equatorial zone, diminishes the counterpressure at the parietal bone, facilitates molding of fetal head, and increases the production of type I dips. The possible damage to the fetal brain resulting from ischemia and deformation deserves further investigation.

#### **Acknowledgment**

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