σ

Papers contributed by staff members
of the
Servicio de Fisiología Obstétrica
Hospital de Clínicas
Montevideo, Uruguay

Selected reprints from

PERINATAL FACTORS AFFECTING HUMAN DEVELOPMENT

Proceedings of the Special Session

held during the Eighth Meeting

of the

PAHO Advisory Committee on Medical Research

10 June 1969

PAN AMERICAN HEALTH ORGANIZATION
Pan American Sanitary Bureau, Regional Office of the
WORLD HEALTH ORGANIZATION

Washington, D.C.

CHANGES IN FETAL HEART RATE ASSOCIATED WITH ACUTE INTRAPARTUM FETAL DISTRESS [1]

C. Méndez-Bauer, J. Monleón, G. Guevara-Rubio, C. Casacuberta, R. Bustos, G. Giussi, L. Escarcena, R. Yabo, and R. Caldeyro-Barcia [2]

In present-day obstetrics, it is very important to diagnose acute intrapartum fetal distress as soon as possible, before irreversible damage is produced to the fetus. Early diagnosis, if followed by immediate treatment, may prevent fetal lesions that would otherwise handicap the individual for the rest of his life.

There are various criteria for assessing the validity of any method of diagnosing acute intrapartum fetal distress. Usually it is accompanied by changes in fetal blood composition (35, 36, 45) and followed by the delivery of a depressed newborn (excluding pharmacological depression). Thus, either of these may be used to diagnose fetal distress (12, 35, 36).

The two phenomena are intimately related. The depression of the newborn (together with some permanent neurological sequelae) is mainly due to damage to fetal cells, particularly those of the nervous system. There are two main causes of fetal cell damage. The abnormal composition of fetal blood during acute intrapartum fetal distress (1, 7, 16, 32, 34, 35, 36, 43, 45), in consequence of impaired exchanges between the mother and the fetus (7), produces fetal

acidosis, hypoxia, hypercapnia, and similar conditions (7, 34, 46, 48, 49). Further, the circulation of fetal blood may not assure an adequate supply of metabolites to all fetal organs and tissues (8, 17, 18, 19, 20, 21, 22, 38). The two causes may function simultaneously.

It follows that the diagnosis of fetal distress can be reliably based on a study of the composition of fetal blood or on the determination of the Apgar score of the newborn (2). A very good correlation between the two criteria has been found (16, 32, 33).

From a practical standpoint, both criteria have drawbacks. The fetal blood composition cannot be analyzed when the obstetrical conditions make fetal sampling impossible; moreover, the information obtained from this procedure is discontinuous and a well-equipped laboratory is necessary. On the other hand, to diagnose fetal distress retrospectively, based on a low Apgar score, has no clinical application.

Our aim is to develop a method for the analysis of the fetal heart rate (FHR) that can be used for an early, reliable, and practical diagnosis of acute intrapartum fetal distress. Monitoring of the FHR can provide a very useful tool for this purpose (5, 6, 8, 10, 27, 28) if it can be proved that changes in the fetal blood composition are related to certain variations in FHR and that these, in turn, are related to the delivery of a depressed newborn.

Abnormal FHR patterns

Changes in FHR, detectable by clinical methods (8), have been found in cases of fetal distress. Possibly as a result of chronically impaired feto-maternal exchanges, transiently aggravated by uterine contractions, the composition of the fetal blood is altered. In these abnormal conditions, several adaptive reactions may occur (8, 13) that apparently tend to minimize the possible harmful consequences of these homeostatic disturbances and to prolong fetal survival. The cardiovascular response of the fetus is one of the most important and best-known of these adaptive reactions and includes changes in FHR, which can be used for the clinical diagnosis of fetal distress (9, 10, 11, 16).

The FHR records obtained from distressed fetuses show a great variety of patterns (6, 9, 10, 11, 15, 27, 28, 29, 35, 37, 38, 39, 41, 42, 43). These patterns combine two main components: (1) sustained and prolonged tachycardia of the baseline (22, 39), and (2) dips II (see below). In combination, these components may give rise to a great variety of syndromes.

Rise of basal FHR over 155 beats/min (tachycardia)

The FHR tracings show at least four different kinds of variations, which have been designated as "small rapid oscillations" (38, 40, 51), "spikes" (25), "transient ascents" (40), and "dips" (14, 24, 39). The baseline upon which these variations are superimposed is the basal FHR (30, 31, 39) (Fig. 1).

The basal FHR, or baseline of the tracings (30), is measured during the intervals between dips, transient ascents, and spikes, and is defined as the average value between the peaks and valleys of the small rapid oscillations usually present in FHR records (Figure 1). During normal labor, the basal FHR averages 143 beats/min (S.E. 2 beats/min) (31). A rise to above 155 beats/min (tachycardia) is considered a sign of fetal distress (5, 8, 31, 35, 36) (Figures 1 and 2).

The tachycardia may appear alone—that is, not be interrupted by dips II or other changes
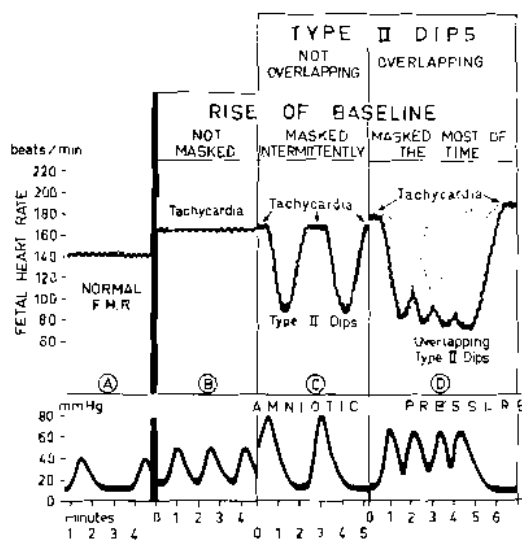

FIGURE 1. Parameters analyzed in tracings of FHR and chronological relationship to uterine contractions (38).

in FHR (Figure 2B). It may be intermittently masked when dips II do occur (Figure 2C). The interruptions in the baseline are more prolonged when several consecutive dips II
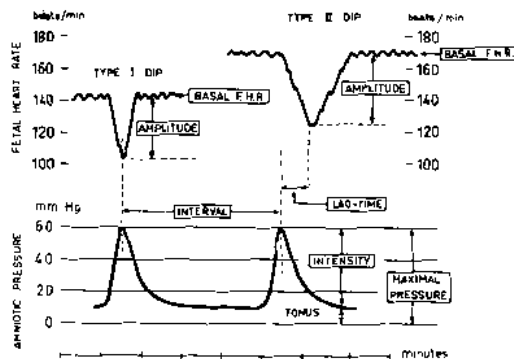

FIGURE 2. Highly schematic representation of basic FHR patterns found in fetal distress. Severity of distress increases from left to right. At bottom, uterine contractions (intraamniotic pressure) assumed to be immediate cause of distress. In A, contractility is normal; in B, moderate uterine tachysystolia; in C, marked uterine hypersystolia; in D, uterine tachysystolia and hypertonus. FHR is normal in A, averaging 140 beats/min; no dips II. Baseline rises abnormally in B, C, and D. In C and D each contraction causes one dip II, totally independent in C and partially overlapped in D. Broken lines in D indicate masked portion (descending and ascending limbs) of individual dips II. Tachycardia of baseline, also masked by overlapping dips II, is indicated similarly (13).

179

overlap (Figure 2C); in these conditions, the tachycardia may be masked most of the time and very difficult to recognize.

Dips II

A dip II is a transient fall in FHR produced by a uterine contraction and occurring some time after it (10, 14, 23, 35, 36). The mean lag-time between the peak of the contraction and the bottom of the dip II is 41 seconds (S.D. = 11 seconds) (Figure 1). A dip I is also a transient fall in FHR caused by a uterine contraction, but it occurs at practically the same time as the contraction. The mean lag-time between the peak of the contraction and the bottom of the dip I is 3.5 seconds (Figure 1). This difference in lag-time is a reliable criterion for distinguishing dips II from dips I. An extended discussion and statistical analysis of this point have already been published (6).

Dips I and dips II are produced by different mechanisms (15, 38), appear under different conditions (3, 24), and have different diagnostic and prognostic significance (3, 8, 31). Dips II are a sign of fetal distress, whereas dips I apparently have no such ominous connotation (10).

Dips II are easily recognized in simultaneous tracings of FHR and uterine contractions and can also be detected by adequate clinical auscultation (8). Their appearance usually coincides with a rise in basal FHR above 155 beats/min (Figure 2C). Dips II may be recorded as individual units, totally independent of each other and clearly recognizable, or they may overlap partially, losing their individuality and merging to produce a more prolonged and marked fall of FHR (Figure 2D).

In addition, dips I and II may be produced by the same uterine contraction. This pattern can easily be recognized from FHR tracings, but its recognition by clinical auscultation alone may be rather difficult.

To confirm the hypothesis that tachycardia and/or dips II are associated with fetal distress, in the following sections the presence of both signs will be studied in relation to the occur-

rence of changes in fetal blood composition and to possible depression of the newborn.

Relationship between variations in FHR and changes in fetal blood composition

Basal FHR and composition of fetal blood

The results of simultaneous studies of fetal blood composition according to Saling's technique (49) and FHR have already been published (7, 35, 36). It can be concluded that the basal FHR increases to over 155 beats/min when fetal pH falls below 7.20 (Figure 3), whereas no clear relationship has been found between changes in basal FHR and pCO_2, base deficit, or oxygenation of the fetal blood.

Dips II and composition of fetal blood

Dips II are usually associated with fetal hypoxia, acidosis and hypercapnia (Figures 4, 5, and 6). They are not associated with an increased base deficit of fetal blood (36).

Relationship between variations in FHR and Apgar score

Complete studies on the relationship between the condition of the newborn, as evaluated by

pH of FETAL CAPILLARY BLOOD		
	Below 7.20	Above 7.20
N = 25	N = 113
x̄ = 165	x̄ = 146
SD = 14	SD = 13
SE = 3	SE = 1
SIGNIFICANT P < 0.001		

$\Delta\bar{x} = 18$
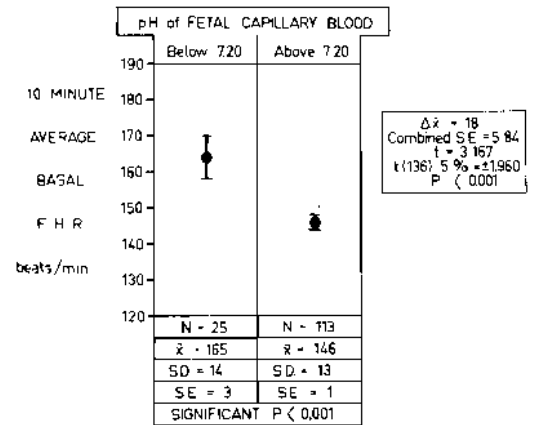Combined S.E. = 5.84
t = 3.167
t(136) 5 % = ±1.960
P < 0.001

FIGURE 3. Average basal FHR in 10-minute period preceding sampling from fetal scalp was measured for 138 samples divided into two groups (pH values over and below 7.20), and group means were calculated. Difference is highly significant (p < 0.001).
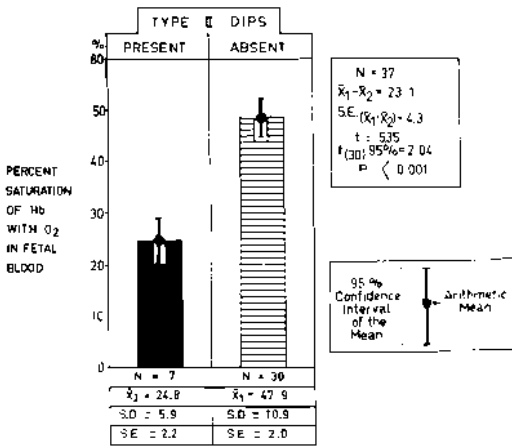
the Apgar score at the first minute of life, and changes in FHR have recently been published (8, 36).

Basal FHR and Apgar score

The group of vigorous newborns (Apgar score 7–10) had an average basal FHR of 143 beats/min, in comparison with an average of 166 beats/min for the "depressed" group (Apgar
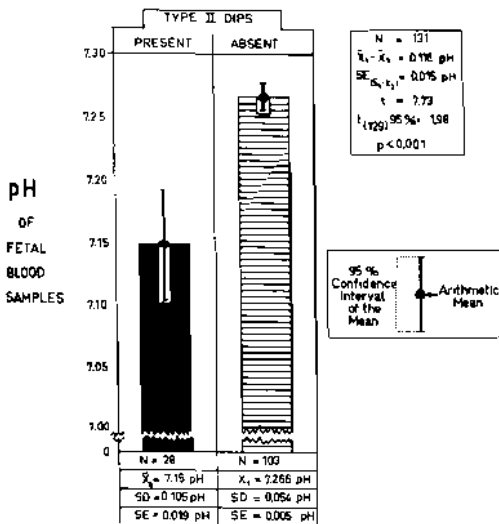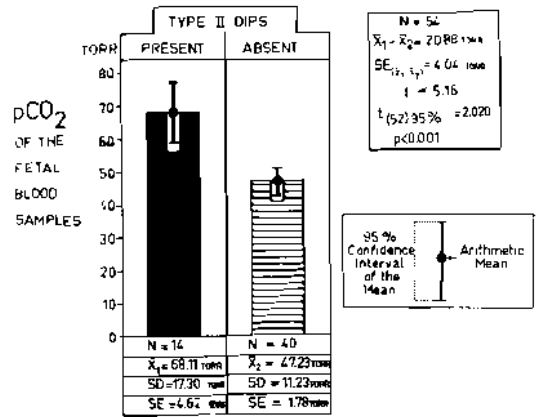
score 1–6). The difference is highly significant. The rise in the baseline above a tentative limit of 155 beats/min is considered a sign of fetal distress. The higher and the more prolonged the rise of the baseline, the worse the prognosis for the infant.

Dips II and Apgar score

Possible correlations were sought between the Apgar score of the newborns at the first minute of life and various quantitative characteristics of the dips II that had occurred in each case.

Mean amplitude of dips II. No significant differences have been found in the mean amplitude of dips II between labors delivering depressed newborns and those delivering vigorous ones. In both groups it was about 20 amplitude units (beats/min).

Total number of dips II. In the group of depressed newborns the average number of dips II recorded during each labor was 91, whereas in the group of vigorous newborns the mean was 11. The difference is highly significant (Figure 7).

It has been suggested (7, 8) that dips II should be virtually absent in a normal labor and that their appearance should be regarded as a sign of fetal distress. However, if the total number of dips II recorded during a complete

181

APGAR SCORE
1st minute

1 to 6 | 7 to 10

$\Delta \bar{x} = 80$
JOINT SE = 17
t = 4.781
$t_{(46)}$ 5% = 2.015
p < 0.001

N = 14 | N = 34
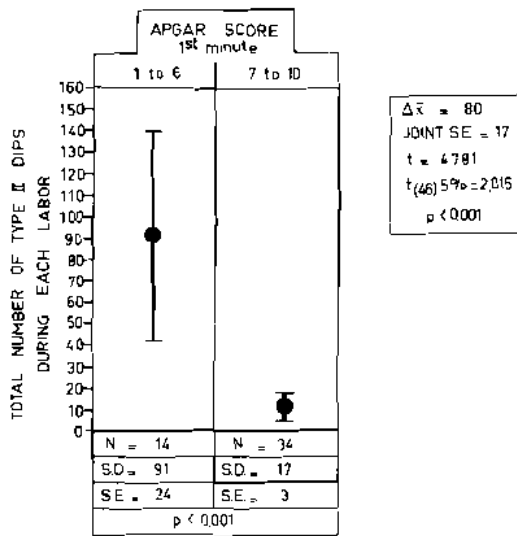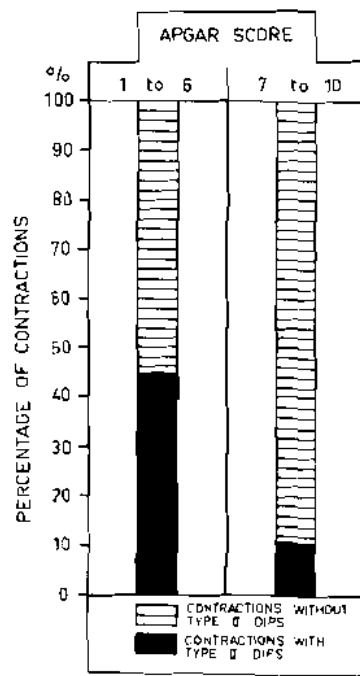S.D. = 91 | S.D. = 17
S.E. = 24 | S.E. = 3
p < 0.001

FIGURE 7. Mean total number of dips II recorded during labor in group of 14 depressed newborns is 91. In group of 34 vigorous infants, mean value is 11. Both mean and fiducial limits (95 per cent) are indicated for each group; difference is significant (p < 0.001) (8).

labor is below a tentative upper limit of 20, the fetal disturbance will not be severe enough to cause a low Apgar score. Above this number the newborn is usually depressed. The few exceptions to this rule were cases in which dips II were recorded in early stages of labor and then disappeared, the FHR tracing being normal for several hours before delivery.

Percentage of contractions causing dips II (Figure 8). In a pool made of all the 3,499 uterine contractions recorded in 34 labors from onset until the delivery of vigorous newborns, only 377 (11 per cent) caused dips II. In the group of 14 labors delivering depressed newborns, 1,273 of the 2,852 contractions recorded (44.6 per cent) produced dips II.

From these results it can be concluded that two signs of FHR (tachycardia of baseline over 155 beats/min and the occurrence of more than 20 dips II) can be used for a reliable diagnosis of fetal distress. In particular, dips II are associated with hypoxia, hypercapnia, and low actual pH of fetal blood; when more than 20 of them occur during labor, the newborn is usually depressed.

CONTRACTIONS WITHOUT TYPE II DIPS
CONTRACTIONS WITH TYPE II DIPS

UTERINE CONTRACTION	APGAR SCORE		
	1 to 6	7 to 10	TOTAL
WITHOUT TYPE II DIPS	1,579	3,122	4,701
WITH TYPE II DIPS	1,273	377	1,650
TOTAL	2,852	3,499	6,351

P < 0.001

FIGURE 8. In 14 labors delivering depressed newborns, 45 per cent of contractions caused dips II; only 10 per cent caused dips II in 34 labors delivering vigorous newborns (χ^2 test, p < 0.001). Total number of contractions analyzed was 6,351 (8).

A practical test for evaluating fetal condition in acute intrapartum fetal distress, based on dips II

The difficulty with using the criterion of 20 or more dips II during labor is that by the time

distress is diagnosed the fetal damage has probably already occurred. The following study was carried out with the aim of finding a way to detect as early as possible any departure from normal in the condition of the fetus.

Material and methods

In a retrospective study of the available records of labor at the Obstetrical Physiology Service in Montevideo, Uruguay, 20 cases were found to have a good FHR tracing in the period immediately preceding the second stage or intrapartum fetal death. These 20 newborns were classified, according to the Apgar score at the first minute of life, into three groups: I (10 newborns), with a score of 7 to 10; II (5 newborns), with a score of 4 to 6; and III (5 newborns), with a score of 0 to 3. Group III included two fetuses who died during labor. Cesarean sections were excluded. Except in one case (No. 1500), in which the umbilical cord was found at birth to be loosely wound around the fetal neck, no cord complications were known to exist.

Several characteristics of dips II were studied in the 20 contractions that preceded the second stage of labor, in order to determine whether any of them were related to the delivery of a depressed newborn.

Results and discussion

Dips II were rare in group I. They did occur in groups II and III, but there was no significant difference in amplitude between the two groups (Figure 9). Thus amplitude cannot be used for predicting the condition of the newborn.

The incidence of dips II was also studied by calculating what percentage of the 20 contractions produced them. In group I (Figure 10) this percentage was 5.5 per cent. The fiducial limits statistically calculated (99 per cent) ranged between 1.5 and 10 per cent. This implies that when up to 10 per cent of the contractions cause dips II, the fetus—if immediately delivered—will have an Apgar score of
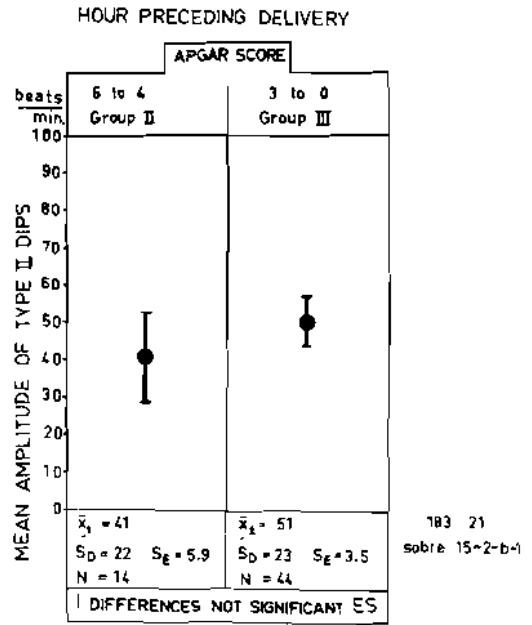

FIGURE 9. Comparison of mean amplitude of dips II in Groups II and III reveals no significant differences (37).

7 to 10 (with 99 per cent confidence). In group II the percentage of contractions causing dips II was 21 per cent (Figure 11). The fiducial limits calculated (99 per cent) ranged from 11 to 31 per cent. Within this range the newborns will be moderately depressed (Apgar score 4–6). In group III a mean of 44 per cent
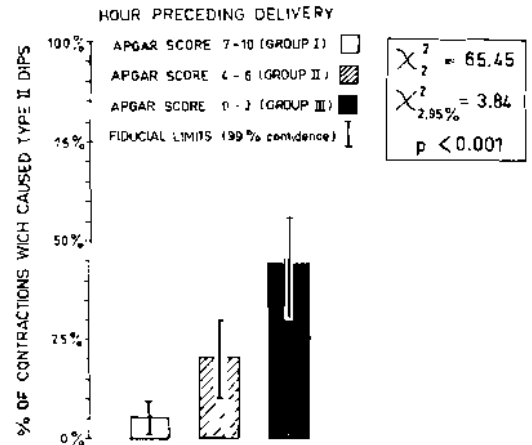

FIGURE 10. Percentages of 20 contractions preceding second stage that caused dips II in Groups I, II, and III. In χ^2 test with two degrees of freedom, differences are highly significant (p<0.001) (37).
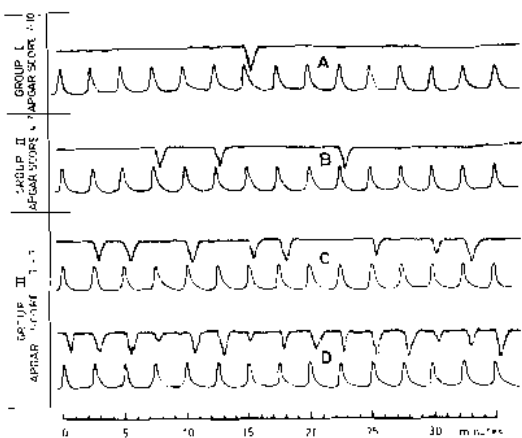
FIGURE 11. Schematic representation of records of FHR and amniotic pressure. Severity of fetal distress increases from top to bottom. At left, theoretical Apgar score (if fetus were delivered immediately). Percentage of contractions causing dips II may indicate, at any moment of labor, condition of fetus (37).

of the contractions produced dips II. The 99 per cent fiducial limits for this group ranged from 32 to 56 per cent. Thus, if more than 32 per cent of the contractions cause dips II the fetus will be delivered extremely depressed or will die during labor.

The percentages of uterine contractions causing dips II in each group were studied by means of a χ^2 test with two degrees of freedom. The difference between these percentages was found to be highly significant ($p < 0.001$) (Figure 10). These differences cannot be accounted for by differences between groups in the maximal pressure of the contractions; all 20 fetuses were exposed to contractions of similar maximal pressure.

In summary, a study of 15 to 20 contractions prior to the second stage of labor to determine what percentage of them produces dips II is enough to enable the obstetrician to predict the Apgar score of the newborn if it is delivered immediately; the amplitude of the dips II, as has been said, may be ignored. The contractions to be considered should have a maximal pressure ranging between 30 and 70 mm Hg, such as are usually found during labor.

If such a test served only to predict the condition of the newborn a few minutes before delivery, its usefulness would obviously be limited. However, these results can be used to develop a test whereby the clinician can diagnose acute intrapartum fetal distress at any moment during labor.

It may be predicted that fetus A in Figure 11, if immediately delivered, would have an Apgar score of 7–10, since less than 10 per cent of the contractions (6.6 per cent) have produced dips II and consequently its condition can at the moment be described as excellent. Fetus B, with 20 per cent of the contractions producing dips II, would have an Apgar score of 4–6; thus, it is in distress. Fetuses C and D are extremely distressed, as can be suspected from the fact that more than 30 per cent of the contractions are producing dips II; they will die very soon, or be severely depressed if immediately delivered.

Practically speaking, it is enough to monitor 15 consecutive uterine contractions with simultaneous auscultation of the fetal heart and uterine palpation. The method to enable the obstetrician to detect dips II easily by clinical procedures has been published elsewhere (8).

For each contraction, the presence or absence of dips II should be represented on a chart similar to that shown in Figure 12. If a contrac-

CLINICAL TEST FOR EVALUATING FETAL CONDITION
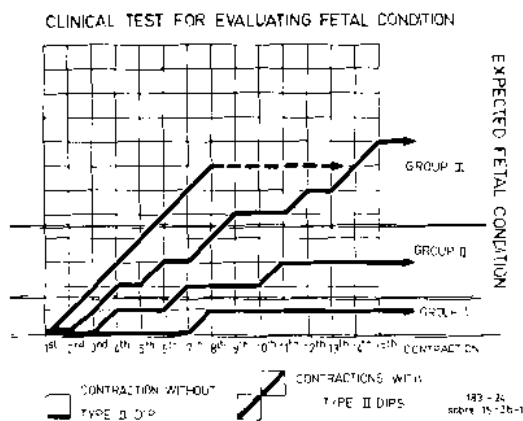
FIGURE 12. Double horizontal lines define groups I, II, and III (expected good, poor, or extremely severe fetal condition). Each vertical line corresponds to one monitored contraction. Heavy lines represent presence (diagonal) or absence (horizontal) of dips II; thus position of continuous line shows after each contraction the condition of the fetus. Number of contractions required to achieve diagnosis of acute intrapartum fetal distress decreases as fetal condition gets worse.

tion does not produce a dip II, a horizontal line is drawn in the square corresponding to it; if it does, an oblique line. If no dips II are detected in the 15 consecutive contractions studied, a continuous horizontal line will be superimposed on the bottom line of Figure 12. One dip II found among the 15 contractions will raise the line slightly, but the fetus will still be in good condition because the percentage (6.6) is within the limits of group I. If 3 or 4 dips II occur in the 15-contraction period, the fetus is distressed and, if immediately delivered, would be depressed (Apgar score 4-6). If more than 4 occur, the fetus is in extremely severe distress and would have a very low Apgar score (0-3).

It can be observed that the number of contractions to be studied diminishes with the severity of fetal distress. For instance, in situation D of Figure 11, in which all the contractions produce dips II, it is unnecessary to wait

for the end of the series of 15, because the percentage is already over 47 by the time of the seventh consecutive contraction. At the usual frequency of 3 to 5 contractions every 10 minutes, the diagnosis of severe distress can thus be made in 12 to 20 minutes. This is the case of the fetus represented by the top line of Fig. 12, in which, after the seventh contraction, it can be stated that the fetus is definitely within the area of group III. The dotted line from the seventh contraction onwards emphasizes the fact that the diagnosis of extremely severe fetal distress has already been made. If this situation is allowed to continue, the consequent fetal damage will be increasingly aggravated; if its cause is corrected by adequate treatment as soon as fetal distress has been recognized, the percentage of contractions causing dips II will diminish, showing the improvement of the fetal condition.

REFERENCES

1. ALTHABE, O., JR., R. L. SCHWARCZ, S. V. POSE, L. ESCARCENA, and R. CALDEYRO-BARCIA. Effects on fetal heart rate and fetal pO_2 of oxygen administration to the mother. *Am. J. Obst. & Gynec.* 98: 858, 1967.

2. APGAR, V. A proposal for a new method of evaluation of the newborn infant. *Current Researches in Anesthesia and Analgesia.* Jul.-Aug. 1953, 260-267.

3. ARAMBURU, G., L. GULIN, and R. CALDEYRO-BARCIA. Relations between the incidence and amplitude of type I "dips" and Apgar score in the human fetus. In R. Caldeyro-Barcia, C. Méndez-Bauer, and G. S. Dawes (eds.), *Effects of Labor on the Fetus and the Newborn* (Symposium held in Montevideo, 1964). Oxford, Pergamon Press, in press.

4. ASSALI, N. S., and H. A. MORRIS. Maternal and fetal circulations and their interrelationships. *Obst. & Gynec. Surv.* 19:923, 1964.

5. BRADY, J., L. S. JAMES, and M. S. BAKER. Fetal electrocardiographic studies. Tachycardia as a sign of fetal distress. *Am. J. Obst. & Gynec.* 86:785, 1963.

6. CALDEYRO BARCIA, R. Effects of uterine pressure on the fetus. In *Neonatal Respiratory Adaptation.* Princeton, U.S. Department of Health, Education and Welfare, Public Health Service, 1963.

7. CALDEYRO-BARCIA, R., C. CASACUBERTA, R. BUSTOS, G. GIUSSI, L. GULIN, L. ESCARCENA, and C. MÉNDEZ-BAUER. Correlation of intrapartum changes

in fetal heart rate with fetal blood oxygen and acid base balance. In K. Adamsons (ed.), *Diagnosis and Treatment of Fetal Disorders.* New York, Springer, 1968, pp. 205-225.

8. CALDEYRO-BARCIA, R., A. A. IBARRA-POLO, L. GULIN, J. J. POSEIRO, and C. MÉNDEZ-BAUER. Diagnostic and prognostic significance of intrapartum fetal tachycardia and type II dips. In *Symposium on Prenatal Life, Detroit, Michigan, Nov. 1-2, 1967.* Springfield, Illinois, Charles C Thomas, 1968.

9. CALDEYRO-BARCIA, R., C. MÉNDEZ-BAUER, J. J. POSEIRO, L. A. ESCARCENA, S. V. POSE, I. C. ARNT, L. GULIN, O. ALTHABE, JR., and J. BIENIARZ. Bases para el diagnóstico precoz del sufrimiento fetal intraparto. *Gac. Med. Méx.* 96:687, 1966.

10. CALDEYRO-BARCIA, R., C. MÉNDEZ-BAUER, J. J. POSEIRO, L. A. ESCARCENA, S. V. POSE, J. BIENIARZ, I. C. ARNT, L. GULIN, and O. ALTHABE, JR. Control of human fetal heart rate during labor. In D. E. Cassels (ed.), *The Heart and Circulation in the Newborn and Infant.* New York, Grune & Stratton, 1966, p. 7.

11. CALDEYRO-BARCIA, R., C. MÉNDEZ-BAUER, J. J. POSEIRO, S. V. POSE, L. A. ESCARCENA, J. BIENIARZ, O. ALTHABE, JR., and L. GULIN. La fréquence du coeur foetal pendant l'accouchement. Signification de ses variations. *Bull. Fed. Soc. Gyn. et Obstét.* 18: 395, 1966.

12. CALDEYRO-BARCIA, R., C. MÉNDEZ-BAUER, J. J. POSEIRO, and S. V. POSE. La respuesta cardiovascular en el "Sufrimiento Fetal". Presented to II Reunión ALIRH. Viña del Mar, Chile, Nov. 20–24, 1966.

13. CALDEYRO-BARCIA, R., J. J. POSEIRO, C. MÉNDEZ-BAUER, and L. GULIN. Effects of abnormal uterine contractions on fetal heart rate during labor. Presented to V World Congress of Gynecology & Obstetrics, Sydney, Australia, 1967.

14. CALDEYRO-BARCIA, R., J. J. POSEIRO, C. NEGREIROS DE PAIVA, C. GÓMEZ-ROGERS, A. FAÚNDES-LATHAM, M. A. ZAMBRANA, G. ARELLANO-HERNÁNDEZ, A. BEAUQUIS, P. PEÑA-ORTIZ, F. AGÜERO-LUGONES, and W. FULLER, JR. Effects of abnormal uterine contractions on a human fetus. Mod. Probl. Pediat. 8:267, 1963.

15. CALDEYRO-BARCIA, R., J. J. POSEIRO, S. V. POSE, C. MÉNDEZ-BAUER, J. BIENIARZ, C. GÓMEZ-ROGERS, and A. FAÚNDES-LATHAM. Evaluation of fetal condition by means of fetal heart rate. In R. Caldeyro-Barcia, C. Méndez-Bauer, and G. S. Dawes (eds.), Effects of Labor on the Fetus and the Newborn (symposium held in Montevideo, 1964). Oxford, Pergamon Press, in press.

16. CRAWFORD, J. S. Maternal and cord blood at delivery. Biol. Neonat. 8:131, 1965.

17. DAWES, G. S. Effects of anoxia on newborn animals. In T. K. Oliver, Jr. (ed.), Adaptation to Extrauterine Life: Report of the Thirty-first Ross Conference on Pediatric Research. Columbus (Ohio), Ross Laboratories, 1959, p. 40.

18. DAWES, G. S. Placental development and umbilical blood flow. J. Obst. & Gynec. Brit. Cwlth. 69:815, 1962.

19. DAWES, G. S. Pulmonary circulation in the foetus and the newborn. Ciba Foundation Symposium on Development of the Lung. New York, Ciba, 1966, p. 332.

20. DAWES, G. S. The umbilical circulation. Am. J. Obst. & Gynec. 84: 1634, 1962.

21. DAWES, G. S., H. N. JACOBSON, J. C. MOTT, J. HEATHER, and H. J. SHELLEY. Some observations on foetal and newborn rhesus monkeys. J. Physiol. 151:271, 1960.

22. DAWES, G. S., J. C. MOTT, and H. J. SHELLEY. The importance of cardiac glycogen for the maintenance of life in foetal lambs and newborn animals during anoxia. J. Physiol. 146:516, 1959.

23. FAÚNDES-LATHAM, A., A. BEAUQUIS, F. AGÜERO-LUGONES, A. GONZÁLEZ DE GIMÉNEZ, and R. CALDEYRO-BARCIA. Influencia del progreso del parto sobre el efecto que las contracciones uterinas tienen en la frecuencia cardiaca fetal. Presented to IV Congr. Mexicano de Ginecología y Obstetricia, junio 1963.

24. FERNÁNDEZ-FUNES, J., L. O. ALVAREZ, and J. J. POSEIRO. "Dips" tipo II. IV Congr. Uruguayo de Ginecotocología, Montevideo 2:867, 1964.

25. FIGUEROA, J. G., G. ARAMBURÚ, C. MÉNDEZ-BAUER, A. MOGGIA, and R. CALDEYRO-BARCIA. Las espicas de la FCF. IV Congr. Uruguayo de Ginecotocología. Montevideo 2:852, 1964.

26. GULIN, L. A., J. J. POSEIRO, S. V. POSE, C. MÉNDEZ-BAUER, L. A. ESCARCENA, I. C. ARNT, J. BIENIARZ, and R. CALDEYRO-BARCIA. Sofrimento fetal. Patologia. Presented to VII Congr. Bras. Obst. & Ginec., Recife, 1966.

27. HESS, O. W. and E. H. HON. The electronic evaluation of foetal heart rate. III. The effect of an oxytocic agent used for the induction of labor. Am. J. Obst. & Gynec. 80:558, 1960.

28. HON, E. H. The diagnosis of fetal distress. Clin. Obst. & Gynec. 3:860, 1960.

29. HON, E. H., A. H. BRADFIELD, and O. W. HESS. The electronic evaluation of the fetal heart rate. V. The vagal factor in fetal bradycardia. Am. J. Obst. & Gynec. 82:291, 1961.

30. HON, E. H. and R. CALDEYRO-BARCIA. Scientific Exhibit at the 16th Annual Meeting ACOG, Chicago, 1968.

31. IBARRA-POLO, A., A. QUINTANILIA, O. ALTHABE, JR., and R. CALDEYRO-BARCIA. Relations between basal fetal heart rate, type II "dips" and Apgar score in the human fetus. In R. Caldeyro-Barcia, C. Méndez-Bauer, and G. S. Dawes (eds.), Effects of Labor on the Fetus and the Newborn (symposium held in Montevideo, 1964). Oxford, Pergamon Press, in press.

32. JAMES, L. S., I. M. WEISBROT, C. E. PRINCE, D. A. HOLADAY, and V. APGAR. The acid-base status of human infants in relation to birth asphyxia and the onset of respiration. J. Pediat. 52:379, 1958.

33. KUBLI, F. and D. BERG. Acid-base balance of amniotic fluid and fetus before and during labor in the human. In R. Caldeyro-Barcia, C. Méndez-Bauer, and G. S. Dawes (eds.), Effects of Labor on the Fetus and the Newborn (symposium held in Montevideo, 1964). Oxford, Pergamon Press, in press.

34. KUBLI, F. and D. BERG. Fetal distress. In H. Ewebeck and V. Friedberg (eds.), Die Übergangstrorungen des Neugeborenen und die Bekaampfung der Perinatalen Mortalität. Stuttgart, Georg Thieme, 1965.

35. MÉNDEZ-BAUER, C., I. C. ARNT, L. GULIN, L. A. ESCARCENA, and R. CALDEYRO-BARCIA. Relationship between blood pH and heart rate in the human fetus during labor. Am. J. Obst. & Gynec. 97:530, 1967.

36. MÉNDEZ-BAUER, C., C. CASACUBERTA, R. BUSTOS, G. GIUSSI, L. ESCARCENA, L. A. GULIN, A. IBARRA-POLO, J. J. POSEIRO, and R. CALDEYRO-BARCIA. Fetal acid base balance and heart rate under physio-

logical and pathological conditions. *Proceedings of the Symposium on Fetal Growth and Development*, San Diego, California, 1968.

37. MÉNDEZ-BAUER, C., G. GUEVARA-RUBIO, J. MONTFÓN, A. CARRILLO-ANDRADE, R. YABO, and R. CALDEYRO-BARCIA. A practical approach for evaluating fetal condition during labor, by the fetal heart rate. *Proceedings of the Symposium on Fetal Growth and Development*, San Diego, California, 1968.

38. MÉNDEZ-BAUER, C., J. J. POSEIRO, G. ARELLANO-HERNÁNDEZ, M. A. ZAMBRANA and R. CALDEYRO-BARCIA. Effects of atropine on the heart rate of the human fetus during labor. *Am. J. Obst. & Gynec.* 85:1033, 1963.

39. MÉNDEZ-BAUER, C., J. J. POSEIRO, A. FAÚNDES-LATHAM, F. AGÜERO-LUGONES, and A. BEAUQUIS. Basis for the study of FHR during labor. In R. Caldeyro Barcia, C. Méndez-Bauer, and G. S. Dawes (eds.), *Effects of Labor on the Fetus and the Newborn* (symposium held in Montevideo, 1964). Oxford, Pergamon Press, in press.

40. MOGGIA, A. V., G. ROMERO-SALINAS, and C. MÉNDEZ-BAUER. Rapid fluctuations and transient ascents of the human FHR during labor. In R. Caldeyro-Barcia, C. Méndez-Bauer, and G. S. Dawes (eds.), *Effects of Labor on the Fetus and the Newborn* (symposium held in Montevideo, 1964). Oxford, Pergamon Press, in press.

41. POSEIRO, J. J., A. FAÚNDES-LATHAM, C. GÓMEZ-ROGERS, and G. ARELLANO-HERNÁNDEZ. Efectos de la hipercontractilidad uterina sobre la frecuencia cardiaca fetal. Cambios que preceden a la muerte fetal. *V Congr. Med. del Uruguay* 4:1799, 1962.

42. QUILLIGAN, E. J. and E. B. KATIGBAK. Correlation of fetal heart rate and acid-base balance at birth. III. Tachycardia. *Obst. & Gynec.* 25:371, 1965.

43. QUILLIGAN, E. J., E. B. KATIGBAK, and J. HOFSCHILD. Correlation of fetal heart rate patterns and blood gas values. II. Bradycardia. *Am. J. Obst. & Gynec.* 91:1123, 1965.

44. ROOTH, G., S. SJÖSTEDT, and F. CALIGARA. The hydrogen concentration, carbon dioxide tension and acid-base in the blood of the human umbilical cord and intervillous space of the placenta. *Arch. Dis. Childhood*, 36:278, 1961.

45. SALING, E. Die Blutgasverhaltnisse und der Saure-Basen-Haushalt des Feten bein ungestortens. *Geburtsavlauf. Z. Geburtsh. u. Gynak.* 161:262, 1963.

46. SALING, E. Mikroblutuntersuchungen am Feten. (Klinischer Einsatz und erste Ergebnisse). *Z. Geburtsh. u. Gynak.* 162:56, 1964.

47. SALING, E. Technik der endoskopischen Microblutentnahme am Feten. *Geburtsh. u. Frauenheil.* 24:464, 1964.

48. SALING, E. A new method of safeguarding the life of the foetus before and during labor. *J. Intern. Fed. Gynaec. Obstet.* 3:101, 1965.

49. SALING, E. Das Kind im Bereich der Geburtshilfe, eine Einfuhrung in ausgewahlte aktuelle Fragen. Stuttgart, Georg Thieme, 1966.

50. SIGGAARD ANDERSEN, O. Blood acid base alignment nomogram. *Scand. J. Clin. & Lab. Invest.* 15: 211, 1963.

51. SWARTWOUT, J. R., W. E. CAMPBELL, JR., and L. G. WILLIAMS. Observations on the fetal heart rate. *Am. J. Obst. & Gynec.* 82:301, 1961.