Fetal distress: role of cardiotocography

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Summary: The FHR monitoring in 320 patients with fetal distress were randomly analyzed and revised over 16 years. The aim of our study, in so long a period, was to evaluate the role of cardiotocography in order to preserve the fetus from irreversible damage.

The results show a progressive improvement of neonatal outcome, due to the development of the experience with this method, and to the improvement in interpretative criteria. However, cardiotocography showed its limits, and the moment has come to seek new integrative methods to associate cardiotocography with a continuous monitoring of the fetal status.

INTRODUCTION

Past and recent studies have shown that the FHR monitoring by means of cardiotocography has a high negative predictive value, whereas the positive predictive value concerning neonatal depression is lower ([1]). The clinical problem with the CTG consists in the possibility of overvaluating the fetal distress, thus resulting in unnecessary interventions. Some Authors actually consider this phenomena as due to an incorrect interpretation of the cardiotocographic data, and moreover to the fact that, among the FHR alterations, deceleration is the only one to which prognostic importance is prevalently given.

Some authoritative clinical observations ([2]), suggest that the specificity of CTG might be improved, if the FHR variability, were to be valued as well as the decelerations; according to these Authors we may consider that a fetus with normal FHR variability is at very low risk of death from asphyxia. A normal O_2 rate in the cardioregulation area, which is responsable for the FHR variation and thus for normal variability, means that the brain is normally oxygenated and therefore in a condition either of normality or of physiological compensation. On the contrary, a hypoxemia occurring in this area, causes a loss of variability and reveals a condition of decompensation. Anyway, the above mentioned considerations are still being debated ([3, 4, 5, 6]). Studies carried out at the Obstet. and Gynec. Depart. of Parma University, only partly confirm these statements. Clinical experience shows that the depression of FHR variability is a much more usual event than deceleration or bradycardia.

The FHR variability may be depressed by several causes not identifying themselves with asphyxia. Congenital SNC diseases, drugs SNC depressors, cardiac congenital blocks, prematurity, fetal sleep,

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are some examples. There is also a certain number of fetuses in physiological conditions, having tendency to show depression of variability, even in the last weeks of pregnancy ([7]). It is still true that different degrees of depression can be recognized (type 1 and type 0 tracings), having different clinical and prognostic significance; it is also necessary to consider the persistence and increase of the loss of variability. We think that the problem of defining fetal distress in its different phases (compensation, decompensation, preterminal) may have implications becoming gradually more complicated, which cannot be completely deduced from the modifications of variability.

In order to put the problem into a larger perspective, we carried out a critical revision of the cardiotocographic data dealing with fetal distress, that we termed more generically « fetal suffering », over a period of 16 years.

MATERIALS AND METHODS

In the period ranging from 1972 to 1987 we considered at random 320 cases of fetal suffering which involved a cesarean section as treatment.

This period was divided into four quadriennia: 1972-75, 1976-79, 1980-83, 1984-87. The cases evaluated for each quadriennium were 80. For each case we considered the obstetrical pathology complicating pregnancy, the eventual course of labor, the presence of cardiotocographic monitoring, the neonatal outcome and the short term neonatal follow-up. The cardiotocographic tracings were reexamined and interpretated by means of Fischer score ([8]), which provides for a quantitative evaluation of the fetal well-being. For each tracing the FHR was analysed for each of the 4 principal parameters: level, variability, reactivity, presence or not of decelerations. Besides we calculated the number of single alterations for each parameter above mentioned. More than 95% of tracings were carried out by an HP ultrasonographic monitor; in the remaining 5% the recording of FHR was obtained by means of phonocardiography or electrocardiography. The results were divided among the four quadriennia considered and confronted.

RESULTS

Of the 320 cases considered over the 16 year period, 70% showed fetal distress in association with an obstetrical pathology complicating pregnancy or labour. Tab. 1 shows the variability of this pathology: we must consider the possibility that several pathologies may have occurred in association in a single case and variably contributed to the fetal distress. We may observe that in addition to the clinical manifestations notoriously linked to the fetal hypoxia, as the placental bleeding, pre-eclampsia, diabetes, there are pathologies whose association with the fetal distress may be casual. It is interesting how in a quite long period of time such as the one considered, some concepts regulating the diagnostic and therapeutical approach in conditions of presumption of fetal distress have changed. For example the diagnosis of hypoestrioluria which sustained a condition of fetus-placental insufficiency surveyed with endocrine control, was present only in the first quadriennium, then it was replaced by the intrauterine poor fetal growth, an event of prevalently clinical and biometrical relief.

For each quadriennium considered, we noticed a share of fetal suffering we termed « sine causa », ranging from 23% to to 37%. As regard these cases, there was obstetrical pathology either in probable relation to the fetal distress, or in casual association. In this group the proportion of depressed neonates was equal to 30%; 100% of these had a pathological or uncertain Fischer score.

We reexamined all the tracings effected, evaluating the percentage of the alterations for the single FHR parameter and the presence of decelerations classified into their different types. These data are reported in Tab. 2, showing that the alteration most represented is the reduction of the FHR variability. Apart from variability the reduction or the loss of reactivity is the most frequent alteration, fol-

Table 1. — *Obstetric complications in population studied.*

Pathology in pregnancy	QUADR. 1		QUADR. 2		QUADR. 3		QUADR. 4	
	n.	%	n.	%	n.	%	n.	%
Pre-eclampsia	9	11.25	9	11.25	3	3.75	5	6.25
Diabetis	9	11.25	4	5.00			2	2.50
RPM	19	23.75	10	12.50	16	20.00	12	15.00
Placental bleeding	5	6.25	3	3.75	1	1.25	2	2.50
Oligo-anhydramnios	3	3.75	4	5.00	1	1.25	4	5.00
Polyhydramnios	1	1.25			2	2.50	2	2.50
Poor history	4	5.00	9	11.5	10	12.50	8	10.00
Maternal hyperthermia	1	1.25	3	3.75			1	1.25
Previous Caesarean section	4	5.00	4	5.00	2	2.50	1	1.25
Amniotitis							3	3.75
Anemia	1	1.25	1	1.25				
Hypoestrioluria	3	3.75						
PIFG			5	6.50	4	5.00	5	6.25
Psychosis			1	1.25	2	2.50		
Obesity			2	2.50			1	1.25
Isoimmunization anti D	1	1.25						
Threatened rupture of uterus			1	1.25				
Uterine dyskinesis	7	8.75	6	7.50	2	2.50	7	8.75
Umbilical cord pathology	5	6.25	5	6.25	5	6.25	9	11.25
Abnormal presentations	6	7.50	5	6.25	3	3.75	4	5.00
Fetal distress	19	23.75	19	23.75	26	32.50	30	37.50
Others	1	1.25	1	1.25				

Table 2. — *Cardiotocographic alterations in each quadriennium.* (1st quadr.: 26 cases; 2nd quadr.: 76 cases; 3rd quadr.: 80 cases; 4th quadr.: 80 cases).

Altered CTG	QUADR. 1		QUADR. 2		QUADR. 3		QUADR. 4	
	n.	%	n.	%	n.	%	n.	%
Variability decrease	22	84.61	47	61.84	53	62.25	61	76.26
Hyporeactivity	7	26.92	38	50.00	47	58.75	54	67.50
Early decelerations	2	7.69	5	6.57	9	11.25	2	2.50
Variable decelerations	16	61.53	30	39.47	27	33.75	25	31.25
Late decelerations	1	3.84	15	19.73	16	20.00	16	20.00
Unclassified deceleration	2	7.69			8	10.00	11	13.75
Bradycardia	14	53.84	18	23.68	7	8.75	12	15.00
Tachycardia	7	26.92	15	19.73	9	11.25	10	12.50

Table 3. — *Meconium contaminated amniotic fluid and pathological Fisher's score rate.*

	QUADR. 1		QUADR. 2		QUADR. 3		QUADR. 4	
	n.	%	n.	%	n.	%	n.	%
Amniotic fluid with meconium	47	58.75	31	38.75	26	32.50	32	40.00
Fisher's score 7-5	23	88.46	54	71.05	65	81.25	56	70.00
Fisher's score 5	3	11.53	20	26.31	13	16.25	22	37.50

lowed by variable decelerations late decelerations, bradycardia and tachycardia in association, early decelerations and those unclassified. Obviously almost the total number of cases showed several alterations in a single tracing.

Data referring to the first 4-year period slightly differ from the others, perhaps because a large number of them was obtained with different recording techniques (phonocardiography).

Each tracing was then reinterpreted using the Fischer score. In Tab. 3 are visualized data relating to this interpretation, together with others relating to the presence of meconium in the amniotic fluid.

In a percentage ranging from 70% to 88% cesarean section was effected in the presence of FHR alterations having uncertain prognostic significance (F.S. = 7-5), while cases ranging from 11% to 27% underwent the intervention because of pathological alteration (F.S. < 5).

In Tab. 4 are represented data relating to the neonatal outcome, in terms of percentage of prematurity, neonatal depression (Apgar score < 7 at 5′), weight < 10 percentile (SGA) and transfers to intensive therapy within the first 24 hours of life. Prematurity shifted from 12.5% in the first 4 year period to 8.75%; in the meantime we recorded an increase in interventions even in conditions of higher prematurity (under 34 weeks) (Tab. 5). The absence of gestational ages preceding the 35th week in the first quadriennium is probably connected with the poor cardiotocographic monitoring at that time.

It is interesting to remark the evident decrease in the percentage of neonatal depression at 5′, shifting from 41% in the first quadriennium to 30% in the fourth; yet the datum is not statistically significant ($p < 0.05$). The transfers to intensive therapy within 24 hours decrease to 10% in the fourth quadriennium compared with 18% of the first, 20% of the second and 13% of the third.

The percentage of neonatal deaths shows a trend towards reduction, with total absence of intrapartum or intrauterine death, which confirms the accuracy

Table 4. — *Newborn outcome.*

Newborn outcome	QUADR. 1		QUADR. 2		QUADR. 3		QUADR. 4	
	n.	%	n.	%	n.	%	n.	%
Premature birth	10	12.50	11	13.75	11	13.75	7	8.75
Apgar < 7 at 5 min.	33	41.25	26	32.50	21	26.25	24	30.00
SGA	18	22.50	17	21.25	15	18.75	19	23.75
Weight < 1500 gr	2	2.50					2	2.50
Transferred within 24 h	15	18.75	16	20.00	11	13.75	8	10.00
Deaths	2	2.50	6	7.50	1	1.25		

Table 5. — *Prematurity in population studied.*

Week	QUADR. 1		QUADR. 2		QUADR. 3		QUADR. 4	
	n.	%	n.	%	n.	%	n.	%
37th	3	3.75	2	2.50	3	3.75	2	2.50
36th	5	6.25	2	2.50	5	6.25	3	3.75
35th	2	2.50	3	3.75			1	1.25
34th			3	3.75	2	2.50	1	1.25
33rd			1	1.25	1	1.25		
32nd			1	1.25				
31st					1	1.25	1	1.25

of the diagnostic survey and the timeliness of the interventions, whereas the most variable neonatal datum must be framed into the global neonatal pathology, summarized in Tab. 6. The data also presented focus on a group of neonates showing a diagnosis of "intrapartum asphyxia" which is, of course, a pediatric diagnosis effected after birth. The datum is, however, interesting, as it concerns the group having, after birth, persistent effect of distress previously surveyed. In others words, these are neonates whose suffering, recognizes the state of "intrapartum asphyxia" as a cause. Having considered the causes of neonatal distress due to respiratory syndrome, to malformations of various types, to sepsis, poor intrauterine growth, having considered the lesions of the CNS whose origin may also be in postnatal period, there is still a small group for whom the fetal distress persists after

Table 6. — *Neonatal pathology.*

Neonatal pathology	QUADR. 1		QUADR. 2		QUADR. 3		QUADR. 4	
	n.	%	n.	%	n.	%	n.	%
Asphyxia intrapartum	6	7.50	5	6.25	4	5.00	1	1.25
Sri Idiopathic apnea Rds	2	2.50	5	6.25	3	3.75	1	1.25
Respiratory insufficiency Lung haemorrhage								
Neurological damage	1	1.25	3	3.75	1	1.25		
SNC congenital diseases	2	2.50						
Congenital cardiopathies	1	1.25	1	1.25				
Sepsis	1	1.25			1	1.25	1	1.25
Deaths	2	2.50	6	7.50	13	16.25		
SGA	10	12.50	11	13.75	13	16.25	9	11.25
Chromosome diseases			2	2.50				
Fetal dystrophy	3	3.35	2	2.50	1	1.25		
Others			1	1.25	3	3.75	3	3.75

birth. The percentage of "intrapartum asphyxia" decreases progressively shifting from 7.5% to 1.25%.

DISCUSSION

The examination of the cases affected with fetal distress that we have considered for each quadriennium has led to some interesting remarks.

In the first 4-year period, the number of cases subjected to cardiotocography, was 26 out of 80, equal to 32.5%; the second quadriennium 76 out of 80 (95%), the remaining two quadriennia 80 out of 80 (100%). These results are due to the fact that CTG has become a routine evaluation during pregnancy and labour.

Though a decrease may be expected in the pathological alterations in comparison with the uncertain, this has not occurred over the years. Before examining all our material we might have expected that the obstetricians would tend towards lesser risks and earlier interventions in the presence of fetal distress. But the logic of safety offered by continuous FHR monitoring and the good results in terms of neonatal outcome must have prevailed.

We should like to discuss two clinical events that our study has identified: fetal distress "sine causa" and intrapartum asphyxia.

Fetal distress "sine causa". There are at least two possible interpretations of this phenomena; 1) has the problem of the CTG false positive to do with it? The predictive value of the CTG as regards the neonatal depression is actually quite low. As a matter of fact these interventions may have been useless, because the fetal well-being was not compromised. 2) It is theoretically possible that a low degree of reduction of the uterine placental flow, occurring in a period fairly near to term would not be able to cause an intrauterine growth reduction or arrest, yet causing a latent state of fetus-placental insufficiency that showed itself during labour. In others words, the fetus subjected to the decreased uteroplacental blood flow, starts physiological adjustments (blood flow redistribution) able to ensure the normal oxygenation of the brain during pregnancy, but not during labour, because of the further decrease in flow and of the placental perfusion caused by uterine contractions. A timely intervention preserving the fetus from a progressive hypoxia, may explain the high incidence of undepressed neonates, thus reducing the problem of the low positive predictive value of CTG. A similar problem presents itself to us when we consider the group of neonates with diagnosis of "intrapartum asphyxia". If we consider these neonates as the only true positives of the cardiotocographic evaluation, we might think that the number of the false positive, very high in the first quadriennium, has further increased in the fourth. We should come to the conclusion that the CTG would led only to useless interventions. On the contrary, we think that there must be another interpretation of this phenomena, based on the premise that the fetal distress should be distinguished from the neonatal one. When a fetus shows cardiotocographic symptoms of hypoxia, damage involving haemodynamic compensatory states have not necessarily occurred. It is the case of the sudden flow interruptions due to cord compression which may cause variable decelerations or bradycardia, depending on the duration and seriousness of the circulatory obstacle, even in a fetus in physiological conditions.

A timely intervention preserves the fetus from a hypoxical noxa and permits the birth of vigorous neonate, in the large majoriety of cases. On the contrary, when the hypoxic noxa (i.e.: uterine contraction) operates in a fetus in previous haemodynamic compensation, there is a higher vulnerability, or the CNS tissues and three different types of outcome may

occur, depending mainly on the persistence of the hypoxia state: 1) birth of vital neonate; 2) birth of a neonate with persistent symptoms of asphyxia, therefore needing reanimation; 3) neonate with neurological damage. The diagnosis of intrapartum asphyxia and the CNS lesions reported in Tab. 6 refer to the last two groups. The achievement of such high percentages of success as those shown in the fourth quadriennium (1.25% of asphyxia and 0% of CNS lesions) has probably required an extended use of the cesarean section even for cases of fetal distress compatible with vaginal delivery: an important consideration especially after birth.

We can make an attempt to summarize the question by quoting J. T. Parer's definition of fetal distress ([9]): "The fetal distress consists in a progressive asphyxia which if not corrected or stopped may lead to the decompensation of the physiological response (redistribution of flow running for preservation of the oxygenation of the vital area) and induce permanent cerebral damage or death". However we should bear in mind that cerebral damage cannot be due exclusively to intrapartum asphyxia; only 10% or less of the CNS permanent lesions affecting neonates are due to asphyxia, as results from studies on babies affected by cerebral palsy ([10]).

Our data agree in reporting a global improvement of neonatal prognosis in cases of fetal distress as regards neonatal outcome, whereas the specific problem of the neurological damage has not been discussed because it occurred marginally in the randomized samples. In our opinion this improvement is prevalently due to the introduction of continuous FHR monitoring during pregnancy and labour. Several esteemed researchers using CTG in their clinical practice consider it at present as the most suitable means of detection and evaluation of fetal distress ([11, 12, 13, 14]). The role of CTG is moreover to guide the therapeutical decision to interrupt the state of hypoxia in case the risk of irreversible damage should approach. We believe instead, that the FHR monitoring is not useful for the recognition of the moment preceding irreversible cerebral lesion. In spite of that, there are cardiotocographic events whose presence is prevalently due to irreversible hypoxic damage such as severe bradycardia persisting for even 15 mins., late decelerations with absence of variability, severe variable decelerations with ominous additional criteria according to Fischer ([8]). An intervention effected after the appearance of these signs may be useless for the prevention of neurological damage.

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