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An approach to interpretation and classification of sinusoidal fetal heart rate patterns

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Summary

Sinusoidal fetal heart rate (SHR) records were obtained in 8 cases, either antepartum (3 cases of fetal Rh disease) or intrapartum (one case with an acute episode of fetomaternal transfusion as possible cause, 2 after meperidine administration to the mother and 2 others without attributable causes). Characteristics of both SHR patterns and related clinical pictures are described and compared to similar cases published elsewhere. The possible underlying mechanisms of SHR are discussed. Two different profiles of SHR patterns (smooth and jagged waveforms) are characterized and correlated with their most usual clinical backgrounds and prognostic significance. A classification of SHR into 2 main types is proposed, with clinical use in mind.

Sinusoidal fetal heart rate; Rh sensitization; Fetomaternal transfusion; Meperidine

Introduction

Sinusoidal fetal heart rate (SHR) has been described in a variety of clinical situations, either antepartum [1–6] or intrapartum [7–10]. It is more frequently seen in association with severe fetal anemia in Rh-isoimmunized fetuses [1,2,5,11] but it also occurs in cases of fetal anemia caused by fetomaternal hemorrhage [3,12,13], after the administration to the mother of alphaprodine [7], meperidine [14,15] or other analgesics [14,16], in severe fetal hypoxia and acidosis [8,9], in association with amnionitis [10] and during episodes of fetal sucking movements [6]. Except in cases

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of analgesic administration to the laboring woman or when it occurs with fetal sucking, SHR is usually considered as implying an ominous fetal prognosis [1,2,17].

Since the original description [1] extended by the contribution of others [2,9], the following features have generally been accepted as characteristics of SHR: (a) stable baseline fetal heart rate (FHR) of 120 to 160 beats per minute (bpm) with regular sine-wave oscillations; (b) amplitude of 5 to 15 bpm, seldom greater; (c) frequency of 2 to 5 cycles per minute; (d) very reduced or absent short-term variability.

Even with strict application of these rules, authors do not always agree on the classification of some FHR tracings as SHR [17]. Difficulties in interpretation are caused by both the possible subjectivity in reading FHR records and the false short-term variability introduced by ultrasound methods of external FHR monitoring [5,14,19].

A clear distinction between 'good' and 'bad' fetal prognosis in cases where SHR is present, based *only* on the analyses of FHR tracings, has not been possible until now. However, other methods for biophysical and biochemical evaluation of the fetal state have been shown to be useful tools for establishing the fetal prognosis when SHR occurs.

In the present paper we report some cases of SHR, discuss their correlations with underlying clinical situations and try to establish differences among SHR waveforms and their prognostic meaning.

Patients and methods

Cardiotocographic records in which an SHR pattern was identified were obtained in 8 cases by means of fetal monitors Corometrics model 112 and Hewlett-Packard model 8040A at a paper speed of 1 cm per min. All the cases have been observed in our department between 1982 and 1986. In cases 6 and 7 FHR was recorded by direct electrocardiogram (ECG) using a spiral fetal scalp electrode; in all other cases an external ultrasound transducer was used.

Patients 1 to 3 were Rh-sensitized, cases 1 and 3 in previous pregnancies and case 2 by blood transfusions performed 7 years before because of idiopathic thrombocytopenic purpura; none of them received prophylactic anti-D immunoglobulin; in all of them SHR occurred antepartum.

In cases 1 and 2 SHR occurred in late third trimester; in both cases a cesarean section (C/S) was decided upon because of cardiotocographic and clinical data, namely amniotic fluid optical density difference at 450 nm (ΔOD_{450}). Neonatal exchange transfusions were needed (one in case 1 and two in case 2); the newborns were discharged in good health on the 8th and 11th days of life, respectively.

In case 3, at 31 weeks of pregnancy, short SHR episodes were identified over a non-reactive nonstress test (NST). The pattern recurred for brief periods during the oxytocin challenge test (OCT) that was immediately performed. Three contractions in 10 min were achieved at an oxytocin infusion rate of 2.8 mU/min; since no late decelerations were identified the OCT was read as negative. Several fetal movements followed by FHR accelerations occurred during the OCT. In the following day a C/S was decided upon after the observation of an almost continuous SHR record.

TABLE I

General clinical data

Patient	Para	Gesta	Blood group	Clinical data	Occurrence of SHR (weeks)	ΔOD_{450} (Liley's zone)	Mode of delivery	Newborn			Cord blood		
								Sex	Weight (g)	Apgar scores 1′–5′	Coombs (direct)	Hb (g/dl)	Bilir (mg/dl)
1	1	3	B−	Rh sensit.	$37\frac{1}{7}$	2a	C/S	M	2610	7–10	+	10.5	10.0
2	0	1	O−	Rh sensit.	$35\frac{4}{7}$	3	C/S	F	2480	4– 7	+	7.5	7.5
3	2	4	O−	Rh sensit.	$31\frac{2}{7}$	2b	C/S	F	1850	5– 8	+	3.2	12.0
4	0	2	O+	Normal pregnancy	41	–	C/S	F	3300	5–10	–	8.5	1.5
5	0	1	O+	Normal pregnancy	$38\frac{2}{7}$	–	Forceps	M	3600	5– 9			
6	0	1	O−	Normal pregnancy	42	–	C/S	M	3990	7– 9			
7	0	3	O+	Normal pregnancy	40	–	Spontan.	M	3380	9–10			
8	0	2	O+	Normal pregnancy	41	–	C/S	M	4180	9–10			

SHR, sinusoidal fetal heart rate; Rh sensit., maternal Rh sensitization; C/S, cesarean section; Hb, hemoglobin; Bilir, bilirubin; Spontan., spontaneous vaginal delivery.

The severely anemic newborn died 8 h after birth during an exchange transfusion. At autopsy enlarged liver and spleen, serosal effusions and intracranial hemorrhage were found.

Cases 4 to 8 were normal term pregnancies in spontaneous labor, SHR occurring intrapartum.

In case 4 the pattern was noted 12 h after admission, when the cervix was fully effaced and 5 cm dilated. Previous FHR monitoring, which was started in early labor, showed no abnormalities. No analgesics had been given to the mother. Because of SHR occurrence a C/S was performed, with the birth of a very pale baby. Cord blood hemoglobin was 8.5 g/dl, hematocrit 27% and 21 erythroblasts per 1000 leucocytes were seen. The newborn exhibited no signs of hemodynamic instability. A few hours later hemoglobin was 9.2 g/dl, hematocrit 32% and a packed red blood cell transfusion was performed. The baby was discharged on the 7th day in good health.

In case 5 routine external fetal monitoring was instituted shortly after admission in early labor. No abnormal patterns were detected. Three hours later, at the beginning of active labor, an intravenous drip of 100 mg of meperidine diluted in saline was started at a 1.5 mg/min rate. Short-term variability completely disappeared 30 min later and an SHR pattern ensued, lasting for about 1 h. Twenty hours after admission, under general anesthesia, a midforceps was performed for lack of descent of the fetal head. The newborn had an Apgar score of 9 at 5 min; umbilical artery pH was 7.20. The baby was discharged 6 days later doing well.

Patient 6 was admitted at 3 cm dilatation with mild contractions occurring at 5 min intervals. External fetal monitoring was started, displaying an FHR at 120 bpm with accelerations and normal variability. At 4 cm dilatation membranes were ruptured and a fetal scalp electrode was inserted. Two hours later, for maternal analgesia, an intravenous infusion of 100 mg of meperidine diluted in saline was instituted at a 2.0 mg/min rate. Twelve minutes after the beginning of meperidine drip an SHR was observed, lasting for 45 min and being followed by an FHR similar to that seen before the medication. For secondary arrest of dilatation a C/S was performed 12 h after admission. The newborn's Apgar score at 5 min was 9 and umbilical artery pH was 7.23. There were no problems during the neonatal period.

In case 7 routine FHR monitoring was started by direct fetal ECG at a cervical dilatation of 2 cm. During the insertion of the fetal scalp electrode light meconial amniotic fluid was seen. From the start of FHR monitoring an SHR pattern was identified. For maternal pain relief 100 mg of meperidine were given by the intramuscular route; about 8 min after the injection the SHR ceased and a normal FHR tracing ensued. A vigorous baby was spontaneously delivered 3.5 h after the beginning of fetal monitoring; umbilical artery pH was 7.25. The newborn was discharged 3 days later.

In case 8 a jagged SHR was obtained in early labor during a routine FHR monitoring. After 25 min the SHR pattern vanished and a normal FHR ensued. Ten hours after admission a C/S was performed for cephalopelvic disproportion. An active, non-anemic, fetus was delivered; umbilical artery pH was 7.23. The baby was discharged in good health on the 6th day of life.

General relevant clinical data of the cases are summarized in Table I.

Results

A summary of the details of SHR records obtained in our 8 patients is given in Table II; patient 3 had 2 tracings (identified as 3A and 3B) recorded in consecutive days.

According to the duration and recurrence of SHR areas in each tracing, records were divided into three categories: (a) *continuous,* when persisting for a minimum of 30 min; (b) *intermittent*, if 2 or more episodes lasting for not less than 3 min are seen in a 30-min period; (c) *transitory,* if one single episode lasting for more than 10 min and for less than 30 min is identified.

Intermittent SHR have only been observed in Rh disease. Continuous tracings appeared in anemic fetuses (patients 2, 3 and 4) and in the meperidine-related cases (patients 5 and 6). In case 3 intermittent and continuous records were seen 24 h apart. Cases 7 and 8, in which an objective cause of SHR was not found, each showed a single transitory area of the pattern.

Maximal oscillatory amplitudes of 15 bpm or more were only seen in cases of fetal anemia. Recurrent late decelerations appeared only in record 3B; in this case, the FHR record obtained 24 h before birth showed only brief episodes of SHR, a negative OCT and several FHR accelerations.

The comparative study of our 9 records showed a clear difference in SHR waveforms: in all the cases of fetal anemia (patients 1 to 4) SHR oscillations are of a rounded, smooth, symmetric shape (Fig. 1); in cases 5 to 8, not related to fetal

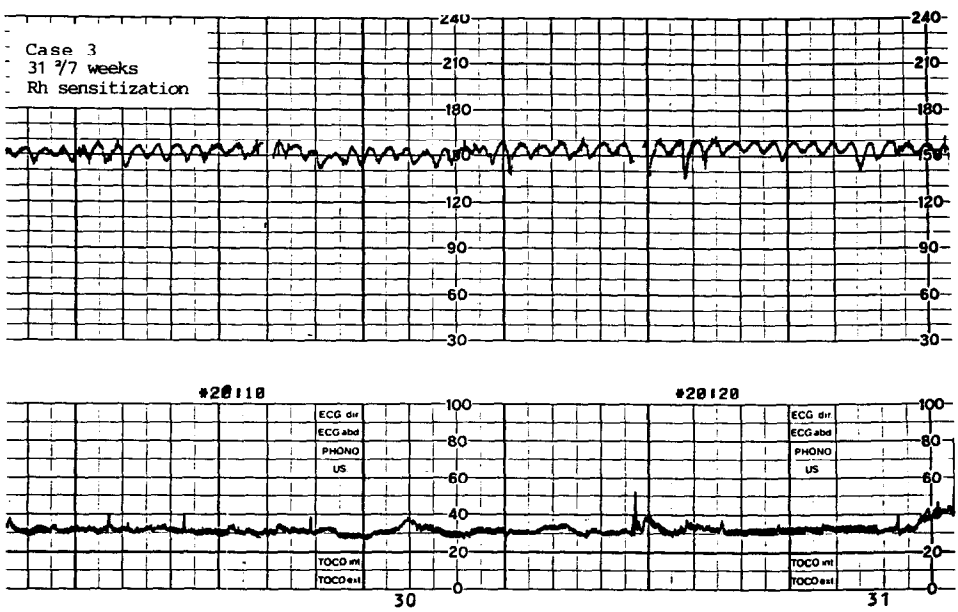

Fig. 1. SHR pattern obtained in a case of Rh disease (patient 3, record 3B). Note the smoothness of oscillations. Paper speed: 1 cm/min.

TABLE II

Characteristics of SHR records

Patient	Baseline FHR (bpm)	Occurrence [a]	Duration of episodes (min)	Amplitude (bpm)		Frequency (cycles/min)	Waveform	Associated recurrent patterns
				Main	Maximal			
1	130	Intermittent	6–20	5–10	25	3–5	Smooth	No
2	150	Continuous	> 30	5–10	10	2–4	Smooth	No
3A	150	Intermittent	3–6	5–10	20	2–4	Smooth	Accelerations
3B	150	Continuous	> 30	5–10	15	2–4	Smooth	Late decelerations
4	160	Continuous	> 30	5–10	15	2–4	Smooth	No
5	130	Continuous	> 30	5	10	4–5	Sawtooth	No
6	120	Continuous	> 30	5	10	4–5	Sawtooth	No
7	165–145	Transitory	16	5–10	10	4–5	Sawtooth	No
8	145	Transitory	25	5	10	3–5	Sawtooth	No

[a] See text for definitions of types of occurrence.

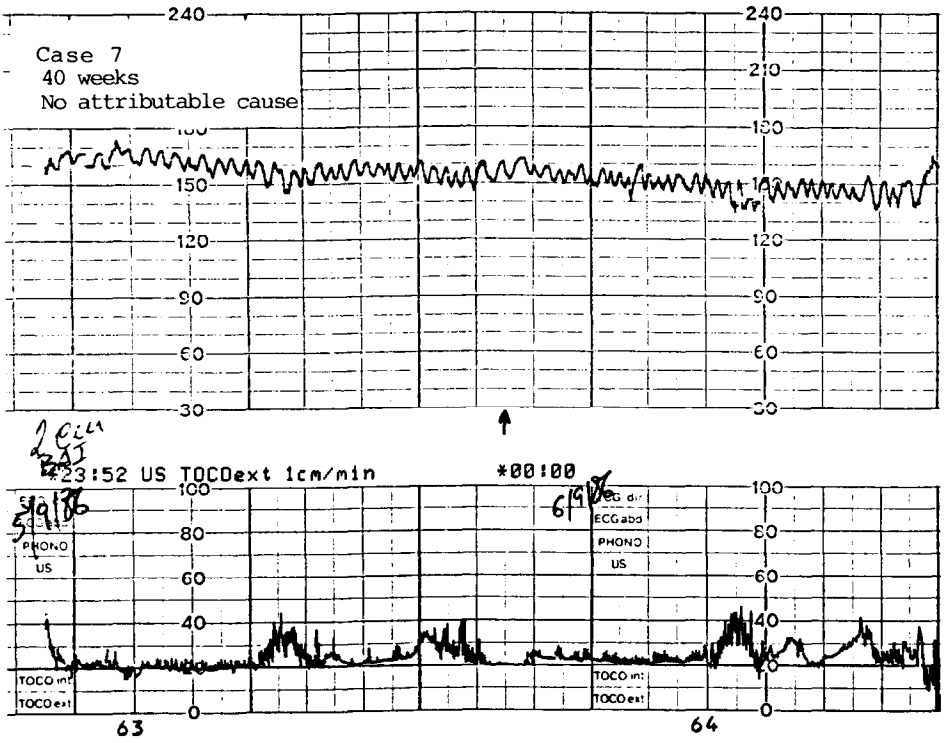

Fig. 2. SHR pattern obtained in case 7 (no attributable cause). Note the jagged waveform. Paper speed: 1 cm/min.

anemia, SHR undulations display a jagged, sawtooth form (Fig. 2). Smooth SHR occurred either antepartum or intrapartum; despite the good Apgar scores at 5 min all the newborns had to receive intensive care and one of them died. The sawtooth pattern appeared exclusively intrapartum; all babies were vigorous at birth and there was no need for neonatal intensive care.

Discussion

Sinusoidal fetal heart rate was first described in 9 antepartum high-risk patients; severe fetomaternal Rh incompatibility was present in 8 of them [1]. Although SHR may occur in several clinical entities, it seems to carry the worst prognostic significance in situations of fetal anemia caused by either Rh disease or fetomaternal hemorrhage.

Our first 3 cases are Rh-sensitized patients with moderate to severe fetal anemia; they confirm the ominous significance ascribed to SHR, since all newborns had umbilical cord blood hemoglobin values below 11.0 g/dl. This set of cases may contribute to reinforcing the assumption that, in Rh disease, FHR monitoring identifies fetuses at most risk [5]. Also, it was considered that spontaneous late decelerations appeared to have a diagnostic importance equal to SHR in Rh disease

[5]. Therefore, it is possible that the appearance of SHR and/or late decelerations in Rh-isoimmunized patients might be better indicators for termination of pregnancy than amniotic fluid ΔOD_{450} alone.

It is important to point out that in case 3, in which a neonatal death occurred, short episodes of SHR were identified being followed by a negative OCT with simultaneous clear FHR accelerations, 24 h before delivery. This observation does not support the opinion of Modanlou and Freeman, who stated that areas of normal variability and reactivity must be absent to confer on SHR an ominous significance [17]. Young postulated that SHR should be observed for at least 10 min before such a classification is made [18], but in the case under discussion no more than intermittent episodes of 3–6 min duration were seen the day preceding a continuous SHR pattern and the birth of a very deteriorated fetus. Based on those observations one is tempted to believe, in accordance with Visser [5], that the appearance of SHR in Rh disease must always be looked upon as a sign of fetal deterioration even when seen for periods shorter than 10 min.

In case 4 SHR occurred concomitantly with severe infant anemia not related to Rh disease. As reported before [3,13], the underlying cause might be fetomaternal hemorrhage, a situation probably more frequent than is clinically suspected. The findings in the newborn may be related to the size of the transfusion and the time at which it occurs [19]. A clinical picture similar to that seen in our case was reported by Modanlou et al. [3], who suggested that fetal anemia at birth, without hemodynamic instability of the newborn, can be interpreted as being caused by a recent acute hemorrhage superimposed on a chronic fetomaternal transfusion too intense for complete hematopoietic compensation; the presence of a large number of erythroblasts in cord blood might be related to a partial compensation for chronic fetal blood losses. A Kleihauer-Betke test would be advisable for definitive confirmation of a sudden episode of fetomaternal transfusion, but it was not performed in our case.

In cases 5 to 8 all the newborns were vigorous at birth and did well during the neonatal period; signs of infant anemia were not seen. Only cases 5 and 6 may be related to meperidine administration, a feature already described [14,15]. The FHR sawtooth waveform observed in our cases is similar to those described in association with alphaprodine [7,14]. In patients 7 and 8 SHR occurred with no attributable causes. In case 7 mean FHR declined from 165 to 145 bpm, a feature not usually seen in SHR records, although reported before [9].

The pathophysiology of SHR remains unclear. Hypoxia of the cardiac center with derangement of central nervous system control of the heart rate has been postulated as the final common pathway in the production of such patterns [18]. Some advances have been made recently in this field using animal models. In fact, the association of fetal anemia and SHR was confirmed with the use of fetal lambs; a significant elevation of arginine vasopressin and a drop in fetal arterial blood pH coincident with the appearance of SHR in the bled lamb fetus were also found [20]. The same inverse correlation between fetal arterial pH and umbilical cord blood arginine vasopressin level and a direct association between the drop in fetal blood pH and the increase of SHR amplitude have previously been reported by others in humans [18,21]. Therefore, the rise of the arginine vasopressin level may be

responsible, either directly or indirectly, for producing and/or enhancing SHR in anemic and/or acidotic fetuses. It was also shown that absent or decreased vagal efferent control of the fetal heart is needed for the initiation of SHR induced by arginine vasopressin [20]. Despite the possible role of other mechanisms in this process (e.g. calcium) it seems reasonable to assume that fetal anemia and acidosis seen in the fetuses severely affected by Rh disease may intervene in vagal efferent control by the brain, thus releasing arginine vasopressin action on the fetal heart.

Other mechanisms must be involved in producing SHR patterns besides fetal anemia and acidosis. Depression of the fetal central nervous system caused by meperidine and other analgesics, together with other factors of fetal compromise, has been suggested [15]. However, the majority of the fetuses presenting SHR induced by analgesics are in good condition at birth, as was found in our cases 5 and 6 and as previously described by others [7,14]. A transitory decrease in autonomic control of the fetal heart, not related to a real fetal compromise, might be the possible cause. On the other hand, case 7 shows a complete reversal of SHR following meperidine administration, thus making the understanding of the underlying mechanism even more difficult; however, this association might be only coincidental. Thus, SHR in cases 7 and 8 may be considered spontaneous, transitory and unassociated with any recognizable obstetric event, as described before [14].

Apparently, the sum of knowledge is too slight to answer all the questions concerning SHR. A pertinent doubt lies in the classification of certain FHR tracings as true SHR patterns, namely the jagged oscillatory records induced by analgesics. If one assumes that all FHR tracings with the characteristics listed before must be classified as SHR, we think that it is important to define two different categories of the pattern, bearing in mind clinical use. In fact, certain features appear to distinguish those SHR records associated with poor fetal and neontatal outcomes from those with normal infant outcome. The survey of the published data and the contribution of our own cases constitute the support for the following proposal for SHR classification, according to the underlying conditions and the shape of the oscillations:

(a) *SHR type I*—*Waveform*: smooth, symmetric oscillations;
Occurrence: either antepartum or intrapartum;
Main causes: fetal anemia; fetal hypoxia;
Prognosis: ominous;
Associated FHR patterns in severe situations: oscillatory amplitude $\geqslant 15$ bpm; recurrent late decelerations.

(b) *SHR type II*—*Waveform*: jagged, sawtooth oscillations;
Occurrence: intrapartum;
Main causes: analgesics; spontaneous, self-limited;
Prognosis: good;
Associated FHR patterns: none.

In fetal evaluation, cardiotocography is just one variable amongst many others, some of these invasive and risky. We believe that the application of the proposed

classification to clinical management will help the recognition of real ominous SHR patterns, thus avoiding emergency obstetric measures or invasive diagnostic procedures (e.g. cordocentesis) in situations not recognized as type I SHR. On the other hand, in all the cases identified as type I SHR a complete fetal biophysical and/or biochemical evaluation must be performed before any definitive clinical decision. Nevertheless, further clinical experience will be needed for validation of the proposed classification.

References

1 Manseau P, Vaquier J, Chevinié J. Le rythme cardiaque foetal "sinusoidal". J. Gynecol Obstet Biol Reprod (Paris) 1972;1:343–352.
2 Rochard F, Schifrin BS, Goupil F, et al. Nonstressed fetal heart rate monitoring in the antepartum period. Am J Obstet Gynecol 1976;126:699–706.
3 Modanlou HD, Freeman RK, Ortiz O, et al. Sinusoidal fetal heart rate pattern and severe fetal anemia. Obstet Gynecol 1977;49:537–541.
4 Visser GHA, Redman CWG, Huisjes HJ, et al. Nonstressed antepartum heart rate monitoring: implications of decelerations after spontaneous contractions. Am J Obstet Gynecol 1980;138:429–435.
5 Visser GHA. Antepartum sinusoidal and decelerative heart rate patterns in Rh disease. Am J Obstet Gynecol 1982;143:538–544.
6 Nijhuis JG, Staisch KJ, Martin Jr CB, et al. A sinusoidal-like fetal heart rate pattern in association with fetal sucking—report of 2 cases. Eur J Obstet Gynecol Reprod Biol 1984;16:353–358.
7 Gray JH, Cudmore DW, Luther ER, et al. Sinusoidal fetal heart rate pattern associated with alphaprodine administration. Obstet Gynecol 1978;52:678–681.
8 Gal D, Jacobson LM, Ser H, et al. Sinusoidal pattern: An alarming sign of fetal distress. Am J Obstet Gynecol 1978;132:903–905.
9 Baskett TF, Koh KS. Sinusoidal fetal heart pattern. A sign of fetal hypoxia. Obstet Gynecol 1974;44:379–382.
10 Gleicher N, Runowicz CD, Brown BL. Sinusoidal fetal heart rate pattern in association with amnionitis. Obstet Gynecol 1980;56:109–111.
11 Elliot JP, Modanlou HD, O'Keefe DF, et al. Significance of fetal and neonatal sinusoidal heart rate pattern: further clinical observations in Rh incompatibility. Am J Obstet Gynecol 1980;138:227–230.
12 Clark SL, Miller FC. Sinusoidal fetal heart rate pattern associated with massive fetomaternal transfusion. Am J Obstet Gynecol 1984;149:97–99.
13 Stiller AG, Skafish PR: Placental chorioangioma: a rare cause of fetomaternal transfusion with maternal hemolysis and fetal distress. Obstet Gynecol 1986;67:296–298.
14 Johnson TRB, Compton AA, Rotmensch J, et al. Significance of the sinusoidal fetal heart rate pattern. Am J Obstet Gynecol 1981;139:446–453.
15 Epstein H, Waxman A, Gleicher N, et al. Meperidine-induced sinusoidal fetal heart rate pattern and reversal with Naloxone. Obstet Gynecol 1982;59:22S—25S.
16 Feinstein SJ, Lodeiro JG, Vintzileos AM, et al. Sinusoidal fetal heart rate pattern after administration of nalbuphine hydrochloride; a case report. Am J Obstet Gynecol 1986;154:159–160.
17 Modanlou HD, Freeman RK. Sinusoidal fetal heart rate pattern: its definition and clinical significance. Am J Obstet Gynecol 1982;142:1033–1038.
18 Young BK, Katz M, Wilson SJ. Sinusoidal fetal heart rate. I. Clinical significance. Am J Obstet Gynecol 1980;136:587–593.
19 Pai MRK, Bedritis I, Zipursky A. Massive transplacental hemorrhage: clinical manifestations in the newborn. M C A J 1975;112:585–589.
20 Murata Y, Miyake Y, Yamamoto T, et al. Experimentally produced sinusoidal fetal heart rate pattern in the chronically instrumented fetal lamb. Am J Obstet Gynecol 1985: 153:693–702.
21 Parboosingh J, Lederis K, Singh N. Vasopressin concentration in cord blood: correlation with method of delivery and cord pH. Obstet Gynecol 1982;60:179–183.