Functional asymmetry of the human myometrium documented by color and pulsed-wave Doppler ultrasonographic evaluation of uterine arcuate arteries during Braxton Hicks contractions

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OBJECTIVE: We hypothesized that arcuate arteries supplying placental and nonplacental myometrial portions would respond with different degrees of change in their resistance during Braxton Hicks contractions.

STUDY DESIGN: We examined 20 healthy pregnant women between 18 and 24 weeks' gestation with pulsed-wave and color-flow Doppler during focal Braxton Hicks contractions identified by real-time ultrasonography by means of the characteristic thickening of the myometrium. Systolic/diastolic ratio was used as an expression of resistance. Statistical analysis was performed by Wilcoxon signed-ranks and Mann-Whitney test.

RESULTS: When the contractions are localized in the subplacental myometrium, the resistance of the arcuate artery did not differ during and after the contraction. In contrast, when the contraction involved only nonplacental myometrium, the resistance during the contraction was significantly higher and in some patients there was complete absence of flow during the diastolic phase. During subplacental myometrial contractions, the main uterine artery resistance was not affected. When the contraction involved the nonplacental myometrium, the resistance of the main uterine artery increased with more pronounced changes when the contraction involved the lateral myometrial wall ipsilateral to the uterine artery under examination.

CONCLUSION: We speculate that the differences in the degree of resistance change are the result of different degrees of contractility exhibited by the subplacental and nonplacental myometrium. We conclude that the intact human myometrium manifests functional asymmetry and our Doppler findings confirm previous in vitro studies. (Am J Obstet Gynecol 1993;168:184-8.)

Key words: Functional asymmetry of myometrium, Doppler, uterine artery

A progressive decline in the uterine artery resistance[1] and an increase in cardiovascular performance[2] during pregnancy help to meet the increasing demands of the developing conceptus. In normal human pregnancy several investigators have documented by Doppler flow velocity studies that the resistance in the uterine artery declines progressively to the lowest point by 24 to 26 weeks' gestation.[3-6] Continuous Doppler ultrasonographic studies of the uterine artery have verified an increase in the uterine artery resistance during uterine contractions in patients who are in active labor.[7, 9] In addition, Bower et al.[10] used color Doppler imaging to show that even Braxton Hicks contractions may affect uterine blood supply by increasing the uterine artery resistance. All of the above investigators have examined the main uterine artery that reflects total uterine vascular resistance, including the placental and the nonplacental myometrial areas.

Csapo et al.[11] have reported on the functional asymmetry of the human myometrium by studying myometrial strips derived from various uterine sites with knowledge of the placental location. They found that the closer to the placenta the higher the progesterone content and the lesser the myometrial contractility. We hypothesized that arcuate arteries supplying placental and nonplacental myometrial portions would respond with different degrees of change in their resistance during Braxton Hicks contractions. We designed this study to evaluate this hypothesis and to examine if functional asymmetry is present in the intact human myometrium.

Material and methods

We studied 20 pregnant women between 18 and 24 weeks of gestation with color-flow imaging and pulsed-wave Doppler ultrasonography (Toshiba 270-30 A

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sonolayer, Toshiba America, Yonkers, N.Y.). The patients were recruited from a group of patients referred to our unit for prenatal diagnosis or routine growth evaluation. All studies were performed before any invasive diagnostic procedures. All fetuses were normal with appropriate growth. The mothers were normotensive, and they all had uncomplicated pregnancies at the time of the examination. Only patients with normal uterine and umbilical artery resistance were recruited. We identified uterine contractions ultrasonographically by the characteristic thickening of the myometrium. Some of the patients had contractions in the nonplacental and some in the placental myometrium, and in some patients the contraction started in one area and in the course of the study migrated to the other area (placental to nonplacental or vice versa).

We identified the intramyometrial arcuate arteries with color-flow imaging and obtained the flow velocity waveforms by placing the pulsed Doppler range gate at the vessel. We obtained waveforms from the main uterine artery by tracing the uterine artery until it crossed over the external iliac vessels and placed the range gate at the portion of the uterine artery next to the iliac artery. This technique provides reproducible results (unpublished data). We analyzed four to five flow velocity waveforms from each vessel. Resistance was evaluated by the systolic/diastolic ratio. When diastolic flow velocities were not present, the systolic/diastolic ratio was considered to be 10 for the purpose of statistical analysis. Studies were performed during the contraction and immediately after. Half the patients developed placental and the other half nonplacental myometrial contractions. In five patients the contraction originated in one of the two myometrial segments and subsequently migrated to the opposite, giving us the opportunity to examine the different responses of the two segments in the same patients.

Statistical analysis of the paired measurements was performed by Wilcoxon signed-ranks test.[6] Comparison of the subplacental and nonplacental arcuate artery flow velocity waveforms was accomplished by Mann-Whitney test. A $p < 0.05$ was considered statistically significant.

Results

Color Doppler imaging demonstrates the significantly different flow patterns during contraction in the nonplacental and placental myometrium (Fig. 1). In the absence of contractions arcuate arteries located in the subplacental myometrium have lower resistance than the arteries in the nonplacental myometrium (1.67 ± 0.13 vs 2.28 ± 0.21, $p < 0.001$). All patients who developed contractions in the myometrial area under the placenta exhibited normal arcuate artery resistance during and after the contraction (1.68 ± 0.13 vs 1.67 ± 0.13, $p = 0.85$, Table I). All patients with contractions that developed in the nonplacental myometrium had normal arcuate artery resistance without contractions, but during contractions the resistance increased significantly (1.98 ± 0.18 vs 6.84 ± 3.13, $p < 0.003$, Table I). In this group half the patients exhibited absence of end-diastolic flow velocities. In the five patients with sequential placental and nonplacental contractions the placental arcuate artery resistance did not change during the contraction, but the nonplacental arcuate artery resistance changed significantly with only minimal or absent diastolic flow velocities present.

The resistance of the main uterine artery did not change during and after contractions involving the subplacental myometrium (2.31 ± 0.24 vs 2.27 ± 0.22, $p = 0.57$, Table II). During contraction of the nonplacental myometrium, however, there was a variable response depending on the location of the contraction in relation to the uterine artery. In two patients with the contraction taking place within the lateral myometrium there were significant changes in the resistance of the ipsilateral main uterine artery evidenced by higher systolic/diastolic ratio and the development of a dicrotic notch on a previously normal waveform (Fig. 2). Overall, in the presence of a nonplacental contraction the main uterine artery closest to the contraction exhibited higher resistance during the contraction than after (3.27 ± 0.37 vs 2.23 ± 0.28, $p < 0.0001$, Table II).

Comment

This study shows significant differences in the response of the arcuate artery resistance during Braxton Hicks contractions. These differences depend on the location of the arcuate artery in relation to the location of the placenta. The uterine artery branches located within the myometrium are subject to external mechanical constriction from the surrounding interlaced myometrial fibers.[7] This constriction may lead to narrowing of the vascular lumen and so increase the impedance to blood flow. Our data suggest that, although the subplacental myometrium maintains some degree of contractility, the intensity of this contractility is lesser than the one exhibited by the nonplacental myometrium.

Csapo et al.[11] reported on the variable degrees of contractility of human myometrium in vitro. The further away from the placental location the more intense the myometrial contractility. This response relates to decreasing progesterone concentrations. Our findings are in agreement with the above and suggest that the intact human myometrium also exhibits functional asymmetry, as evidenced by the contraction's variable effect on the uterine artery resistance.

Although our findings suggest that the resistance of the subplacental arcuate arteries does not change during a contraction, they by no means suggest that the subplacental myometrial perfusion does not decrease during contractions. To the contrary, it is logical to assume that the subplacental myometrial perfusion de-

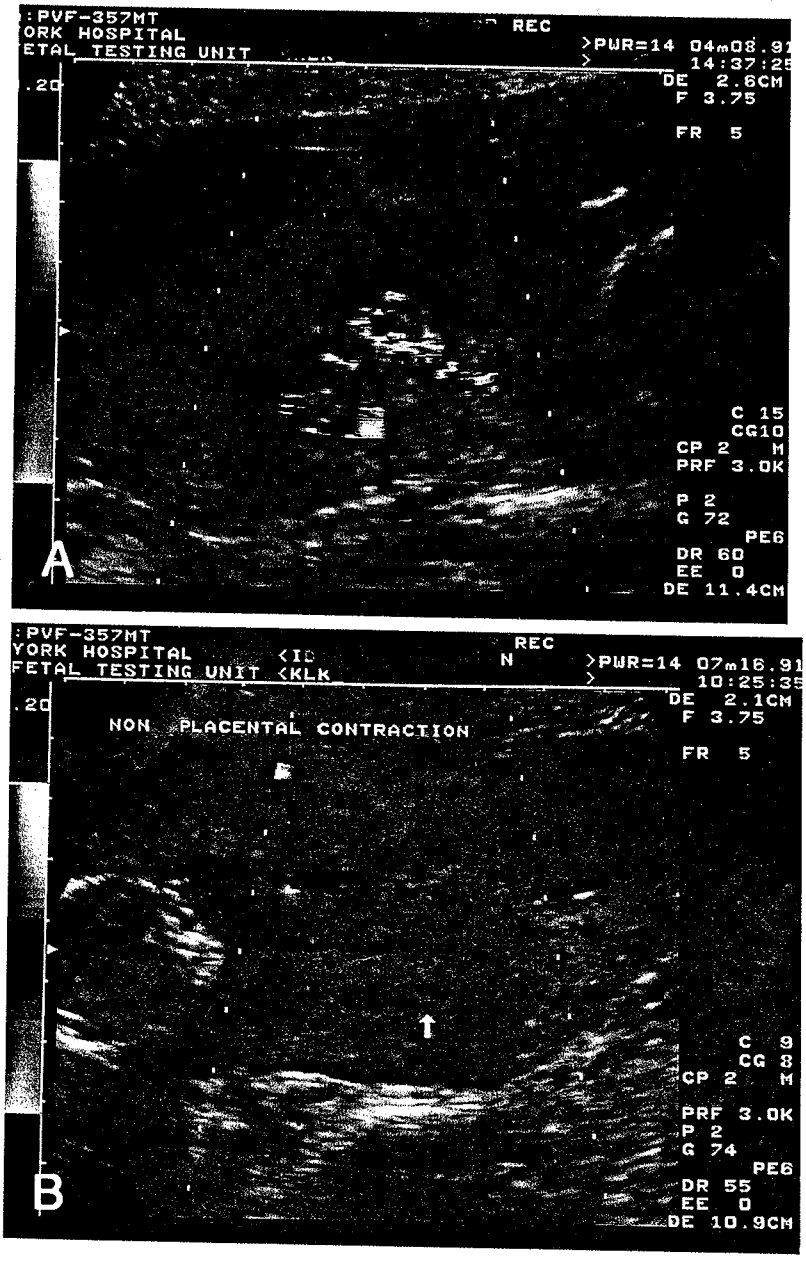

Fig. 1. A, Subplacental myometrium maintains abundant flow during contraction. **B,** Nonplacental myometrium demonstrates only scant flow during contraction.

Table I. Comparison of arcuate uterine artery resistance (systolic/diastolic ratio) during and after contraction in patients with subplacental and nonplacental myometrial contractions

Location of contraction	*No.*	*Arcuate artery*		*Significance*
		Contraction	*No contraction*	
Subplacental	10	1.68 ± 0.13	1.67 ± 0.13	$p = 0.85$
Nonplacental*	10	6.84 ± 3.13	1.98 ± 0.18	$p < 0.003$

*Five of the 10 patients had absent diastolic flow velocities.

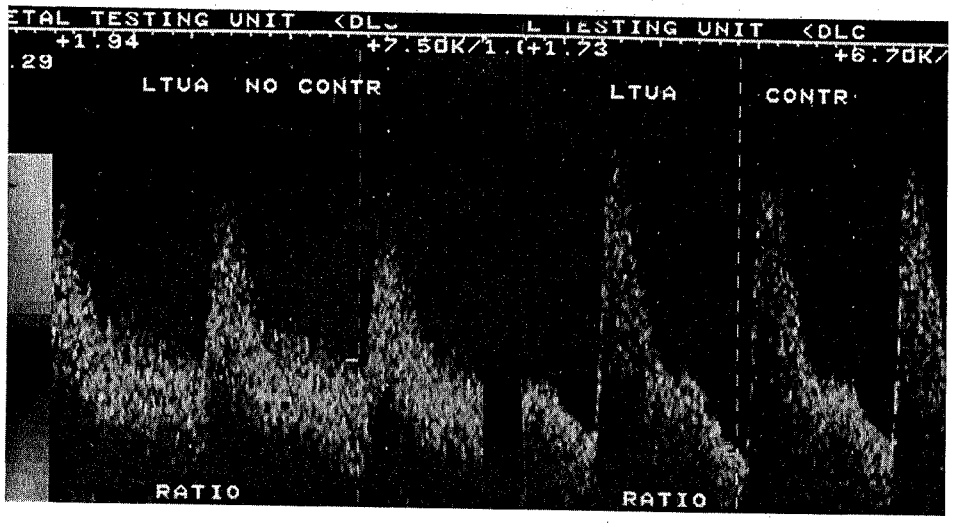

Fig. 2. Notice significant change in main uterine artery flow velocity waveform during contraction *(CONTR)* involving the ipsilateral nonplacental myometrium. *LTUA,* Left uterine artery.

Table II. Comparison of main uterine artery reistance (systolic/diastolic ratio) during and after contraction in patients with subplacental and nonplacental myometrial contractions

Location of contraction	*No.*	*Main uterine artery*		*Significance*
		Contraction	*No contraction*	
Subplacental	10	2.31 ± 0.24	2.27 ± 0.22	$p = 0.57$
Nonplacental	10	3.27 ± 0.37	2.23 ± 0.24	$p < 0.001$

creases to the extent that the subplacental spiral arterioles represent the terminal branches of arcuate arteries that originate within the nonplacental myometrium and thus are subject to constriction. Although this constriction originates within the nonplacental myometrium, it will exert a decreasing effect on the distally (subplacental) located arterial branches.

The fact that all of our subjects exhibited only focal and clinically weak contractions increases the validity of our findings. It is reasonable to assume that if a weak focal contraction can exert such an increase in the arcuate artery resistance, then a generalized and more powerful contraction should exert a more significant increase in the regional and overall myometrial vascular resistance.

In conclusion, our findings suggest that the intact human myometrium manifests functional asymmetry as evidenced by altered arcuate artery resistance, confirming previous in vitro studies. In addition, this study suggests that even mild focal myometrial contractions increase the resistance of the nonplacental uterine vessels but have no effect on the resistance of the subplacental uterine vessels. The overall impact of the above change in the resistance most likely exerts a decreasing effect on the myometrial blood supply.

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Effect of fetal movement on the amniotic fluid index

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OBJECTIVES: Fetal movement has been shown to change the size and location of amniotic fluid pockets during measurement of the amniotic fluid index. The effect of redistributing the fixed intrauterine fluid volume on the amniotic fluid index is unknown. Therefore we tested the hypothesis that the amniotic fluid index was unaffected by fetal movement.
STUDY DESIGN: A single examiner prospectively determined the amniotic fluid index before and after three discrete episodes of fetal movement during 96 biophysical profiles. A reliable blinded examiner provided a second postmovement measurement as a control. Data were analyzed by the paired *t* test.
RESULTS: The mean change in the amniotic fluid index after fetal movement was 1.5 ± 0.1 cm and 2.5 ± 0.2 cm for postmovement determinations by the same examiner and blinded observer, respectively ($p < 0.001$).
CONCLUSION: Interobserver and intraobserver variation can account for the change in the amniotic fluid index after fetal movement. (Am J Obstet Gynecol 1993;168:188-9.)

Key words: Ultrasonography, antepartum fetal assessment, amniotic fluid index

The amniotic fluid index provides a four-quadrant ultrasonographic assessment of amniotic fluid volume.[1] This measurement is superior to other methods of identifying abnormal amniotic fluid volumes[2] and inversely correlates with unfavorable perinatal outcome.[3] In spite of the technique's proved reliability, potential contributors to error such as fetal movement have been suggested.[4] We have observed the apparent creation and obliteration of fluid pockets caused by fetal movement during biophysical profile testing. It is uncertain how shifts of a stable intrauterine fluid volume affect the amniotic fluid index in a given patient. Therefore we designed a prospective before-and-after single-blind controlled study to test the hypothesis that fetal movement does not change the amniotic fluid index.

Material and methods

The study population consisted of 96 patients ranging from 26⅙ to 41⅕ weeks' gestation (mean 35⅕ weeks) referred for biophysical profile testing. All subjects had intact membranes and singleton fetuses that demonstrated three discrete episodes of movement within a 30-minute period. A single movement comprised extremity flexion and extension or trunk motion combined with extremity flexion and extension. Excluded were patients with fetal anomaly, abnormal fetal karyotype, multiple gestation, ruptured membranes, or failure to exhibit the required number of fetal movements.

An amniotic fluid index was determined for each subject by the method of Phelan et al.[1] On completion of the biophysical profile[5] the amniotic fluid index was

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