# Sinusoidal fetal heart rate pattern

**Shinji Katsuragi, Julian T. Parer, Tomoaki Ikeda**

*Abstract*— **We describe 2 cases of term anemic fetuses with different sinusoidal pattern morphology, and possible mechanisms.**

*Cases*—**The first patient noted sudden cessation of fetal movement on the day of presentation. She had a sinusoidal FHR pattern. The newborn had a hemoglobin of 3.7 g/dl, and umbilical artery pH was 7.10 and BE -7 mEq/l.**

**The second patient noted decreased fetal movement for several days. She had a FHR pattern with absent FHR variability, and intermittent sinusoidal elements, with late decelerations. The newborn's hemoglobin was 1.5 g/dl, umbilical artery pH was 7.07 and BE -10.2 mEq/l. Both cases had positive Kleihauer-Betke tests.**

*Index Terms*—**Sinusoidal, Fetal Heart rate, FHR, HB,**

## I. INTRODUCTION

Sinusoidal fetal heart rate patterns have most commonly been associated with anemic fetuses. We describe 2 cases with different sinusoidal pattern morphology, and possible mechanisms.

### Cases

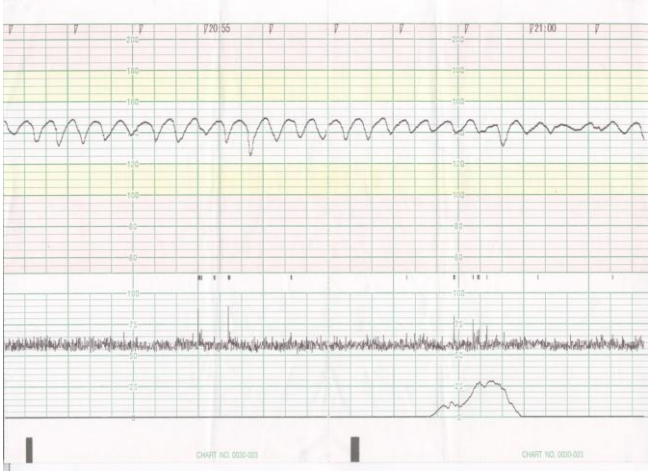
Case 1: The patient was a 27 yo G1 P0 at 37 $^{5}/_{7}$ weeks, with a classical, persistent sinusoidal pattern, with a sinusoidal frequency of about 3/min cycles, and baseline FHR within the normal range (Figure 1). She related sudden cessation of fetal movement that day, and underwent Cesarean delivery. The newborn weighed 2976g, Apgar scores was 2 at 1 min, 5 at 5 min, umbilical artery pH 7.10 and BE -7 mEq/l. The hemoglobin and hematocrit were 3.7 g/dl and 12.9%. The Kleihauer-Betke test showed 5% fetal cells in maternal blood. The baby did well after total transfusion of 30 ml/kg of red cells.

**Shinji Katsuragi**, Department of Obstetrics and Gynecology, Sakakibara Heart Institute, Fuchu, Japan, M.D. 3-16-1, Asahi-Cho, Fuchu, Tokyo, Japan, Tel: +81-42-314-3111, Fax: +81-42-314-3133

**Julian T. Parer**, Department of Obstetrics, Gynecology and Reproductive Sciences, University of California, San Francisco, USA

**Tomoaki Ikeda**, Department of Obstetrics and Gynecology, Mie University, Tsu, Japan

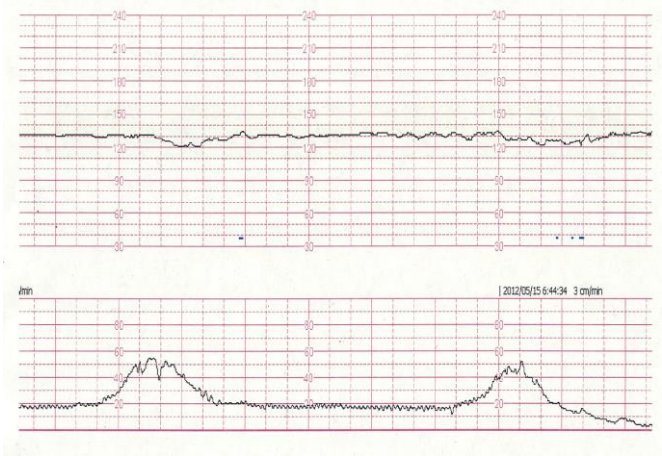
Figure 1



Fetal heart rate tracing of case 1, showing a classical, persistent sinusoidal pattern, with a frequency of about 3/min cycles, and normal baseline rate.

Case 2: The patient was a 28 yo G1 P0 at 41$^{0}/_{7}$ weeks, with normal baseline FHR, but without variability and accelerations, mostly flat but with some blunted sinusoidal complexes, and late decelerations with intermittent uterine contractions (Figure 2). She related decreased fetal movement for "several days" before she sought care. At Cesarean delivery the newborn weighed 3316g, had Apgar scores of 1 at 1min and 5 at 5 min and umbilical artery pH of 7.07 and BE -10.2 mEq/l. The hemoglobin was 1.5g/dl. The Kleihauer-Betke test showed 3.5% fetal cells in maternal blood. The baby developed pulmonary hypertension, hypoxemia and required prolonged intubation and blood pressure support. The AVP levels in case 1 was higher than case 2.

Figure 2



Fetal heart rate monitoring of case 2, showing a normal baseline FHR without FHR variability and accelerations, mostly flat but with some blunted sinusoidal complexes, and late decelerations with intermittent uterine contractions.

## II. Discussion

We propose that the morphology of the sinusoidal pattern depends on the acuteness of onset of the anemia. Murata and coworkers[1)] have implicated arginine vasopressin (AVP) in the sinusoidal pattern. Sinusoidal FHR patterns and increased AVP blood levels were produced in fetal lambs by hemorrhage, and AVP infusion into vagotomized or atropinized fetuses. These authors proposed that the direct effect of AVP on the sinus node may have affected calcium transfer, resulting in the pattern.

The less striking blunted pattern seen in the second case may reflect decreased AVP levels as the fetus adapts to its anemic state over several days. This is similar to the observation that fetuses anemic due to Rh alloimmunization frequently have the second type of pattern due to the slow development of anemia[2)]. This theory is supported by higher AVP levels in case 1 than in case 2.

## III. Conclusion

We propose that the arginine vasopressin levels in the first case are high due to the acuteness of feto-maternal bleeding, and that they are lower in the second case as the fetus adapted to its anemic state.

## References

[1] Murata Y, Miyake Y, Yamamoto T, et al. Experimentally produced sinusoidal fetal heart rate patterns in the chronically instrumented fetal lamb. Am J obstet Gynecol 1985; 153: 693-702.

[2] Milio LA, Arnold SA, Parer JT. The relationship between fetal heart rate variability and hematocrit in Rh isoimmunization. Abstracts of Scientific Papers, 8th Annual Meeting, Society for Perinatal Obstetricians, Las Vegas, 1988.