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Fetal responses to induced maternal relaxation during pregnancy

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Abstract

Fetal responses to induced maternal relaxation during the 32nd week of pregnancy were recorded in 100 maternal-fetal pairs using a digitized data collection system. The 18-minute guided imagery relaxation manipulation generated significant changes in maternal heart rate, skin conductance, respiration period, and respiratory sinus arrhythmia. Significant alterations in fetal neurobehavior were observed, including decreased fetal heart rate (FHR), increased FHR variability, suppression of fetal motor activity (FM), and increased FM-FHR coupling. Attribution of the two fetal cardiac responses to the guided imagery procedure itself, as opposed to simple rest or recumbency, is tempered by the observed pattern of response. Evaluation of correspondence between changes within individual maternal-fetal pairs revealed significant associations between maternal autonomic measures and fetal cardiac patterns, lower umbilical and uterine artery resistance and increased FHR variability, and declining salivary cortisol and FM activity. Potential mechanisms that may mediate the observed results are discussed.

“When a pregnant woman falls, the baby in the womb answers” West African proverb

Literary and historical works are replete with descriptions of the impact of maternal perceptions, sensations and emotions on the fetus. There are no direct neural connections between mother and fetus, but maternal experiences generate a cascade of physiological and neurochemical consequences that may alter the intrauterine milieu either directly or indirectly and thereby generate a fetal response. Provocative reports of linkage between maternal stress and fetal heart rate and behavior have appeared in the academic literature since the 1930's (Sontag & Wallace, 1934). Anecdotal evidence reported since that time suggests temporary dysregulation of either fetal heart rate or behavior following a maternal fall (Hepper & Shahidullah, 1990), an earthquake (Ianniruberto & Tajani, 1981), and sounding of an air raid alarm during the Gulf War (Yoles, Hod, Kaplan, & Ovadia, 1993).

Only a handful of studies measure fetal responses to experimental manipulation of maternal psychological state. Fetal tachycardia was observed in response to a variety of situations imposed on pregnant women, ranging from the mild (i.e., presentation of loud sounds) to more alarming (i.e., deceiving women that their fetus was inadequately oxygenated) (Copher &

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Huber, 1967). The use of a less threatening stimulus to induce maternal arousal, a tape recording of a crying infant, has been associated with a decelerative fetal heart rate response in anxious, but not in non-anxious or depressed women (Benson, Little, Talbert, Dewhurst, & Priest, 1987). Three studies report fetal responses to maternal arousal induced by a common cognitive challenge that incurs a sympathetic response, the Stroop Color Word Test. These include increased variability in heart rate concomitant with suppression of motor activity (DiPietro, Costigan, & Gurewitsch, 2003) and increased fetal heart rate in fetuses of women with high trait anxiety or depression (Monk et al., 2000; Monk et al., 2004).

To date, experimental research on the transmission of maternal state to the fetus has focused on stressful manipulations. This orientation is consistent with long-standing interest in more chronic effects of maternal psychological distress on untoward pregnancy outcomes (Paarlberg, Vingerhoets, Passchier, Dekker, & van Geijn, 1995). As a result, pregnant women are often urged to relax more frequently. Research has begun to ascertain the efficacy of this admonition by examining whether prolonged stress reduction interventions, including yoga (Narendran, Nagarathna, Narendran, Gunasheela, & Nagendra, 2005), progressive relaxation (Janke, 1999), and massage (Field, Diego, Hernandez-Reif, Schanberg, & Kuhn, 2004) help maximize pregnancy outcomes. Such therapeutic interventions reliably generate beneficial alterations to mood, anxiety, and depressive symptoms and there is modest support for a positive impact on gestational duration and/or birth weight (Janke, 1999; Narendran et al., 2005), as well as prenatal and perinatal complications (Field et al., 2004). A number of potential mechanisms for these findings have been proposed (Tiran & Chummun, 2004), and there is some empirical support for long-term mediation through alterations in neuroendocrine systems, including cortisol (Urizar *et al.*, 2004) and catecholamines (Field et al., 2004).

However, these interventions have been implemented in advance of basic understanding of the immediate maternal and fetal effects that such activities generate. Only one report examines contemporaneous effects of an active relaxation protocol (i.e., guided imagery directed by a therapist) on physiological functioning in pregnant women; findings include reduced maternal heart rate and cortisol in response to the intervention (Teixeira, Martin, Prendiville, & Glover, 2005). We have been unable to identify any study that has systematically examined how or whether maternal relaxation affects fetal functioning. The goal of the current research was to extend inquiry regarding the effects of maternal relaxation during pregnancy into the fetal domain. A number of maternal physiological indicators were included to confirm the efficacy of the experimental relaxation manipulation and provide information regarding source mechanisms. These include heart and respiratory rates, measures commonly used in studies to evaluate systemic relaxation responses. Both are influenced by non-neural and neural processes, as well as parasympathetic and sympathetic control. To better isolate sympathetic effects, electrodermal activity was also measured. Skin conductance reflects changes in conductivity of the skin mediated by eccrine glands which are singly innervated by the sympathetic branch of the nervous system (Venables, 1991). Activity in the hypothalamic-pituitary-adrenal (HPA) axis was assessed via salivary cortisol. In addition, blood flow in the uterine and umbilical vessels was inferred by measuring resistance in these arteries using Doppler technology. Decreased blood flow to the uterus can generate increased placental resistance to umbilical arterial flow and a stress on fetal cardiac function (Trudinger, 1994). Uterine artery resistance has been linked to factors that affect maternal peripheral blood flow, including anxiety (Sjostrom, Valentin, Thelin, & Marsal, 1997; Teixeira, Fisk, & Glover, 1999).

Ascertainment of fetal responsiveness to maternal relaxation was based on fetal heart rate, motor activity, and their interrelation. These measures of fetal functioning are typically referred to as neurobehaviors, develop in predictable ways over the course of gestation, and are widely regarded as indicators of the developing fetal nervous system (Hepper, 1995; James, Pillai, &

Smoleniec, 1995; Maeda et al., 2006; Nijhuis & ten Hof, 1999; Yoshizato et al., 1994). In particular, fetal heart rate variability is a well-known indicator of fetal well-being (Parer, 1999), and the degree of coupling between brief acceleratory changes in fetal heart rate in response to motor activity provides an indicator of integration between neural circuits (Baser, Johnson, & Paine, 1992; DiPietro et al., 2006).

Induced maternal relaxation was expected to invoke maternal autonomic responses consistent with sympathetic withdrawal and/or parasympathetic activation, indicated by reduced maternal heart rate, slowed respiration, decreased skin conductance, and increased respiratory sinus arrhythmia, as well as transient HPA suppression ascertained through cortisol output, and decreased vascular resistance. Given the lack of available evidence relevant to fetal effects, our hypotheses were based on the converse of observations generated under acute or persistent conditions of maternal stress or arousal. As such, we expected that induced maternal relaxation would generate decreased fetal heart rate, increased fetal heart rate variability and fetal movement-fetal heart rate coupling, and increased fetal motor activity. In addition, we expected that variation in the magnitude of the maternal physiological responses would correspond to the magnitude of the fetal response within maternal-fetal pairs.

Methods

Participants

Eligibility was restricted to normotensive, non-smoking women with uncomplicated pregnancies at the time of enrollment carrying singleton fetuses. Accurate dating of the pregnancy was required and based on early first trimester pregnancy testing or examination and confirmed by ultrasound. A total of 100 self-referred pregnant women were enrolled. Participants continued to have generally healthy pregnancies; significant subsequent pregnancy complications were uncommon but included a range of conditions such as gestational diabetes ($n = 2$) and anemia ($n = 3$). Most participants (96%) delivered at term. Sociodemographic characteristics reflect a sample of mature, college-educated (M age = 31.1, $sd = 4.8$, range 21–43; M years education = 16.7 years, $sd = 2.3$, range 12 to 20), and married (91%) women. Mean maternal weight and height, collected by self-report, were 148 lbs and 64.5 inches, respectively. Most (81%) women were non-Hispanic white; the remainder was African-American (12%), Hispanic or Asian (7%). Forty-eight percent of the fetuses were female and this was the first child for 56% of the sample.

Design and Procedure

The relaxation protocol took place during the 32nd week of gestation. Testing commenced at 13:30 and women were instructed to eat 1.5 hours prior to the visit but not thereafter. Upon arrival, a brief ultrasound scan was administered to determine fetal position, collect Doppler blood flow data (see below), and provide photographs for parents. Maternal-fetal monitoring began once women were comfortably positioned in a semi-recumbent, left-lateral posture. Eighteen minutes of baseline, undisturbed data were collected, followed by an eighteen minute long guided imagery, progressive relaxation audio recording (“Beach Summer Day”, Suki Productions, Cincinnati, OH) delivered through headphones with lights dimmed. Progressive relaxation can involve either systematic tensing followed by relaxation of muscle groups or conscious release of tension without initial contraction. The latter approach was selected because it has been shown to evoke a greater electromyographic relaxation response (Lucic, Steffen, Harrigan, & Stuebing, 1991). The instructions guided women through imagery designed to release tension and foster a relaxed state. After the relaxation interval, the lights were abruptly switched on and women were asked a series of questions pertaining to the experience. An additional 18-minute post-relaxation period followed, during which time women listened to their choice of several instrumental musical selections (classical or New

Age). Music was used in this period to discourage further conversation which may provide a separate source of stimulation to the fetus.

To control for the possibility of temporal effects on maternal or fetal responses related simply to maternal recumbency or postural change during mid-day and not the manipulation, an additional 18 minute baseline was instituted for approximately half (41%) of the subjects, generated by a random number table. This pre-baseline preceded the baseline period used in data analyses.

Psychological responses

Women completed the Physiological Tension and Physical Assessment subscales of Relaxation Inventory (Crist, Rickard, Prentice-Dunn, & Barker, 1989), designed specifically to assess the efficacy of relaxation protocols, upon arrival and following the relaxation period. This inventory includes 35 items (e.g., my jaw is set tight; I have a clear mind) rated on 7-point Likert scales. Test-retest reliability for the Physiological Tension subscale is .87 over three days and .95 over within-day trials with alpha coefficient of .89. Comparable values for the Physical Assessment subscale were .87 (days), .97 (trials) and .95 (alpha). After reverse scoring of some items, higher scores reflect greater relaxation. Participants were queried regarding the degree to which they regularly practice assorted relaxation techniques.

Maternal-fetal monitoring

Maternal physiological signals were amplified using a multi-channel, electrically isolated, bioamplifier (Model JAD-04; James Long Company, Caroga Lake, NY). Data were digitized on a personal computer at 1000 Hz via an external analog to digital board using Snapstream data acquisition system (HEM Data Corporation, Southfield, MI). Electrocardiogram was recorded from 3 carbon fiber disposable electrodes in triangulated placement (right mid sub-clavicle, left mid axillary thorax, and upper left thigh for ground lead). Electrodermal activity was monitored from two silver-silver chloride electrodes with a gelled skin contact area placed on the distal phalanges of the index and second fingers of the nondominant hand. Electrodes were affixed with adhesive collars to limit gel contact to a 1 cm diameter circle and secured with Velcro. Respiration was measured from a bellows apparatus stretched across the ribcage below the breasts.

Data quantification proceeded off-line using the PHY General Physiology System and IBI Analysis Systems (James Long Company). ECG data underwent R-wave detection, manual editing for artifact, and interbeat interval computation in ms; data were converted to heart rate to maintain comparability with fetal measures. Skin conductance level (SCL) was measured by administering a constant 0.5 volt root-mean-square 30 Hz AC excitation signal and detecting the current flow. SCL was scaled from 0 to 25 μ S. Respirations were measured by quantifying inspiration to inspiration and expiration to expiration periods based on the detected peaks and troughs of the respiratory waveform and respiratory period (RP), the interval between breaths, was computed (s). These data were also used to quantify respiratory sinus arrhythmia (RSA); units represent peak to valley changes in IBI from inspiration to expiration (ms) (Grossman et al., 1990).

Fetal data were collected using a Toitu (MT320) fetal actocardiograph. This monitor detects fetal movement (FM) and fetal heart rate (FHR) with a single, wide array transabdominal Doppler transducer and processes this signal through a series of autocorrelation techniques. The actograph detects fetal movements by preserving the remaining signal after bandpassing frequency components of the Doppler signal that are associated with FHR and maternal somatic activity. Reliability studies comparing actograph based versus ultrasound visualized fetal movements have found the performance of this monitor to be highly accurate in detecting both

fetal motor activity and quiescence (Besinger & Johnson, 1989; DiPietro, Costigan, & Pressman, 1999; Maeda, Tatsumura, & Utsu, 1999).

Fetal data were digitized and time synchronized with the maternal data. Data were resampled and analyzed off-line using software developed in our laboratory (GESTATE; James Long Company, Caroga Lake NY). FHR data underwent error rejection procedures based on moving averages of acceptable values as needed. Variable extraction included FHR and FHR variability (standard deviation of each 1-min epoch aggregated over time). Fetal movement data represent raw voltage values generated from the actograph, calibrated by multiplying by a conversion factor with adjustment for offset, and scaled from 0 to 100 in arbitrary units. A movement bout was considered to begin when the first spike of the actograph attained amplitude of 15 units and end when there was a cessation of 15 unit signals for at least 10 s. The number of movements was counted. The relation between FM and FHR (hereafter, FM-FHR coupling), was calculated as the proportion of fetal movements associated with excursions in $FHR \geq 5$ bpm over baseline within 5 seconds before the start of a movement or within 15 seconds after the start of a movement, consistent with previously developed criteria (Baser et al., 1992; DiPietro, Hodgson, Costigan, Hilton, & Johnson, 1996).

Maternal and fetal data were epoched in 1 min intervals. Mean values during each interval (pre-baseline, baseline, relaxation, and post-relaxation) were computed.

Doppler velocimetry

Uterine and umbilical artery blood flow was measured with Doppler ultrasound (Picus, Pie Medical) using a 3.5 MHz transabdominal probe. Color Doppler imaging was used to locate the main branches of the right and left uterine arteries and the Doppler gate was positioned close to their junction with the internal iliac artery. The angle of insonation was adjusted to maximize the systolic peak with an angle less 35° . A series of 3 consecutive waveforms were recorded and traced. A standard clinical indicator of arterial resistance (i.e., Resistance Index; RI) was calculated using values derived from the single best waveform. The umbilical artery was similarly imaged with color Doppler and the RI was computed based on sampling near the midpoint. Umbilical data were collected on all subjects; uterine artery data collection was not initiated until after the study had begun, commencing with the 14th participant. Logistical considerations, including the necessary alteration to maternal positioning which may have influenced other results, and the time needed to collect velocimetry data, precluded more frequent measurement or a different schedule of administration for participants with and without a pre-baseline control period.

Cortisol

Saliva was collected at 6 points during the procedure immediately following: arrival, ultrasound scan, baseline (the second baseline for individuals with two), relaxation, post-relaxation, and again shortly before departure. Saliva was collected by placing a small, specially cut, filter paper (2.5×9.0 cm, Whatman Grade 42) in the participant's mouth and having them thoroughly moisten the filter. Excess saliva was eliminated as the paper passed between the lips upon removal. Filters were allowed to air dry and were extracted by cutting a fixed section. Cut filter papers were placed in a 1.4 ml microcentrifuge tube to which 0.5 ml of assay buffer was added. Tubes were shaken for 24 hr after which the extraction buffer was added in duplicate to the appropriate wells of the EIA assay plate. Extraction dilutes the saliva approximately 1:5. After taking the dilution into consideration, the detection limit is 0.019 $\mu\text{g/dl}$ for cortisol. This procedure and validation has been described in detail previously (Neu, Goldstein, Gao, & Laudenslager, 2007). Salivary cortisol concentration in the extraction buffer was determined using a commercial expanded range high sensitivity EIA kit (No. 1-3002/1-3012, Salimetrics) that detects cortisol in the range of .003 – 3.0 $\mu\text{g/dl}$. Standard curves were fit by a weighted

regression analysis using commercial software (Revelation 3.2) for the ELISA plate reader (Dynex MRX). This kit shows minimal cross reactivity (4% or less) with other steroids present in the saliva. Controls were run on every plate for determination of inter- and intra-assay variability, which are less than 6% for this procedure.

Data analysis

Examination of the effects of the experimental manipulation on maternal-fetal measures was accomplished by 2×3 repeated measures analysis of variance conducted by group (control pre-baseline period versus none) by time (baseline, relaxation, post-relaxation). Repeated measures analysis of variance was also used to ascertain effects on velocimetry and psychological state (2×2 , pre to post periods) and cortisol (2×6 , based on 6 cortisol samples). Results are interpreted in terms of change over the periods of study as well as whether groups with and without an additional baseline period differed in level (main effect) or pattern of change over time (interaction). Maternal response patterns were analyzed by parity (nulliparous v multiparous); fetal responses were examined for sex differences.

Correspondence between maternal and fetal responsivity was evaluated in several ways depending on the sampling interval. Change scores for reactivity (baseline to relaxation) and recovery (relaxation to recovery) were computed for maternal physiological and fetal neurobehavioral measures and correspondence was evaluated by correlation coefficients specific to each adjacent interval pair. Although there has been some past dispute over the value and characteristics of change scores, pursuant discourse has established the utility of change scores in the evaluation of individual differences (Willett, 1997). Because Doppler data were collected only twice, at the onset and conclusion of the protocol, change scores for these measures were analyzed in relation to computation of the area under the curve (AUC) scores for each fetal measure, which consolidates change over an entire observation period (Pruessner, Kirschbaum, Mienlshmid, & Hellhammer, 2003). Selection of change score computation from the multiple cortisol sampling was based on consideration of the typical temporal lag of approximately 20 minutes between an exposure and HPA activation as reflected in salivary cortisol (Dickerson & Kemeny, 2004; Gozansky, Lynn, Laudenslager, & Kohrt, 2005). Since the initiation of the laboratory visit included experiences that may affect cortisol (e.g., parking and locating laboratory facility) and the expected delay between experience of the relaxation manipulation and HPA suppression, the period selected for analysis encompassed that from post-ultrasound scan (2nd sample) to after the 18-minute recovery period (5th sample).

Results

No differences were detected in participants randomly assigned to the pre-baseline control in sociodemographic measures (e.g., maternal age, education, parity), fetal characteristics (e.g., sex), past use of relaxation procedures, or maternal report of current state on the relaxation questionnaire as compared to those who were not. In addition, there were no group differences in initial cortisol level or Doppler blood flow measures. Testing was discontinued for a single individual due to a maternal condition (i.e., persistent cough) that generated signal artifact; data analysis is based on the remaining 99 participants.

Maternal response to experimental procedure

As expected, women reported a significant increase in indicators of psychological and physiological relaxation immediately following the experimental manipulation, M pre-relaxation = 98.8, sd = 3.1; M post = 131.7, sd = 2.8; F (1, 98) = 169.74, p < .0001).

The experimental manipulation also generated the intended effect in all four maternal physiologic measures. Data are presented in Table 1. In general, MHR and SCL declined during

the intervention while RP and RSA increased ($ps < .0001$). It is difficult to ascertain whether the increase in RSA reflects parasympathetic activation or is an artifact of the induction of slowed respiration. Post hoc analyses indicated significant changes over time from the baseline period to relaxation and relaxation to recovery for each variable ($ps < .0001$). Table 1 also includes data stratified by pre-baseline condition. Condition did not influence findings for MHR, RP, or RSA. However, participants in the pre-baseline condition showed a significant increase in SCL from the pre-baseline to baseline period which was maintained throughout testing, $F(1,97) = 6.00, p < .05$, and a more pronounced reduction in SCL to the relaxation procedure, as indicated by a significant time X group interaction, $F(2,194) = 15.11, p < .0001$. However, when analyzed separately, both groups showed significant change over time in SCL from baseline to relaxation, and relaxation to recovery.

Most (66%) women reported that they participated in no routine activities to encourage relaxation. Of those that did, the most commonly nominated techniques included yoga (7.3%) and meditation (4.5%). However, few of these reported using relaxation techniques daily (2.7%). Prior maternal experience, which may have generated practice effects, was found to be unrelated to maternal responsiveness to the procedure. There were no differences in maternal responsivity based on parity.

Maternal cortisol responses to experimental protocol

Serial cortisol data were collected and analyzed for only a subset of the sample; 61 cases had complete data at each of the 6 collection points ($ns = 34$ and 27 with and without prebaseline periods). Data analyses were based on natural log transformation of all cortisol values; however untransformed values are presented in Table 2. Cortisol declined significantly over time, $F(5, 295) = 38.42, p < .0001, \eta_p^2 = .394$. Paired comparisons for successive intervals, noted in Table 2, indicate significant decline in cortisol between each period except the first two and last two. There was no main effect for condition and post-hoc contrasts revealed no significant differences at any time point between groups. However, there was a significant overall group by time interaction, $F(5,295) = 2.57, p < .05, \eta_p^2 = .042$, indicating less decline in the pre-baseline group. Cortisol values were unaffected by maternal parity.

Maternal and fetal blood flow responses to the experimental procedure

As a result of inclusion of uterine velocimetry after the study began and/or failure to obtain an adequate series of waveforms, a total of 83 cases were available for repeated measures analysis of the right artery and 75 for the left artery. No changes were detected in the RI in either the right or left maternal uterine artery during the protocol. However, resistance in the umbilical artery declined over time (from $MRI = .638, sd = .06$ to $MRI = .619, sd = .07$; $F(1,92) = 7.63, p < .01, \eta_p^2 = .077$). Six cases were excluded from this analysis because a sufficient waveform could not be obtained at one or both measurement periods. There was no interaction with condition and post-relaxation umbilical RI values did not differ between those with and without a pre-baseline condition.

Fetal response to maternal relaxation procedure

The first set of analyses describes the overall pattern of responses for both conditions collapsed over the baseline, relaxation, and recovery periods. The available sample for analysis of FM activity and FM-FHR coupling data was reduced by 5 cases as a result of extended periods of fetal hiccups or breathing motions during one or more experimental segments which generated artifact in the FM signal. There was significant change in all four fetal parameters over time. Figure 1 through Figure 4 illustrate each measure, and include significance levels of changes between adjacent intervals. The overall F for time for FHR (Figure 1) was significant, $F(2,194) = 3.07, p < .05, \eta_p^2 = .031$, however post hoc analysis indicates that this change was limited primarily to the decline from the baseline to relaxation intervals, $F(1,98) = 9.74, p < .01, \eta_p^2$

= .090. While FHR variability (Figure 2) also changed significantly over time, $F(2, 194) = 5.81, p < .01, \eta_p^2 = .057$, the increase was linear through the post-relaxation period. Once again, the primary change was observed from the baseline to relaxation period, $F(1, 98) = 8.65, p < .01, \eta_p^2 = .081$. In contrast, FM activity (Figure 3) also changed over time, $F(2, 182) = 12.45, p < .0001, \eta_p^2 = .120$, but the suppression observed from baseline to relaxation was significant, $F(1, 91) = 24.63, p < .0001, \eta_p^2 = .213$ as was the motor rebound from relaxation to recovery, $F(1, 91) = 16.27, p < .0001, \eta_p^2 = .150$. Similarly, the significant change in FM-FHR coupling (Figure 4), $F(2, 182) = 7.25, p < .001, \eta_p^2 = .074$, included both augmentation from baseline to relaxation periods, $F(1, 91) = 11.45, p < .001, \eta_p^2 = .112$, and reduction from relaxation to recovery $F(1, 91) = 7.62, p < .01, \eta_p^2 = .076$. No differences in responsivity were detected by fetal sex.

The second set of analyses considered whether inclusion of a pre-baseline period altered the findings in a manner that is inconsistent with attributing the fetal response to the manipulation. There was a near significant time by condition interaction for FHR, $F(2, 194) = 2.66, p < .10$. Inspection of Figure 1 and post-hoc analyses reveal a significant decrease in FHR from baseline to relaxation only for the group without a pre-baseline interval, $F(1, 56) = 12.03, p < .001, \eta_p^2 = .180$, and not the pre-baseline group, $F(1, 41) = .19$. No other fetal variable was affected by the additional recumbent control period.

Correspondence between individual maternal-fetal responses

Maternal reactivity and recovery responses were significantly associated for MHR, $r(97) = .28, p < .001$, SCL, $r(95) = .24, p < .05$, RP, $r(84) = .70, p < .001$, and RSA, $r(86) = .59, p < .001$, such that individuals with greater relaxation responses also showed greater recovery after the procedure. Two outliers were identified for SCL and RP responsivity each and are excluded from these correlations. Similarly, fetuses that mounted a large initial response in each parameter showed greater rebound following termination of the relaxation manipulation: FHR, $r(97) = .31$, FHR variability, $r(97) = .34$, FM activity, $r(91) = .41$, and FM-FHR coupling, $r(91) = .64, ps < .001$. Fetuses of women who reported greater psychological relaxation to the procedure showed greater FHR reactivity and recovery, $r(96) = .27, p < .01$ and $r(96) = .20, p < .05$, respectively. To control for the law of initial values, these and subsequent correlations control for the first value (i.e., baseline for reactivity or relaxation for recovery) for each maternal change score. Maternal SCL and RP reactivity were related to FHR reactivity, $r(94) = .22$, and $r(90) = -.21$, respectively, $ps < .05$, and there was a trend level association between maternal RSA and FHR variability reactivity, $r(90) = .19, p < .10$. Maternal HR recovery was significantly related to both FHR, $r(96) = .43, p < .001$, and FHR variability recovery, $r(97) = .21, p < .05$. There were no significant associations between maternal autonomic measures and FM activity or FM-FHR coupling.

Right and left uterine artery change scores were averaged (M decline = .01; sd = .06). Uterine RI change was significantly associated with AUC scores for FHR, $r(58) = .30, p < .05$, and FHR variability, $r(58) = -.36, p < .01$, controlling for initial averaged RI. The associated maternal factor for uterine artery change was the degree to which women slowed their breathing during the relaxation procedure, $r(63) = -.24, p < .05$, although there was a trend association with MHR change, $r(63) = .22, p < .10$. Greater reduction in umbilical resistance was also associated with increased FHR variability, $r(74) = -.28, p < .01$.

Given the lag time in HPA reactivity, cortisol change scores from pre-baseline sample to the post recovery period (M change = .08 $\mu\text{g/dl}$; sd = .06) reflect responsiveness through the completion of the relaxation period. There was a significant association between the degree of cortisol reactivity and FM suppression, $r(56) = .31, p < .05$, and a trend association with FHR variability, $r(60) = -.24, p = .06$.

Discussion

Consistent with expectations regarding evoked relaxation, during the relaxation protocol women demonstrated reductions in psychological tension, heart rate, skin conductance, respiration, and cortisol levels, and increased respiratory sinus arrhythmia. The degrees to which these were elicited by the guided imagery relaxation manipulation per se, as opposed to simple rest and/or recumbency, requires evaluation of response patterns between the subsample that received a pre-baseline period as compared to the group that did not. Respiratory period provided the most clarity in this regard by exhibiting an unambiguous pattern of augmentation, followed by a return to baseline coincident with protocol onset and offset. Given the focus on directed breathing in such interventions, this demonstrates maternal compliance with task demands and provides a foundation for other autonomic changes. Reduction of maternal heart rate showed a similar temporal linkage to the relaxation protocol, although there was evidence of less recovery following its conclusion. Suppression of sympathetic activation, as measured through skin conductance, was less clear because there was an initial rise from baseline level and maintenance of higher level throughout in the group that received a pre-baseline period. This may indicate that an unintended consequence of this extra period was a maternal anticipatory response as a by-product of providing women with additional time to ruminate over what was to come next. Nonetheless, when analyzed separately, both groups showed significant SCL decrease followed by increase, despite the difference in initial level.

The degree to which the salivary cortisol decline can be attributed to the manipulation is less evident. Despite the elevation in maternal cortisol levels during pregnancy, (Scott, McCarrigle, & Lachelin, 1990) the diurnal rhythm typically observed in non-pregnant populations (Kirschbaum & Hellhammer, 1989) is also observed in pregnancy (Harville *et al.*, 2007). In the current study, cortisol began to decline commencing only after the baseline period and continued in a linear fashion through the post-recovery period. Given the compression of the change to within approximately a 45-minute time period (i.e., from 14:30 to 15:16), but not in the 30 minutes before or 20 minutes after, it is unlikely that the typical afternoon decline accounts for this finding. Moreover, the degree of decline (i.e., .08 $\mu\text{g/dl}$) observed during this period is comparable to that reported for the normal daily decline in pregnancy between the hours of 11:00 and 17:00 (Harville *et al.*, 2007). However, since the decline began during the baseline recording, we are unable to distinguish whether it was a result of rest or more actively directed relaxation. At least one other study reported a reduction in cortisol following both active maternal relaxation during pregnancy, effected through guided imagery, and simply resting with a magazine while seated (Teixeira *et al.*, 2005).

We turn now to the focus of this study, which involves evaluation of whether there is a fetal response to induced maternal relaxation. A pre-baseline control period was used rather than a second day of testing without a relaxation procedure for a number of reasons. Paramount among these is that maternal postural changes are a known source of stimulation for the fetus (Lecaneut & Jacquet, 2002) and it is common for women to report an increase in fetal motor activity following recline although it is unclear whether this is a result of enhanced perception or actuality. In addition, what constitutes an appropriate control period for a relaxation intervention is difficult to ascertain, particularly since successful fetal monitoring requires relative maternal immobility. Given these issues, and the within-subject nature of the data analyses, a pre-baseline period was selected as the most useful design control.

Two of the four fetal measures, FM and FM-FHR coupling, showed clear responsiveness to relaxation protocol onset and offset; the pre-baseline failed to generate a significant effect on either measure, supporting the linkage to the relaxation procedure. As hypothesized, FM-FHR coupling was augmented during the procedure, but in contrast to expectations, fetal motor activity was suppressed. While it is tempting to conclude that maternal relaxation generates

fetal motor “relaxation”, there is reason to think otherwise. The pattern of fetal motor suppression and release observed here is very similar to the fetal motor response observed to an acute maternal stressor (i.e., the Stroop Color-Word Test), despite a different pattern of maternal physiological activation (DiPietro et al., 2003). We will return to this observation subsequently.

Interpretation of the fetal cardiac measures is somewhat less clear. For heart rate, the expected decline followed by partial rebound was observed in fetuses without the pre-baseline period but was not evident in those with the additional 18-minute period. For that group, the magnitude of the initial decline from pre-baseline to baseline is consistent with that observed for the first period in the other group, suggesting that the initial decline in FHR is the result of maternal recumbency. While there is slight rebound in FHR, levels do not return to baseline, signifying a persistent effect of maternal rest on this parameter. A more striking illustration of a prolonged consequence of maternal rest was displayed by FHR variability, which, in contrast to all other measures, showed a linear increase from the pre-baseline condition through recovery. Thus, although maternal parameters normalized during the recovery period that included rousing, turning on the lights, and completion of a short interview, FHR variability remained elevated. Uterine arterial resistance, which did not show an overall group effect to the manipulation, confirming a report by another group (Teixeira et al., 1999), and umbilical resistance, which did, were both associated with FHR variability within individuals. Although participants with an additional pre-baseline period of rest did not show higher magnitude in umbilical resistance decline, a conservative approach is to assume that resistance decline was the result of simple rest since measurements were taken only twice. However, it appears that increased blood flow, which in turn is associated with enhanced perfusion, benefits fetal heart rate variability, irrespective of origin.

The observed fetal effects demonstrate a fetal response elicited by presentation of a stimulus, in this case, an auditory one, to only the mother. To date, such an effect has been demonstrated only by stress-inducing manipulations designed to increase, not decrease, physiological arousal (Copher et al., 1967; Monk et al., 2000). All maternal psychological events must be physiologically transduced to the fetus and the observed associations between responses of individual maternal-fetal pairs suggest some mediation between maternal autonomic measures and FHR parameters. However, the magnitudes of the detected associations between maternal autonomic measures and fetal cardiac patterns are quite modest but are consistent with those observed elsewhere under both elicited (DiPietro et al., 2003; Monk et al., 2004) and baseline (DiPietro, Irizarry, Costigan, & Gurewitsch, 2004) conditions. Similarly, although there was a significant association between the degree of cortisol decline and suppression of movement, as well as a trend association between cortisol decline and FHR variability change, the strongest association accounted for only 9% of the variance.

Although no study can exhaustively measure all potential maternal physiological mediators, the comparability between the fetal results generated by both a relaxation procedure and a stressful cognitive challenge (i.e., fetal motor activity suppression and FHR variability augmentation) (DiPietro et al., 2003) in the presence of opposing maternal heart rate and skin conductance directional changes, supports a more unorthodox interpretation than one founded on direct maternal physiological influence. We suggest that at least some of the observed response may be mediated by fetal perception of changes in the intrauterine milieu inspired by the manipulation, and as such, reflects a fetal orienting response. This possibility has been broached previously based on the rapidity of onset of a fetal response to a maternal event (DiPietro et al., 2003; Novak, 2004). FHR responses have been observed within seconds of disruptions of the maternal environment in investigations of sensory capacities, including maternal postural changes (Lecaneut et al., 2002) and auditory stimuli (Groome *et al.*, 2000) and it is clear that sounds generated by maternal vasculature and the digestive tract are

prominent in the uterine auditory environment (Querleu, Renard, Boutteville, & Crepin, 1989). It is possible that induced maternal relaxation may generate a biphasic response that includes a rapid sensory-mediated component as well as a secondary response mediated by neurohormonal or vasodilatory processes that extends beyond the confines of this study's protocol.

There is burgeoning academic and clinical interest in application of training in relaxation techniques to ameliorate potentially hazardous consequences of stress, reduce pregnancy complications, and maximize the labor and delivery experience (Bastani, Hidarnia, Montgomery, Aquilar-Vafaei, & Kazemnejad, 2006; Nickel *et al.*, 2006; Saisto, Toivanen, Salmela-Aro, & Halmesmaki, 2006). Despite the well-educated, fairly affluent nature of our sample, the prime population seeking complementary therapies, few pregnant women routinely engage in relaxation techniques. The concept that relaxation is beneficial to pregnancy but is rarely practiced is echoed in a sample of Bangladeshi women of low socioeconomic status (Akram *et al.*, 2000). The goal of this study was not to evaluate regimes to foster relaxation in pregnancy but rather to reveal the acute effects on fetal neurobehaviors. However, it is not unreasonable to expect that repeated and systematic exposure to periods of relaxation might generate more persistent alterations to maternal and fetal functioning, with potential longer-term consequences for child development. However, significant additional inquiry is needed to adequately distinguish between the effects of simple maternal repose from active relaxation strategies in the generation of these effects, particularly with respect to broader alterations to the intrauterine milieu related to blood flow and the neurohormonal environment.

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Figure 1.

Although fetal heart rate (bpm) changed significantly over time, $F(2,194) = 3.07, p < .05$, the change between segments was limited to the baseline to relaxation periods for the group without a pre-baseline period only, $F(1, 56) = 12.03, ***p < .001$. Unlike other figures, significance notation here refers to this group only.

Figure 2.

Fetal heart rate variability (bpm) showed linear increase over time, $F(2, 194) = 5.81, p < .01$, but change between segments was focused on the baseline period to relaxation, $F(1, 98) = 8.65, **p < .01$.

Figure 3.

Fetal movement (arbitrary units) suppression was displayed during the relaxation period with return to baseline levels following the recovery period; this pattern was unaffected by pre-baseline condition. *** $p < .0001$.

Figure 4.

Fetal FM-FHR coupling was augmented during the relaxation period and returned to baseline levels following the recovery period; this pattern was unaffected by pre-baseline condition.

** $p < .01$. *** $p < .001$.

Table 1

Maternal physiological responsiveness during relaxation protocol

	Pre-baseline <i>M</i>	Baseline <i>M</i>	Relaxation <i>M</i>	Recovery <i>M</i>	<i>F</i> Time (<i>df</i>)
Heart rate (bpm)					
All (<i>n</i> = 99)	--	86.40	82.57	83.94	54.00*
Group 1 (<i>n</i> = 57)	--	85.88	81.92	83.59	(2,194)
Group 2 (<i>n</i> = 42)	88.04	87.11	83.47	84.41	
Skin Conductance (μS)					
All (<i>n</i> = 99)	--	6.37	5.64	7.22	64.13*
Group 1 (<i>n</i> = 57)	--	5.33 ⁺	4.98 ⁺	6.80	(2,194)
Group 2 (<i>n</i> = 42)	5.84	7.78	6.53	7.78	
Respiratory period (s)					
All (<i>n</i> = 88)	--	3.86	4.68	3.93	42.49*
Group 1 (<i>n</i> = 52)	--	3.75	4.48	3.85	(2,172)
Group 2 (<i>n</i> = 36)	3.85	4.00	4.96	4.03	
Respiratory sinus arrhythmia (ms)					
All (<i>n</i> = 88)	--	39.23	54.79	41.67	24.01*
Group 1 (<i>n</i> = 52)	--	38.41	54.77	41.95	(2,172)
Group 2 (<i>n</i> = 36)	39.32	40.41	54.81	41.28	

**p* < .0001.

Note. *F*(time) refers to overall sample result with condition included in model.

⁺ refers to significant main effect by condition.

Table 2

Maternal cortisol levels ($\mu\text{g/dL}$) during relaxation protocol ($n = 61$)

	Arrival	Post-scan	Post-baseline	Post-Relaxation	Post-recovery	Departure
All	.295	.288	.251 ***	.230 **	.208 **	.206
Time	13:37	14:01	14:30	14:52	15:16	15:35
Group 1	.289	.282	.243 ***	.230	.213 *	.222
Time	13:36	13:58	14:27	14:47	15:07	15:25
Group 2	.303	.295	.261 *	.229 *	.201 **	.185
Time	13:38	14:03	14:35	15:11	15:29	15:48

* $p < .05$

** $p < .01$

*** $p < .001$

Significance level denotes change from prior period.