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INFLUENCE OF THE RUPTURE OF MEMBRANES ON COMPRESSION OF THE FETAL HEAD DURING LABOR[1]

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In a previous paper (*10*) it has been shown that when uterine contractions of labor exert a strong compression on the fetal head, and this pressure is higher than the pressure in the amniotic fluid cavity, the fetal vagus is transiently stimulated and a transient fall (dip I) occurs in the tracing of the fetal heart rate (FHR).

Dips I occur simultaneously with the contraction (*2*) in such a way that the bottom of the dip is recorded almost at the same time as the peak of the contraction (Figure 1).[3] They are similar to the "early decelerations" described by Hon and Quilligan (*6*).

In this paper we shall present the influence of some obstetric factors, such as the rupture of membranes, on the incidence of dips I throughout labor and shall also discuss the probable mechanism of action.

Twenty-six pregnant women at term were studied during labor. Intrauterine (amniotic) pressure and FHR were continuously recorded from the beginning of labor to delivery by methods described previously (*2*). The progress of cervical dilatation and that of the station of

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[2] Presented by Dr. Althabe.

[3] Dips II, which occur 30 to 60 seconds after the contraction (*2*), will not be discussed in this paper.

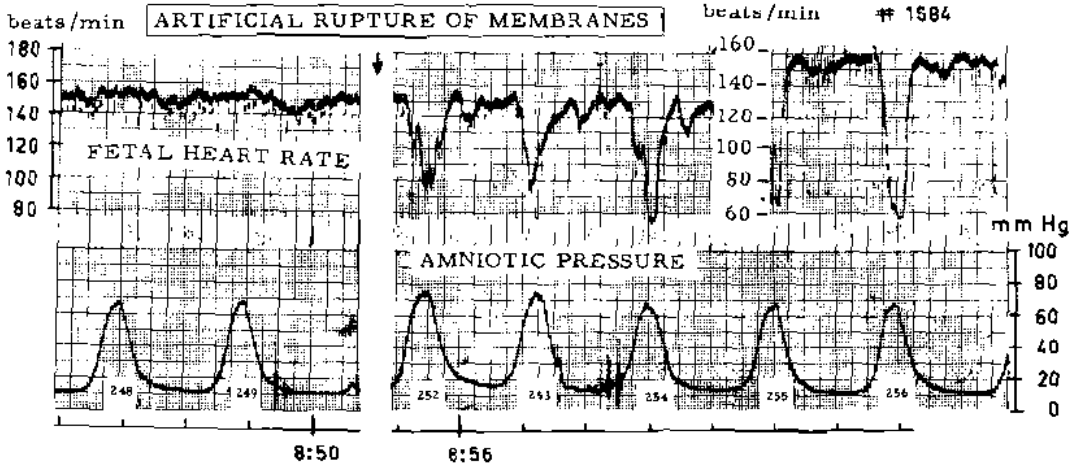

FIGURE 1. Record obtained during first stage of labor. Cervical dilatation 6 cm. Before rupture of membranes, dips I are absent from the FHR tracing. After rupture, each uterine contraction causes a dip I of large amplitude.

the fetal head were periodically checked by vaginal examination.

Effect of the rupture of membranes

The production of dips I by uterine contractions was greatly increased by the rupture of membranes. Figure 1 shows one typical record that illustrates such effects. Before the rupture, the contractions did not cause dips I (they only produced dips II of very small amplitude). After rupture of the membranes each uterine contraction caused a dip I of large amplitude (a small dip II follows each dip I).

Figure 2 and Table 1 show the results obtained in all 26 women. Before rupture of the membranes, 2,157 contractions were recorded and only 82 of them (3.8 per cent) produced dips I. After the rupture of membranes, 2,243 contractions were recorded and 747 of these (33.3 per cent) caused dips I. The difference between the percentages before and after the membranes were ruptured is highly significant ($p < 0.001$).

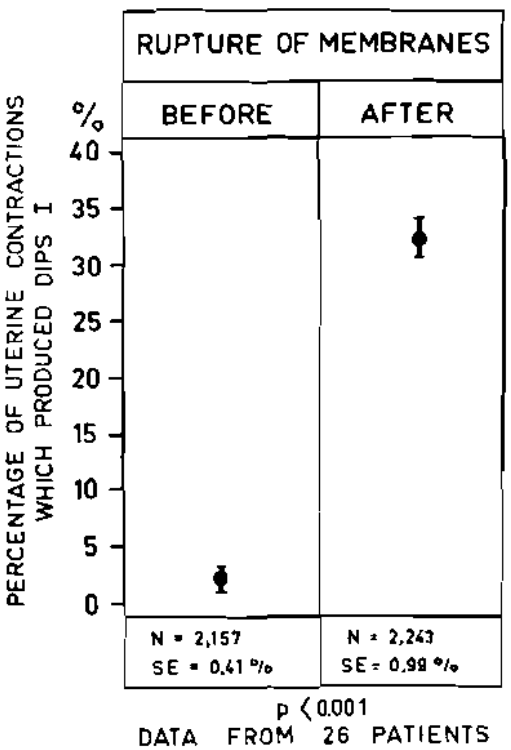

FIGURE 2. Incidence of dips I is significantly higher after rupture of membranes than before.

TABLE 1. Number of contractions causing dips I before and after rupture of membranes

	TOTAL CONTRACTIONS	CAUSING DIPS I		NOT CAUSING DIPS I
		NO.	% *	
Before rupture of membranes	2,157	82	3.8	2,075
After rupture of membranes	2,243	747	33.3	1,496
Total	4,400	829		3,571

* $p < 0.001$.

Interpretation

Our working hypothesis is schematically shown in Figure 3. When the membranes are intact and the head is floating (the cephalic equator being above the inlet), there is amniotic fluid all around the fetal head and it transmits the pressure of the uterine contractions equally in all directions (Pascal's law). The head receives the same pressure on its entire surface and is not deformed during the contractions (Figure 3A). Furthermore, the same pressure is also exerted on the fetal body, umbilical cord, and placenta. The contractions produce no changes in blood flow through the fetal brain. Under these conditions they do not produce dips I.

During engagement and descent of the head, the cephalic equator fits tightly in the lower uterine segment (Figure 3B). There is no longer free communication between the amniotic cavity and the forewaters. The pressure exerted by the uterine contractions on the cephalic equator is greater than that above and below the equator (*8, 10*). The fetal head is slightly deformed (molding) and it becomes lengthened from chin to occiput and shortened in all other directions; as a result, the parietal bones bulge and become disaligned with the frontal and occipital bones. The rise in intracephalic pressure during the contractions may be

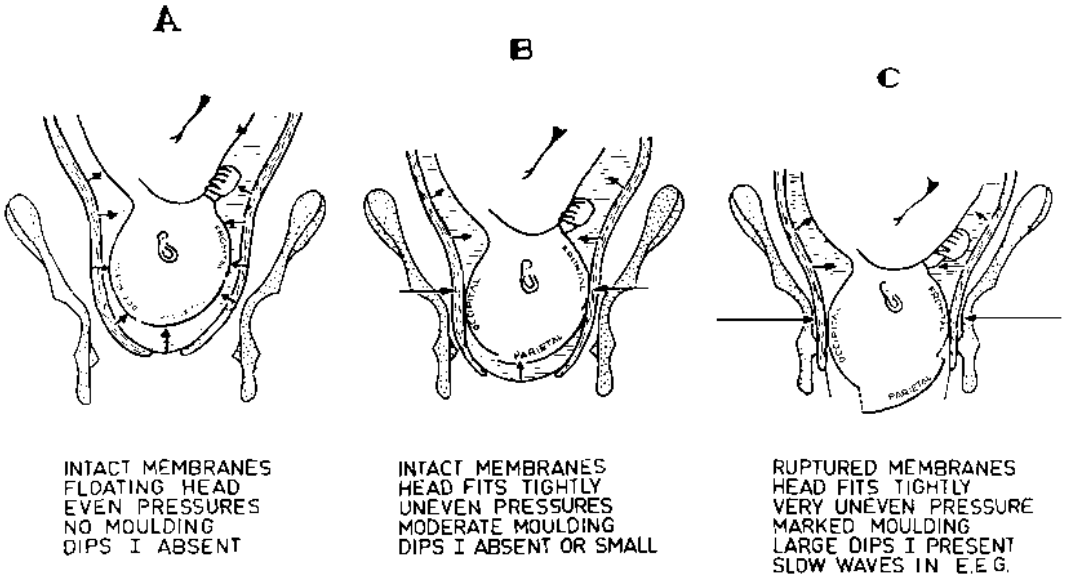

FIGURE 3. Diagram illustrating forces acting on fetal head under different conditions of labor and resulting cephalic deformation (molding).

greater than that occurring in the amniotic cavity and in the fetal body fluids; the consequence is a transient reduction of cerebral blood flow. In these conditions, the incidence of dips I is low and their amplitude small.

After the rupture of membranes, there is no longer any counterpressure exerted by the bag of waters against the lower part of the head and the pressure on the cephalic equator during contractions is increased (9) (Figure 3C). The rupture of membranes thus facilitates the deformation of the fetal head by the contractions (Figure 3C) and the disalignment of the parietal bones is very marked.

The rise in the intracranial pressure during uterine contractions is now markedly greater than that occurring in the amniotic sac and the fetal body fluids and may cause a significant reduction in cerebral blood flow. The resultant ischemia, hypoxia, and hypercapnia of the brain are known to cause direct stimulation of the vagal center, a mechanism that explains the transient fall in FHR occurring simultaneously with these contractions.

Cerebral ischemia may explain the change in the EEG pattern (high-voltage slow waves) observed at the time of the peak of strong contractions that produce dips I (5). Cerebral ischemia also stimulates the vasomotor center and may cause fetal arterial hypertension, which in turn, acting through the baroreceptors of the carotid sinus and aortic arch, will reflexively stimulate the vagus, causing the dip I (7).

The deformation of the fetal head may stimulate mechanoreceptors (3) in the face and head, which may also reflexively stimulate the vagus and contribute to producing dips I. The deformation of the cranial cavity may also disturb blood flow and contribute to cerebral ischemia, eliciting the mechanism described above. Since the rupture of membranes increases the compressive effects of uterine contractions on the fetal head (deformation and cranial hypertension), it naturally encourages the production of dips I.

Effects of the progress of cervical dilatation

This study was made in the same 26 patients. Labor was divided into eight consecutive periods according to cervical dilatation (Figure 4). The second stage was included in the eighth period. All the uterine contractions recorded during each of the eight periods in all 26 patients were pooled. The percentage of contractions causing

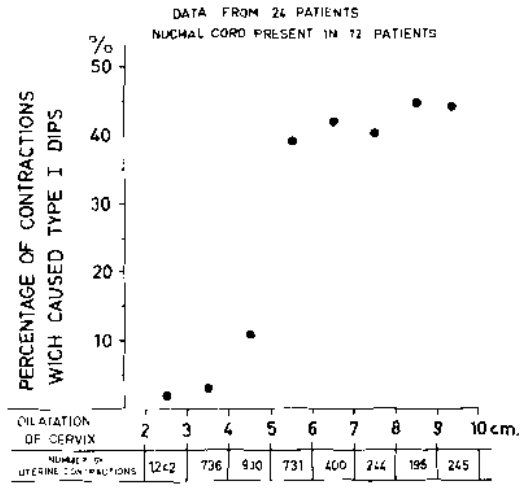

FIGURE 4. Incidence of dips I increases as cervical dilatation augments. A marked rise is produced between 4 and 6 cm of dilatation.

dips I—that is, their incidence—was very low for the first three periods (cervical dilatation from 2 to 5 cm) and increased markedly (about 40 per cent) for the remaining periods. This confirms previous reports by Faúndes *et al.* (*4*) and Aramburú *et al.* (*1*).

For statistical analysis, labor was divided in

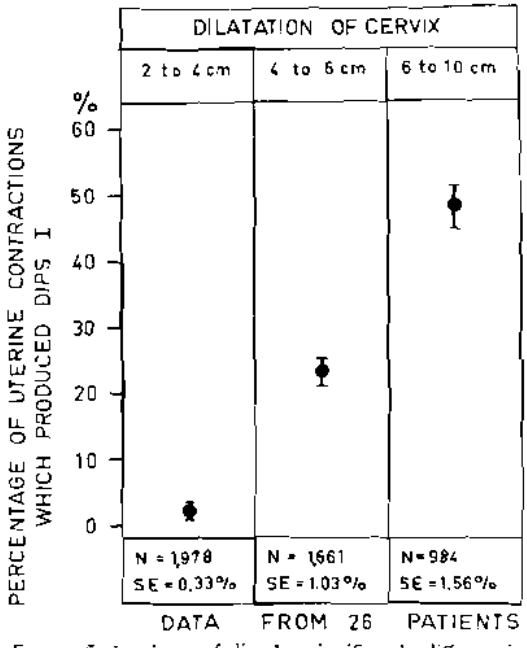

FIGURE 5. Incidence of dips I is significantly different in the three periods of labor divided according to dilatation of cervix.

TABLE 2. Number of contractions causing dips I in relation to progress of cervical dilatation

DILATATION	TOTAL CONTRACTIONS	CAUSING DIPS I		NOT CAUSING DIPS I
		NO.	%	
2–4 cm	1,978	44	2.22	1,934
4–6 cm	1,661	385	23.18	1,276
6–10 cm	984	470	47.76	514
Total	4,623	899		3,724

three periods: early first stage (dilatation 2–4 cm); mid-first stage (dilatation 4–6 cm); and advanced labor (dilatation 6 to 10 cm and second stage) (Figure 5). The difference between the three periods in the incidence of dips I was highly significant (Table 2).

Three factors may explain these results:

1. *Cervical dilatation by itself.* When the cervix is closed (Figure 3A) the part of the lower uterine segment that faces the lower part of the head would reinforce (if membranes are intact) or exert (if membranes are ruptured) the counterpressure that could minimize cephalic deformation. This counterpressure diminishes gradually as cervical dilatation progresses, facilitating cephalic deformation.

2. *Coincidence of progress in cervical dilatation with rupture of membranes.* In about 80 per cent of the 26 patients studied, the membranes were artificially ruptured when cervical dilatation was between 4 and 6 cm (Figure 6).

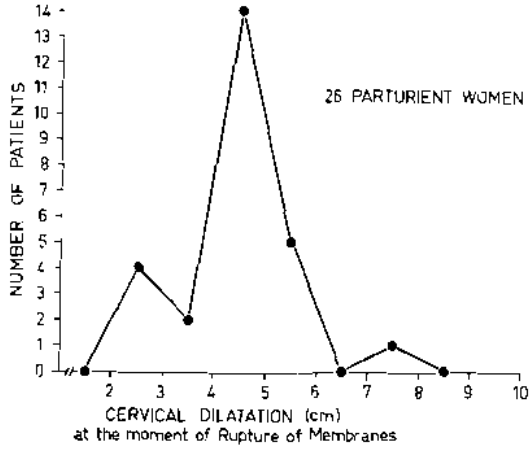

FIGURE 6. Frequency polygon showing cervical dilatation in 26 patients at moment of rupture of membranes.

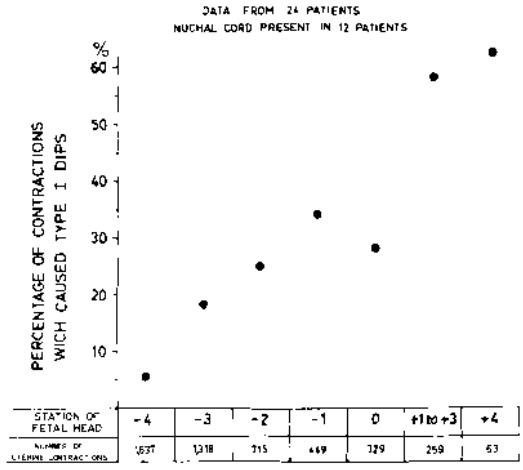

FIGURE 7. Incidence of dips I rises as fetal head engages and descends in birth canal.

This may explain the abrupt rise in the incidence of dips I when cervical dilatation progressed to this point (Figure 4).

3. *Coincidence of the progress in cervical dilatation with progressive engagement and descent of the fetal head in the birth canal.* This factor will be discussed below.

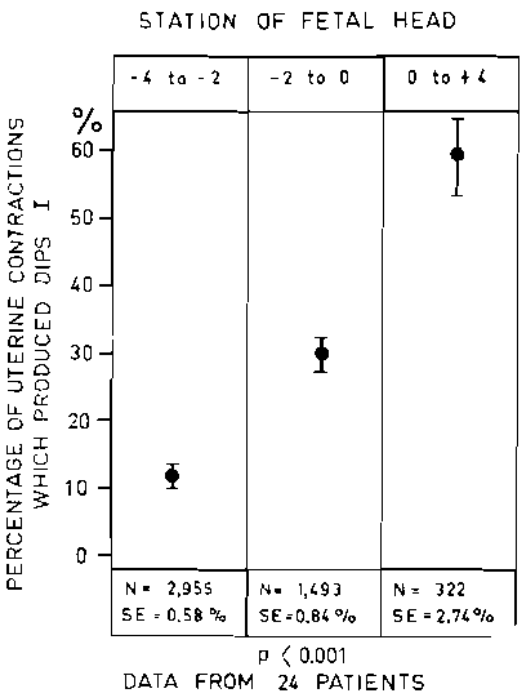

FIGURE 8. Incidence of dips I differs significantly in the three periods of labor divided according to the station of the fetal head.

Influence of station of fetal head

Figure 7 shows that the incidence of dips I rises progressively as the fetal head descends in the pelvis, confirming previous reports of Aramburú *et al.* (*1*).

To study this problem statistically, labor was divided into three periods according to the station of the fetal head (Figure 8, Table 3). In the first period (station –4 to –2) the incidence of dips I was only 11 per cent. It increased to 30 per cent when the station was between –2 and 0, and to 60 per cent when the head was between stations 0 and +4. The difference in incidence between these three periods is highly significant ($p < 0.001$).

The progress of the fetal head through the birth canal may in itself increase the incidence of dips I, because as the head becomes more engaged the compression exerted by the uterine contractions on the cephalic equator augments (Figure 3B and C). However, these effects may be influenced by the coincidence with simultaneous progress in cervical dilatation, which might have a direct effect of its own.

Furthermore, the number of cases with ruptured membranes increases as the fetal head descends, and this may also influence the results. The linear relationship illustrated in Figure 8 between incidence of dips I and the station of the fetal head suggests that the timing of the rupture of membranes has been more or less evenly distributed between stations –4 and –1.

There is a need for studies to evaluate the influence of each of the three factors mentioned

TABLE 3. Number of contractions causing dips I in relation to progress of fetal head through birth canal

STATION	TOTAL CONTRACTIONS	CAUSING DIPS I		NOT CAUSING DIPS I
		NO.	%	
—4 to —2	2,995	333	11.27	2,622
—2 to 0	1,493	444	29.74	1,049
0 to +4	322	189	58.70	133
Total	4,770	966		3,804

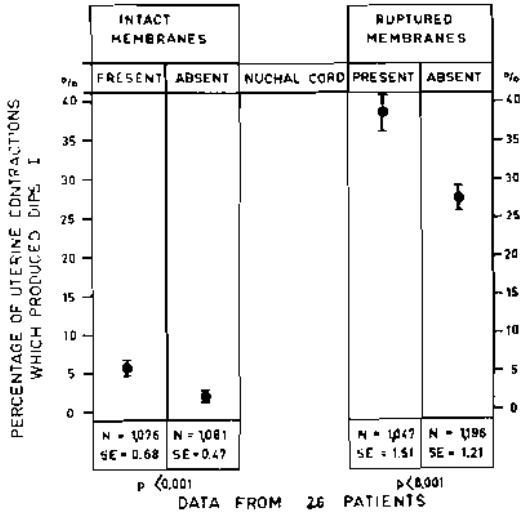

FIGURE 9. Presence of nuchal cord increases incidence of dips I. Differences between groups with and without nuchal cord are significant, both with intact and with ruptured membranes.

—rupture of membranes, cervical dilatation, and descent of the fetal head.

Influence of the presence of nuchal cord

Nuchal cord was present in 13 of the 26 patients studied. With intact membranes the incidence of dips I was 2.4 per cent when nuchal cord was absent and 5.2 per cent when it was present. The difference is significant ($p<0.001$) (Table 4). With ruptured membranes, the incidence was 28 per cent without nuchal cord and 39 per cent with. This difference is also highly significant ($p<0.001$) (Table 5).

TABLE 4. Number of contractions causing dips I before rupture of membranes in patients with and without nuchal cord

NUCHAL CORD	TOTAL CONTRACTIONS	CAUSING DIPS I NO.	CAUSING DIPS I % *	NOT CAUSING DIPS I
Present	1,076	56	5.20	1,020
Absent	1,081	26	2.41	1,055
Total	2,157	82		2,075

* $p < 0.001$.

TABLE 5. Number of contractions causing dips I after rupture of membranes in patients with and without nuchal cord

NUCHAL CORD	TOTAL CONTRACTIONS	CAUSING DIPS I NO.	CAUSING DIPS I % *	NOT CAUSING DIPS I
Present	1,047	410	39.16	637
Absent	1,196	337	28.18	859
Total	2,243	747		1,496

* $p < 0.001$.

The presence of nuchal cord increases the production of dips I, both when the membranes are intact and when they are ruptured. However, its influence is far less striking than that of the rupture of membranes.

The presence of one or several loops of the cord around the fetal neck may facilitate the compression or stretching of the umbilical vessels by uterine contractions. These stimuli are known to elicit a reflex fall in FHR (6).

Summary

Dips I are transient falls of fetal heart rate (FHR) occurring simultaneously with uterine contractions. Much evidence indicates that they are caused by a strong compression and deformation of the fetal head resulting in vagal stimulation. This stimulation may result either from the cephalic deformation or from cerebral ischemia due to intracranial hypertension produced by cephalic compression. The association of dips I with EEG alterations agrees with the latter hypothesis. It is not known whether permanent brain damage may result.

The incidence of dips I in a given period of labor is expressed as a percentage of the uterine contractions that caused them. In a group of 26 parturient women the incidence was significantly greater after the rupture of membranes (33 per cent) than when these were intact (4 per cent). It rose markedly as cervical dilatation increased and the station of the fetal head progressed. In advanced labor (cervical dilatation greater than 6 cm, fetal head beyond

station 0, and ruptured membranes) it was about 50 per cent—significantly higher than in early labor (intact membranes, cervical dilatation smaller than 4 cm, and fetal head above –3 station), when it was about 2 per cent.

In this study the membranes were ruptured when cervical dilatation was between 4 and 6 cm, as has become accepted practice. It would be highly interesting to make a similar study in a group of patients in whom the membranes could remain intact until the second stage of labor.

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