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EFFECTS OF UTERINE CONTRACTIONS ON THE EEG OF THE HUMAN FETUS DURING LABOR[1]

Elio García-Austt

Human fetal electroencephalograms were first obtained by Lindsley (*13*) by means of electrodes applied on the maternal abdominal walls. Later, similar results were reported with an improved technique using intravaginal electrodes (*2, 3, 4, 5, 17, 19*).

With this technique human fetal EEGs were recorded during advanced labor. It would be important to establish the possible influence on the EEG of fetal anoxia and acidosis during the last stages of labor, when uterine contractions provoke or increase the systemic metabolic disorders. Moreover, a number of findings indicate that uterine contractions causing transient falls in fetal heart rate (FHR) (*1, 20*) also produce a compression and deformation of the fetal head (*20*) and probably a transient reduction in fetal brain blood flow.

It is thus of interest of find out what changes occur in the fetal EEG during uterine contractions and whether these modifications are correlated with the strength of the contraction and the variations in FHR The initial results obtained in nine fetuses will be presented in this paper. Preliminary observations have been submitted recently (*9*).

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Material and methods

Nine full-term pregnant women were studied during labor, six of them under carbocaine epidural and/or caudal anesthesia. The effect upon the EEG of 124 uterine contractions was analyzed.

The fetal EEG was recorded during advanced labor after rupture of the membranes and with cervical dilatations greater than 4 cm. In every case the head was at or beyond station 0. Three to six electrodes were inserted in the scalp over both parietal and occipital bones, which were the easiest zones to reach through the birth canal. The electrodes were hook-shaped platinum needles, enamel-insulated except at the tip. An 8-channel Grass electroencephalograph (Model III) was used. The EEG was obtained by means of bipolar leads between all the electrodes but one which was grounded. The paper speed was 15 mm/second.

The uterine contractions were inscribed by recording the intrauterine pressure with a thin polyethylene catheter introduced into the amniotic sac by the transabdominal or transvaginal route (*6*). The catheter was connected to a pressure transducer and the latter to an 8-channel recording Poly Viso, with a paper speed of 10 mm/minute. In the first recording the transducer was also connected to the electroencephalograph by means of a Grass Balance Demodulator Unit, in order to have the record of the contraction on the same paper as the EEG. This

simultaneous recording, of course, made it possible to study the relationship between the two variables.

The FHR was recorded by means of an instantaneous cardiotachometer, which was triggered by the R waves of the fetal ECG. The ECG was obtained with a scalp electrode that supplied a fetal signal free from maternal interference (6). The FHR was recorded in the Poly Viso.

All these variables, including the EEG in the last five cases, were recorded on magnetic tape in an instrumental tape recorder (Stanborn-Ampex Model 2007). Subsequently the playback of the tape was recorded on a 6-channel Grass Polygraph (Model V), and all these data were compared at a convenient speed (15 mm/sec) and amplification. Different time constants were used (½ amplitude low frequency 0.6/sec and ½ amplitude high frequency 3/sec and 15/sec).

The Apgar score was determined twice, one and five minutes after birth. A neurological examination was carried out within the first 48 hours of life and between the third and fifth days. Postnatal EEGs were obtained in the same electroencephalograph using the 10/20 system for electrode position.

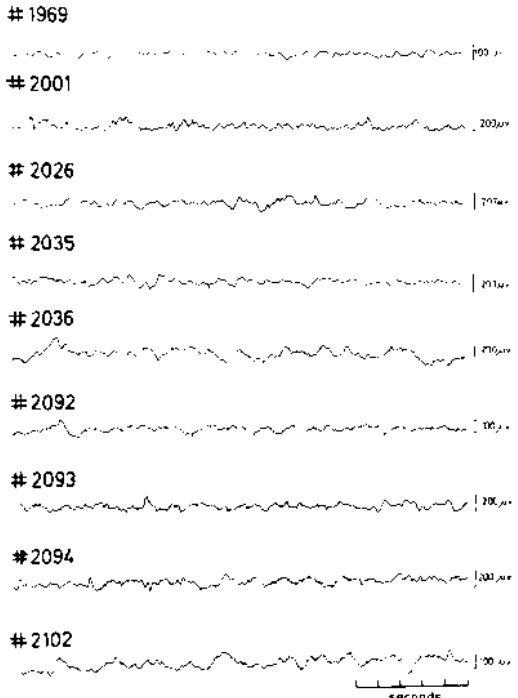

FIGURE 1. Samples of background EEG of 9 fetuses studied. Most common pattern is slow irregular activity of 2–5/sec and amplitude of 50–150 μV (2026 and 2093). Slower waves of 0.5–1/sec are also observed (2036 and 2102). 2001 shows rhythm of 2/sec in middle of record. In 2093 and 2094 irregular low-voltage fast activity is superimposed on slow waves.

Results

The fetal EEG exhibited constantly slow waves during most of the uterine contractions. In some instances epileptiform activity was also recorded. The characteristics of the background fetal EEG—that is, the recording obtained between contractions when the uterus was relaxed—the slowing of the EEG observed during contractions, and the epileptiform activity will be described successively.

Background fetal EEG during labor

The most constant pattern recorded (Figure 1) was a slow irregular activity with a frequency of 2–3/sec and an amplitude of 50–100 μV (cases 2026 and 2093). Slower waves of 0.5–1/sec were also currently observed (cases 2036 and 2102). In one case a regular rhythm of 2/sec was quite consistent along the record (case 2001). In some instances irregular low-voltage fast activity was superimposed on the slow waves (cases 2093 and 2094).

Changes in the EEG not related to the uterine contractions, and apparently spontaneous, were observed (Figure 2). An increase in the amount and amplitude of slow waves, the appearance or increase of low-voltage fast activity, and a considerable reduction in amplitude were the most common variations observed. On the whole, these changes were not consistent, the various patterns alternating throughout labor. In some cases, however, a reduction in amplitude was noted as labor progressed (case 2094). In two cases (2094 and 2102) that exhibited both a low Apgar score and epileptiform activity related

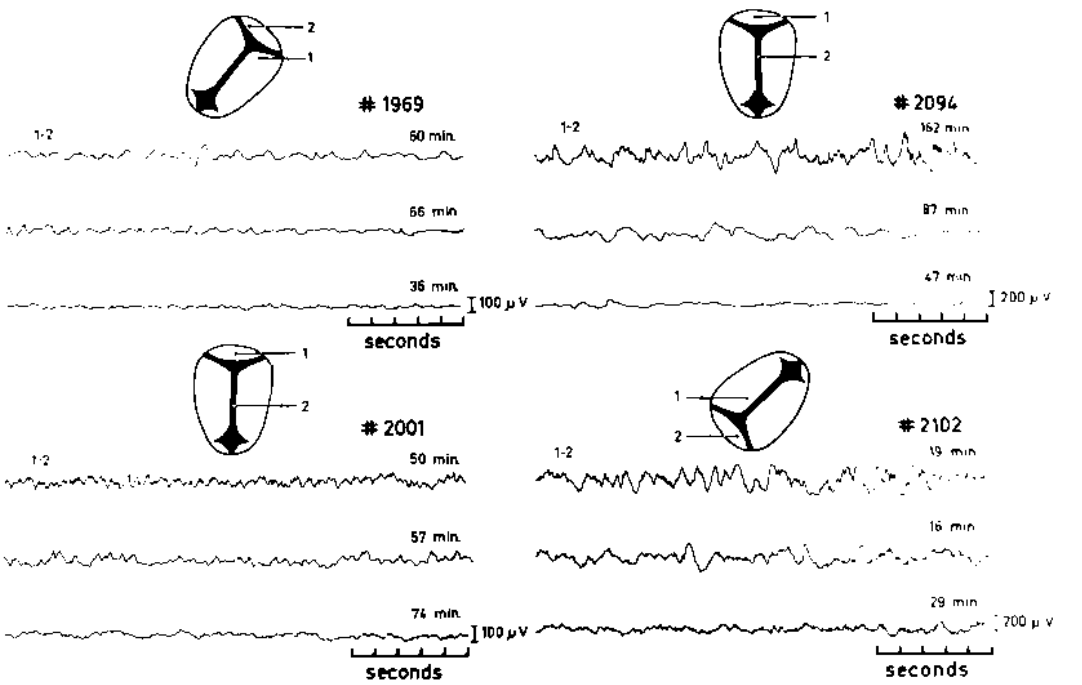

FIGURE 2. "Spontaneous" changes of background fetal EEG during labor. Samples obtained with leads indicated are arranged according to decreasing amplitude in each case. Time is minutes before delivery. Case 1969 (high Apgar score) has irregular slow activity without low-voltage fast activity superimposed. Case 2001 (also high Apgar score) has faster EEG with low-voltage faster activity superimposed. Cases 2094 and 2102 were born with low Apgar scores. Both show considerable amount of low-voltage slow waves with faster activity superimposed. In case 2094 only, reduction in EEG amplitude was progressive throughout labor.

to uterine contractions, patterns of high-voltage slow waves were found to predominate.

Suppression-burst periods were never observed in these full-term fetuses.

Slowing of the EEG during uterine contractions

Of the 124 contractions studied, 76 (61 per cent) provoked consistent changes in the EEG (Table 1). The most frequent variation was the appearance of irregular high-amplitude slow waves of 100–200 µV and 0.5–1/sec (Figures 3 and 4). These changes were present in all the fetuses studied, whether or not the contractions were accompanied by bearing-down efforts.

This slowing of the EEG was gradual and was observed with a latency of 5 to 15 seconds from the beginning of the contractions. The maximum slowing coincided with the peak of the contractions and subsided a few seconds before they ended. In those cases in which it was possible to obtain various simultaneous leads with several electrodes distributed over the scalp, it was found that the slowing of the EEG was generalized and appeared simultaneously in all the leads studied (Figure 5). No statistical dependency between the strength of the contractions and the slowing of the EEG was found.

TABLE 1. Slowing of EEG and dips I during uterine contractions

	PRESENT	ABSENT	TOTAL
EEG slowing			
No.	76	48	124
%	61	39	100
Dips I			
No.	60	62	122
%	49	51	100

$\chi^2 = 8.2$; $r = 0.21$; $p < 0.05$.

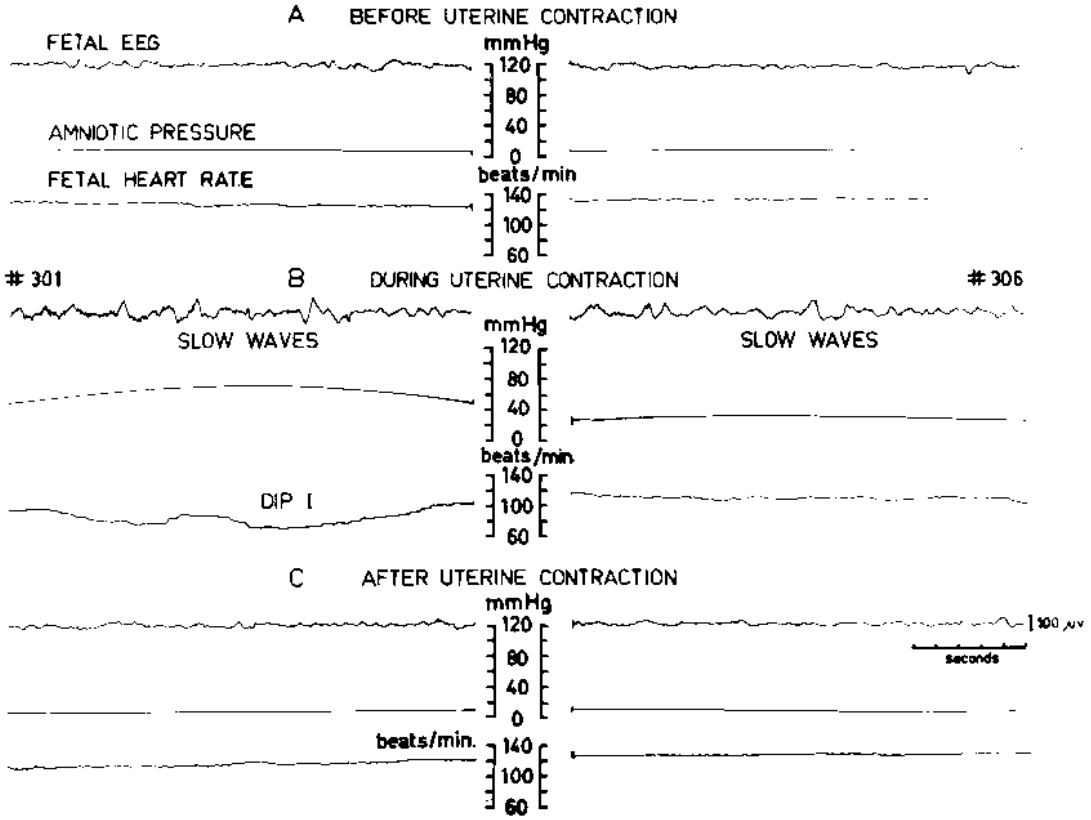

FIGURE 3. Slowing of fetal EEG during uterine contractions. Effects of two contractions (left and right). Before contraction patterns are similar. During contractions similar slowings are observed though contraction 306 (left) is weaker and provokes no changes in FHR (dips I). After contraction both EEG's show same pattern as before.

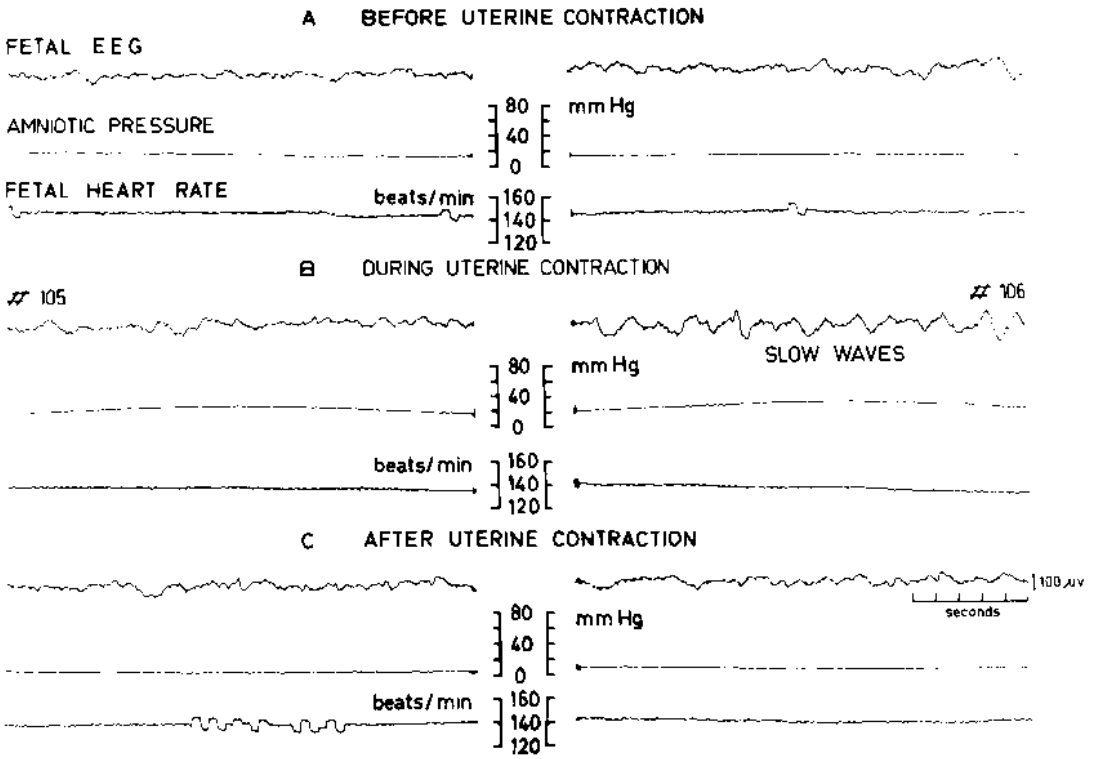

FIGURE 4. Slowing of fetal EEG during uterine contractions. Effects of two contractions (left and right). Before contractions EEG patterns are similar. During and after contraction 105 (left) no significant changes. During contraction 106, considerable slowing of EEG, which reacquires its initial characteristics after contraction. No FHR changes in either contraction.

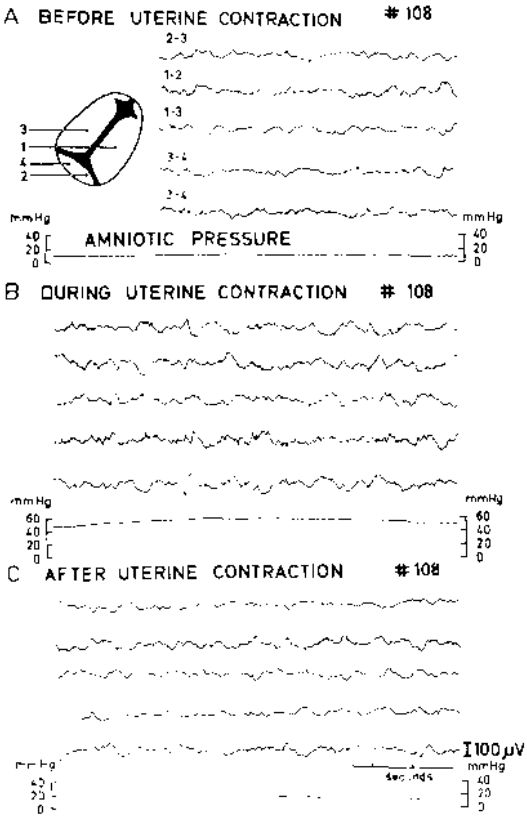

FIGURE 5. Generalized slowing of EEG during uterine contraction. First five records are EEGs obtained simultaneously from different regions of scalp at electrode positions indicated; bottom record is amniotic pressure. Background EEG (A) shows only slight differences between leads. At peak of contraction (B), generalized slowing of EEG, with certain predominance in some leads. After contraction (C) EEG reacquires its initial characteristics.

In 60 of the 124 contractions (49 per cent), transient falls in FHR (dips I) were observed, which were synchronous with the peak of the uterine contractions (Table 1). A statistical dependency between the two phenomena was found (χ^2 8.2) but there was not a significant correlation ($r = 0.21$) between the intensity of the contractions and the slowing of the EEG (Figures 3 and 4).

This slowing simultaneous with the uterine contractions was evident in all fetuses, even those that at birth were in apparently good condition as evaluated by the Apgar scores. In two cases exhibiting a low Apgar score the

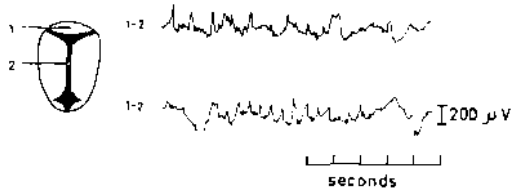

FIGURE 6. Random and rhythmic fetal epileptic activity. Samples obtained from same fetus at different moment with same lead at electrode position indicated. Above, random single sharp waves; below, same waves are repeated rhythmically.

slow waves persisted after the contractions or reappeared a few seconds later. These changes coincided with descents in FHR that appeared or persisted after the contraction (dips II). Under these circumstances the changes were complex ones, as will be discussed below.

Epileptiform activity

Some of the fetuses evidenced random and/or rhythmic epileptiform activity during labor (Figure 6). Random epileptiform activity consisted of slow sharp waves of 200–300 msec duration and 200–400 µV amplitude. These sharp waves were single or repeated many times at a random frequency (Figure 7).

But the most common finding was rhythmic epileptiform activity (Figure 7). Regular

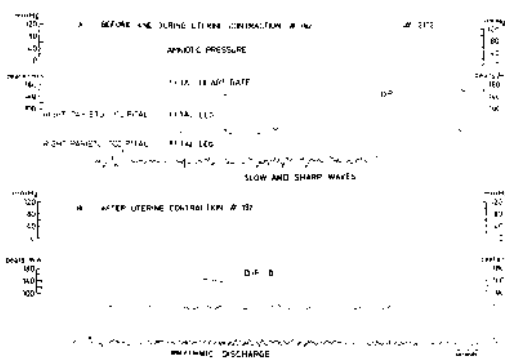

FIGURE 7. Random and rhythmic epileptiform activity during uterine contraction. A and B continuous records. Fetal EEG was recorded simultaneously with two different time constants: long (upper), with slow waves best observed, and short (lower), with faster waves. Before contraction (A), a few high-voltage slow waves and occasionally single sharp waves; during contraction (A), increase in slow waves and a dip I. After contraction (B), a rhythmic discharge develops, best seen in lower EEG, and a dip II is observed.

rhythms of sinusoidal or half-rectified waves with progressive changes of frequency were observed during 5 to 50 seconds. Sometimes the initial frequency was higher (4–5/sec) and decreased at the end of the discharge (1–1.5/sec). In other instances the opposite sequence was recorded. The amplitude of these discharges was of the same order as or lower than that of background activity. As the discharge progressed the amplitude increased. This recruitment or build-up of epileptiform discharge was a common finding.

The epileptiform discharges did not exhibit a great tendency to propagate. On some occasions it was possible to observe propagation over one hemisphere from one electrode to the next. In other instances discharges were seen in both hemispheres, although not synchronous. Bilateral synchronous activity was never observed.

In most cases the epileptic discharges coincided with the uterine contractions (Table 2, Figure 7). They usually appeared a few seconds after the contraction, coinciding with the descents in FHR observed about 40 seconds after the contractions (dips II). This relationship was observed in two fetuses born with a low Apgar score (cases 2094 and 2102). In three others, born with a high Apgar score and not showing dips II, the discharges likewise had the same time relationship with the contractions. Less frequently the epileptiform activity appeared at the onset or at the peak of the contraction. A statistical dependency and correlation between dips II and the fetal epileptiform activity was found ($\chi^2=52.7$; $r=0.672$) (Table 2).

In the two cases with a low Apgar score a number of epileptiform discharges unrelated to the contractions were also observed. In one fetus rhythmic epileptiform activity was set up when compressing the anterior fontanel and upon placing the forceps.

All but one of the cases in which epileptiform activity was recorded during labor exhibited activity of the same type in the EEGs obtained after birth (Figure 8).

The number of cases studied is as yet not large enough to substantiate data of statistical significance. We shall confine ourselves to three typical examples (Table 3):

TABLE 2. Epileptic activity and dips II during uterine contractions

	PRESENT	ABSENT	TOTAL
Epileptic activity			
No.	35	89	124
%	28	72	100
Dips II			
No.	44	80	124
%	35.5	64.5	100

χ^2 52.7; four-fold point correlation = 0.672; $p<0.001$.

Case 2035 had a normal delivery. Its heart rate presented only dips I. The Apgar score at the first and fifth minutes was 7 and 10, respectively, and the neurological examinations were normal. The fetal EEG showed slow waves during contraction but no epileptiform activity. The subsequent EEGs obtained at one hour, two and a half months, four months, and one year of life were normal. In this case all the findings concurred to confirm the normal delivery of a normal baby.

Case 2094 presented a great amount of epileptiform activity during delivery, in addition to slow waves coincident with uterine contractions. Dips II were observed along with dips I. The Apgar scores were low and the neurological examinations abnormal. The postnatal EEG studies exhibited epileptiform activity up to the fifth day of life. An EEG taken two and a half months later was normal. In this case abnormal signs were apparent in labor (epileptiform activity and dips II) and persisted for some time after birth (low Apgar score, abnormal neurological examinations, and epileptiform activity).

In case 2026, in addition to slow waves epileptiform activity appeared during labor. Dips II were not present. After birth the Apgar scores were high and the neurological examinations normal. On the sixth postnatal day the EEG persisted abnormal. The last EEG, recorded one year later, was normal. Evidently in this case there was a discrepancy between the EEG find-

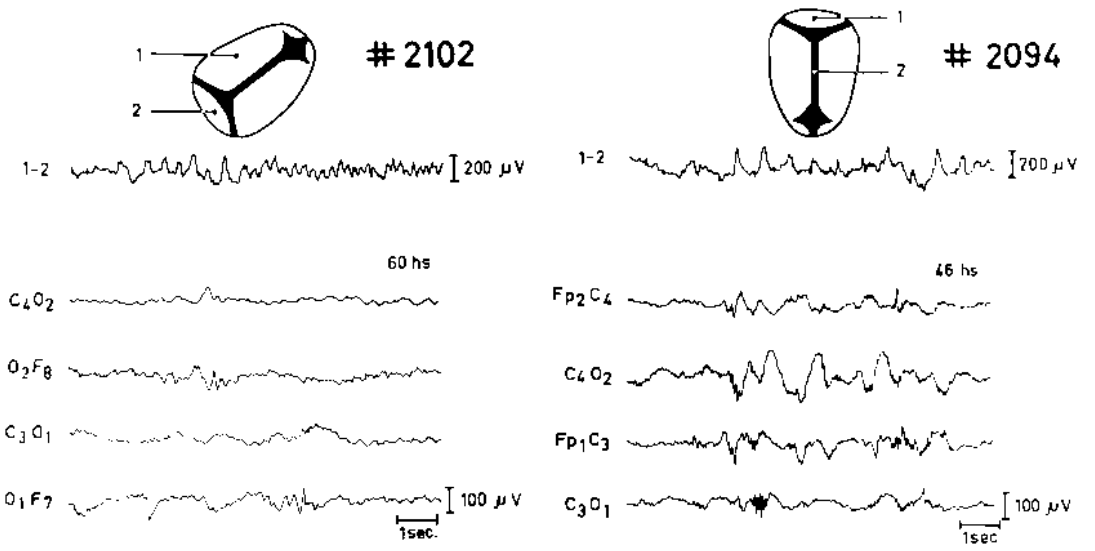

FIGURE 8. Epileptiform activity in fetal and postnatal EEGs. In case 2120, fetal EEG (top, lead indicated) shows rhythmic discharge. Other recordings are multilead EEG registered 60 hours after delivery using 10/20 system for electrode positions; asynchronous sharp waves in both hemispheres. In case 2094, fetal EEG shows waves repeated rhythmically at frequency of 1.5/sec. EEG 46 hours after birth shows rhythmic and random epileptiform activity synchronous and asynchronous in both hemispheres.

ings showing epileptiform activity before and after delivery and the remaining findings, which were all normal.

Discussion

During the uterine contractions and owing to movements of the mother, artifacts were usually recorded, but these can be differentiated by their shape and amplitude from the other slow waves registered during contractions. Moreover, the EEG waves attributed to the effect of contractions were recorded with epidural anesthesia in the absence of pain and mother movements. Artifacts produced by fetal movements that are commonly observed during uterine contractions can be also recognized. The PGR have a different temporal course from that of the slow waves. However, we do not know the characteristics of the electrical activity originating in the fetal cutaneous glands.

It could be postulated that the slow waves recorded on the fetal scalp during contractions are a reflection of the electrical activity of the uterine muscle, or electrohysterogram. Thus, in addition to a slow electrical potential whose time course is concomitant with that of the uterine contraction (*12*) faster activity of 0.5–2/sec of sinusoidal or more or less irregular wave forms has been described (*21*). Sometimes these waves have the same shape as that of the waves recorded on the scalp. However, no electrohysterogram with these characteristics

TABLE 3. Comparative EEG and clinical study of three cases

CASE NO.	FETAL EPILEPTIC ACTIVITY	POSTNATAL EPILEPTIC ACTIVITY					APGAR SCORE		NEUROLOGICAL EXAMINATION	
		1ST	2ND	3RD	4TH	5TH	1ST MIN	5TH MIN	48 HOURS	3–5 DAYS
2035	No	No	No	No	No	—	7	10	Normal	Normal
2094	Yes	Yes	Yes	Yes	Yes	No	1	4	Abnormal	Abnormal
2026	Yes	Yes	Yes	No	—	—	10	10	Normal	Normal

has ever been recorded from the cervix and inferior segment by means of intravaginal electrodes (*22*). Furthermore, the EEG slowing was observed in only 61 per cent of the uterine contractions studied, the electrohysterogram being a constant phenomenon when the contractions are normal, as indeed they were in all the cases studied. Therefore, we believe that the electrical activity recorded on the scalp during the contractions was not of uterine origin.

Epileptiform activity had the same wave shape as in newborn infants and children. The possibility that this activity might be an artifact is eliminated by the fact that it persists after birth with similar characteristics.

The amplitude and frequency of fetal background EEG found in these studies correspond with the descriptions of other authors (*2, 3, 4, 5, 17, 19*). Obviously the EEG slowing is a consequence of uterine contractions even though no relationship between the EEG change and the strength of the contractions was found.

The changes in the EEG of the human fetus reported in this paper occur under conditions in which the fetal head is quite likely being very strongly compressed by the uterine contractions. This transient compression may cause an increase in intracranial pressure, cephalic deformation, and consequent reduction in cerebral blood flow. Ischemia of the brain may explain the occurrence of slow waves and rhythmic discharges in the EEG during the peak of the contractions. The dependency between dips I and the slowing of the EEG in the same stage of labor agrees with this hypothesis, since dips I are known to occur when the fetal head is strongly compressed by uterine contractions (*1, 20*). Moreover, the reduction in brain pO_2 during intracranial hypertension has been demonstrated in animals (*16*) and in the human fetus (*15*). Changes in the EEG pattern similar to those occurring during the peak of uterine contractions have been classically described during brain anoxia in man (*7, 10, 11, 18*). Similar changes have also been reported by Mann (*14*) in the fetal lamb when the ewe is made anoxic by breathing pure nitrogen.

So far there is no evidence that these transient periods of brain ischemia and anoxia may cause permanent damage to the brain. As far as is inferable from the Apgar score of the newborn, there is no correlation between the incidence of dips I during labor and the condition of the newborn (*20*).

In addition to the reduction in brain pO_2 as a possible mechanism of production of slow waves, a direct mechanical action on the brain could also be postulated. The considerable cephalic deformation observed during uterine contractions would seem to support this interpretation.

Dips I were not the cause of the slow waves observed during contractions, because this slowing could be observed in their absence. This eliminates the possibility that decreased heart rate would reduce arterial pressure and blood flow through the brain.

A different problem is involved in the persistence of slow waves and the presence of epileptiform activity immediately after the contractions correlated with dips II. All these fetal signs might also be the result of fetal brain anoxia and/or brain lesion caused by cephalic deformation. These changes were observed only in cases showing a low Apgar score. It should therefore be assumed that in these cases the changes were more marked than in the rest. Moreover, the EEG continued to exhibit epileptiform activity several days after birth. This finding indicates that fetal epileptiform activity observed during labor was caused by abnormal brain conditions that persisted in the newborn. Presumably, after many contractions producing brain anoxia the brain function is consistently disturbed; this makes epileptiform discharges and slow waves appear independently of the contractions.

The presence of epileptiform activity in the fetal EEG is not surprising because the immature brain has a high sensitivity to the hypersynchronous discharge. It is well known that the newborn and the infant develop convulsions with relative ease. In the chick embryo it has been reported (*8*) that EEG epileptic activity

may be present almost simultaneously with the onset of the background electrical activity. The presence of focal cortical seizures with slight or no tendency to propagation is likewise a characteristic of the abnormal EEG in the newborn infant. Hence it is not surprising that the fetus should present similar patterns.

The cases showing discrepancies between EEG and clinical data, of which an example was presented, pose the question of the possible significance of fetal EEG in the management of labor. It is worth noting that in all these cases abnormal EEG patterns were recorded after delivery, a likely indication of important changes in brain function. All the same, in order to gain a better understanding of the future implications of the findings derived from these babies, a more extensive experience is clearly required.

Summary

Fetal EEGs were recorded during advanced labor by means of enamel-insulated platinum needle electrodes inserted in the fetal scalp. They were introduced via the vagina and cervix after rupture of the membranes. The cervical dilatation was greater than 4 cm and the fetal head was beyond station 0. A Grass electroencephalograph was used. Uterine contractions were inscribed by recording the intrauterine pressure. Fetal heart rate was also recorded.

The background EEG activity (recorded between uterine contractions) had a frequency of 2–3/sec and an amplitude of 50–100 μV, occasionally with superimposed faster activity. Apparently spontaneous variations in the patterns were observed.

Most of the uterine contractions provoked consistent EEG changes, which consisted in the appearance of irregular, high-amplitude slow waves of 100–200 μV and 0.5–1 sec. These changes were present in all the fetuses studied.

Some fetuses exhibited random and/or rhythmic epileptiform activity, which also coincided with the uterine contractions. In these cases the newborn EEGs also demonstrated epileptiform activity. This finding indicates that the fetal epileptiform activity observed during labor was caused by abnormal brain conditions that persisted in the newborn.

The changes caused in the fetal EEG by uterine contractions may be explained by a transient episode of ischemia of the fetal brain due to cranial hypertension or deformation (molding) of the fetal head. This interpretation is supported by the obstetrical conditions present (ruptured membranes, advanced cervical dilatation, head deeply engaged in the pelvis), all favoring strong compression of the equatorial zone of the fetal head by the uterine contractions.

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