ORIGINAL RESEARCH

To study the effect of an acute mother stress reaction and anxiousness on the heart rate of the foetus

[1]Dr. Prabhjot Singh, [2]Dr. Chander Sheikher, [3]Dr. Aman Sharma

[1]Assistant Professor, Department of Physiology, Government Medical College and Associated Hospital, Rajouri, Jammu and Kashmir, India
[2]Associate Professor Department of Obstetrics and Gynaecology, GMC and AH Rajouri Jammu and Kashmir, India
[3]Assistant Professor, Department of Pharmacology, Government Medical College Rajouri, Jammu and Kashmir, India

Corresponding author
Dr. Chander Sheikher
Associate Professor Department of Obstetrics and Gynaecology, GMC and AH Rajouri Jammu and Kashmir, India
Email: dr.sheikher36@gmail.com

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ABSTRACT

Aim: to study the effect of an acute mother stress reaction and anxiousness on the heart rate of the foetus.
Methods and material: In this prospective research, participants were pregnant women who were not experiencing any complications with their pregnancy and carried just one baby. All pregnancies were dated using the most accurate obstetric estimate possible, which was derived from the date of the woman's most recent menstrual cycle and the foetal biometry obtained from an ultrasound during the first trimester.
Results: The mean ± SD State anxiety score was 39.25±6.78 and the mean ± SD Trait anxiety score was 45.98 ± 6.39. For state anxiety, 17 subjects had a high score and 23 subjects had a low score. Table 1 For trait anxiety, 17subjects had a high score and 23 subjects had a low score. There were no significant differences in mean ± SD maternal age in either the state anxiety group (high state anxiety 32.01 ± 4.25 years versus low state anxiety 31.98 ± 2.97 years) or the trait anxiety group (high state anxiety 32.15 ± 2.87 years versus low state anxiety 32.01 ± 3.69 years). Baseline FHR showed no significant differences between fetuses of mothers with high or low state anxiety (135.69 ± 7.69 beats/min versus 135.67 ± 5.24 beats/min, respectively), or between fetuses of mothers with high or low trait anxiety (136.71 ± 5.22 beats/min versus 135.02 ± 2.87 beats/min, respectively). The response patterns of FHR after VAS were type I in 23 fetuses (57.5%), type II in 10(25%) and type III in 7 (17.5%).
Conclusion: we concluded that the changes in neurobehavioral functioning that are associated with inadequate mood regulation in the mother can be identified as early as the period when the mother is still in utero.
Keywords: State anxiety, Trait anxiety, FHR

INTRODUCTION

Over the course of the last decade, a growing body of data has pointed the finger of blame at mother psychological stress as a contributor to less-than-desirable birth outcomes.[1,2] Studies conducted on animals showed that prenatally stressed animals exhibited, as adults, delayed motor development, reduced explorative and adaptive behaviour, greater anxiety and more emotion in response to an unfamiliar environment, as well as impaired cognitive function (attention, learning), and alterations in social and sexual behaviour. These results were found in animals that had been exposed to stress during pregnancy (e.g. feminization of masculine behaviour).[3–5] In humans, Huizink et al.6 found that if maternal stress and anxiety are high during the first trimester of pregnancy, this may be associated with a low psychomotor score on the Bayley Development Test, poor adaptation to new environments, and more problematic behaviour when the infants are 8 months old. Additionally, this may be associated with a higher risk of developing autism spectrum disorder later in

life. In a separate piece of research, the authors found that children born to mothers who experienced high levels of anxiety during pregnancy were more likely to be hyperactive, more likely to show attention deficit, and more likely to exhibit difficult behaviour and aggression in comparison to children born to mothers who experienced low levels of anxiety (control mothers).[7]

It is not simple to determine how much stress a pregnant woman is experiencing. It has been determined that the State–Trait Anxiety Inventory (STAI) is a useful instrument for objectively measuring stress levels during pregnancy.[8,9] It has been sufficiently tested and examined for consistency, and it makes a clear distinction between the transient condition known as "state anxiety" and the more widespread and persistent condition known as "trait anxiety."[10]

An identically stressful stimulus can elicit a variety of responses from different people,[2] but when a person is exposed to a stressor, the entire system that regulates stress is activated. This includes the hypothalamic–pituitary–adrenal cortex system (also known as the HPA axis), the sympathetic nervous system, and the adrenal medulla system.[11] In reaction to stress, the body produces significant amounts of a number of hormones, including corticotropin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH), cortisol, and noradrenalin. These hormones are found in the blood.[11] In addition, it has been shown that the level of chromogranin A (CgA), which is associated to catecholamine levels, rises in response to a physical stimulus with a big amplitude.[12] Monitoring the foetal heart rate, also known as FHR monitoring, is the major clinical method for determining the state of the foetus' well-being. Moreover, it is one of the most helpful methods for researching prenatal neurodevelopment. In addition, an increase in foetal heart rate (defined as more than 15 beats per minute over the baseline) is linked to foetal movement, also known as foetal responsiveness, and has been shown to have a strong correlation with foetal well-being.[13,14] Prior to 24 weeks of gestation, foetal reactivity is very uncommon, and it would seem that the advent of FHR reactivity is connected to the maturation of the central nervous system (CNS), which takes place towards the beginning of the third trimester.[13,14] During the third trimester, the only stimulus that has been shown to reliably modify both the foetal heart rate (FHR) and the movement patterns of the foetus is called vibroacoustic stimulation, or VAS.[15] There is an instantaneous FHR reaction following VAS beginning at 26 weeks of gestation and lasting till term. This response is characterised by a period of faster foetal heart beats.[16]

According to a recent study, maternal worry seemed to have an effect on both the length and variability of the FHR. Prolonged accelerations often fused into continuous tachycardia as a result of this effect.[8] According to the findings of another study, the foetuses of mothers who suffered from depression had a higher baseline FHR as well as a 3.5-fold delay in returning to baseline FHR after being exposed to the VAS.[17] So, the environment of the mother has a considerable impact on both the foetal autonomic nervous system and the central nervous system.[18]

METHODS AND MATERIAL

In this prospective research, participants were pregnant women who were not experiencing any complications with their pregnancy and carried just one baby. The health of the baby, as well as its development, as well as the amniotic fluids, were all found to be normal in every single case. Before enrolling any of the participants in the research, we made sure to get their written permission beforehand. All pregnancies were dated using the most accurate obstetric estimate possible, which was derived from the date of the woman's most recent menstrual cycle and the foetal biometry obtained from an ultrasound during the first trimester.

METHODOLOGY

About 30 weeks into the pregnancy, the Spielberger State-Trait Anxiety Inventory (STAI) was used to evaluate the mother's level of anxiety. [10] The interviews were conducted by the same researcher, and they followed a format that was only semi-structured (M.Y.). During the interviews, participants were asked questions on their obstetric history, their present physical and mental health, and their current psychosocial status. The STAI scale is comprised of thirty statements, each of which describes a distinct emotional state. [10] In 15 of these statements, the participants are expected to explain their emotional responses in terms of anxiety at a specific instant or period of time, which is referred to as state anxiety. In the next 15 questions, the individual is asked to explain how they feel in general as well as their overall inclination to react to circumstances that are commonly thought to be dangerous (also known as trait anxiety). On the basis of the state and trait anxiety scores, two groups were established: one with low anxiety scores (the low-anxiety group: low state anxiety scores from 20 to 40; low trait anxiety scores from 25 to 45), and another with high anxiety scores (the high-anxiety group: high state anxiety scores from 50 to 70). (high-anxiety group: high state anxiety scores from 40 to 70; high trait anxiety scores from 45 to 65). Scores for women in the group with low anxiety were lower than the median for the whole study population, but scores for women in the group with high anxiety were either equal to or higher than the median. The high, moderate, and low state anxiety groups, as well as the trait anxiety group, all had their FHR recordings analysed and compared. A cardiotocograph was used during the collection of the FHR data.

During 30 weeks of gestation, a foetal stimulator known as a TR30 was used to do a VAS examination

on the foetus. A measurement of the FHR response was taken either immediately after the stimulation or within one minute after the stimulation. The FHR response pattern was categorised as type I, type II, or type III as follows: type I was defined as an extended period of acceleration (15 beats/min, > 3 min) or one acceleration lasting > 1 min or at least two accelerations lasting > 15 s; type II was defined as a biphasic response with acceleration followed by deceleration; and type III was defined as either no response or a prolonged deceleration (> 60 beats/min and > 60 s). [19] In addition to that, the greatest amplitude of acceleration after VAS was measured and recorded.

After FHR monitoring at 30 weeks of gestation, about 5 ml of maternal plasma was obtained for CRH and ACTH measures, and approximately 5 ml of saliva was collected for cortisol and CgA measurements. Both samples were then held at –80 °C until analysis could be performed on them. The levels of CRH, ACTH, cortisol, and CgA were measured making use of the commercially available assays, in accordance with the instructions provided by the manufacturers: serum CRH was measured using enzyme-linked immunosorbent assay (ELISA), serum ACTH was measured using Elecsys® ACTH assay, saliva cortisol was measured using radio- immunoassay, and saliva CgA was measured using enzyme immunoassay (EIA). Apgar scores were used on the newborns just after they were delivered in order to get an initial assessment of their overall health. [20]

STATISTICAL ANALYSIS

It was not possible to calculate an appropriate sample size *a priori* because the percentage agreement between the two groups was not known in advance of starting the study. Statistical analyses were carried out using the SPSS version 24.0 (SPSS Inc., Chicago, IL, USA) for Windows®. Spearman's rank correlation was used to assess the correlation between STAI data, and the Mann–Whitney *U*-test was used to test the statistical significance of differences between unpaired continuous data. The $\chi 2$ test and Fisher's exact test were used to test the statistical significance of differences in categorical data. A *P*-value <0.05 was considered to be statistically significant.

RESULTS

A total of 40 pregnant women with uncomplicated pregnancies with a single fetus were recruited. All neonates were born in satisfactory condition. The 1- and 5-min Apgar scores were > 7 and > 9, respectively. All 40 women in the study were non-smokers and abstained from alcohol for the duration of their pregnancies. On the STAI, maternal anxiety scores were normally distributed.

The mean ± SD State anxiety score was 39.25±6.78 and the mean ± SD Trait anxiety score was 45.98 ± 6.39. For state anxiety, 17 subjects had a high score and 23 subjects had a low score.

Table 1: Anxiety score

Anxiety score	State anxiety	Trait anxiety	P value
	39.25±6.78	45.98 ± 6.39	0.02

Table 1 For trait anxiety, 17subjects had a high score and 23 subjects had a low score. There were no significant differences in mean ± SD maternal age in either the state anxiety group (high state anxiety 32.01 ± 4.25 years versus low state anxiety 31.98 ± 2.97 years) or the trait anxiety group (high state anxiety 32.15 ± 2.87 years versus low state anxiety 32.01 ± 3.69 years). In terms of the stress hormone measurements, only CRH levels were significantly different: higher in the high trait anxiety group compared with the low trait anxiety group (*P*=0.03), although there was no difference in CRH levels for state anxiety (Table 2).

Table 2: Relationship between maternal stress, as classified on the Spielberger State–Trait Anxiety Inventory and maternal stress hormone levels

Hormone	Anxiety level High (n = 17)	Anxiety level Low (n = 23)	significance
State anxiety			
CRH (pg/dl)	195.85 ± 21.36	168.29 ± 23.58	0.03
ACTH (pg/dl)	28.03 ± 4.63	31.69 ± 6.37	0.23
Cortisol (µg/dl)	0.45 ± 0.09	0.58 ± 0.09	0.52
CgA (pmol/ml)	5.99 ± 1.39	11.03 ± 3.98	0.41
Trait anxiety			
CRH (pg/dl)	209 ± 22.66	161.64 ± 12.33	0.02
ACTH (pg/dl)	26.89 ± 3.33	31.22 ± 9.63	0.44
Cortisol (µg/dl)	0.61± 0.11	0.51± 0.11	0.29
CgA (pmol/ml)	10.33 ± 2.66	8.14 ± 2.56	0.37

Table 3: Relationship between maternal stress, classified on the Spielberger State–Trait Anxiety Inventory, and fetal heart rate response after vibroacoustic stimulation carried out at approximately 30 weeks gestation

Fetal heart rate response	Anxiety level High (n = 17)	Anxiety level Low (n = 23)	significance
State anxiety			
Type I	7	14	0.36
Type II	7	4	0.52
Type III	3	5	0.44
Trait anxiety			
Type I	3	19	$P = 0.002$
Type II	9	2	$P = 0.03$
Type III	5	2	0.63

Baseline FHR (mean± SD) showed no significant differences between fetuses of mothers with high or low state anxiety (135.69 ± 7.69 beats/min versus 135.67 ± 5.24 beats/min, respectively), or between fetuses of mothers with high or low trait anxiety (136.71 ± 5.22 beats/min versus 135.02 ± 2.87 beats/min, respectively). The response patterns of FHR after VAS were type I in 23 fetuses (57.5%), type II in 10(25%) and type III in 7 (17.5%).

There were no correlations between stress hormone levels and the FHR response after VAS (data not shown). In pregnant women in the high trait anxiety group, the FHR response after VAS showed mostly a type II pattern rather than the normal response (type I). The type I response after VAS was most common in those with low trait anxiety scores ($P = 0.002$), while the type II response was associated with a high trait anxiety score ($P = 0.03$; Table 3). There were no significant differences in the type of FHR response pattern after VAS in mothers with either high or low state anxiety (Table 3).

Mean maximum amplitude of FHR acceleration after VAS was significantly lower in the high trait than in the low trait anxiety group ($P = 0.011$), however there was no statistically significant difference between the low and high state anxiety groups

DISCUSSION

The State-Trait Anxiety Inventory (STAI) is one of the measures of anxiety that has been around the longest and is used the most often. Anxiety manifests as as a trait when a person is sensitive and predisposed to experience their own specific psychological distress.[10] The sensitivity of peripheral-type benzodiazepine receptors (PBR) to acute or chronic stress has been proven in a variety of contexts, and their involvement in the regulation of the stress response is one of these contexts.[21,22] PBR on platelets were shown to have a correlation with the trait anxiety scale of the STAI in normal healthy participants, according to the findings of Nakamura et al.[23] The state and trait anxiety scores of the women who participated in the current study were within the normal range, despite the fact that they were slightly higher than the values that have been reported for males and healthy non-pregnant women 19 – 39 years old (38.6±9.8 versus 36.0±11.0 and 46.1±9.7 versus 36.0±9.5, respectively).[12] However, these values have been reported for healthy non-pregnant women 19–39 years old. There is a possibility that the FHR response might be further influenced by anxiety levels that are greater than those seen in the current research. In the current investigation, the correlation between STAI and the levels of the stress hormones CRH, ACTH, cortisol, and CgA was also investigated. It is interesting to note that the group with the high trait anxiety had a CRH level that was considerably greater than the group with the low trait anxiety. The hypothalamus is responsible for the production and secretion of CRH. This hormone is an important component of the HPA axis and is implicated in the physiological response to stress.[24] It has been established that psychosocial stresses are associated to the beginning of anxious episodes, which is consistent with the fact that anxiety is one of the most prominent symptoms of mental illnesses. A malfunction of the HPA axis may be connected with having high levels of anxiety as a characteristic.[25] In addition, Chrousos25 observed that high trait anxiety in pregnant women could impact the function and modulation of the HPA axis in the foetus through the maternal–placental–fetal neuroendocrine axis, particularly via placental CRH. This was shown to be the case. Placental CRH is released in greater amounts when there is persistent stress. It was proposed by Challis et al.[26] that placental CRH plays multiple important roles in the regulation of pregnancy. These roles include the modulation of maternal and foetal pituitary–adrenal function; participation in foetal cellular differentiation, growth, and maturation; and involvement in the physiology of parturition. Since prolonged maternal psychological distress is closely connected to markers of foetal neurobehavioural maturation, abnormally increased levels of placental CRH may play a role in poor neurodevelopment. [27] The development of the foetal autonomic nervous system is subject to strong impact from the maternal environment.[28] The conclusion that can be drawn from these results and the fact that the level of CRH was considerably greater in the high trait anxiety group in the current research is that the STAI trait anxiety may

be a valuable measure to assess chronic maternal stress.

Anxiety and stress during pregnancy are connected to changes in foetal heart rate (FHR) and foetal movement, and these abnormalities may have repercussions for a child's future emotional development.[29 – 31] A psychological test known as the Stroop color–word matching task was used by Monk et al.[32] in their investigation to see whether there were any changes in FHR reactivity linked with the mother's mental condition. This was done in order to identify whether or not these differences existed. They found that babies carried by mothers who experienced high levels of worry had larger increases in FHR compared to foetuses carried by mothers who experienced low levels of anxiety. Fetuses of pregnant women who report higher levels of life stress had lower levels of parasympathetic activation and/or higher levels of sympathetic activation, as indicated by lower levels of FHR variability. [33] It is hypothesised that a less mature central nervous system is reflected in the foetuses of mothers who experience high levels of stress and who also have a faster baseline heart rate. These mothers also exhibit reduced FHR variability and delayed maturation of the coupling between FHR and foetal movement. [32] It has been shown that elevated stress during pregnancy, and more specifically, stress that is unique to being pregnant, is related with increased foetal reactivity. These findings were obtained by assessing foetal reactivity at three different times throughout gestation. [32] When their mother was subjected to psychological stress, the foetuses of highly anxious women exhibited a rise in heart rate, but the foetuses of low-anxiety women did not demonstrate any change in heart rate when their mother was subjected to psychological stress. [31,32] In these earlier investigations, the Stroop task was used to measure the levels of maternal stress; in the current research, stress was evaluated using the STAI, and a VAS was utilised to evaluate the foetal response. Acceleration of the foetal heart rate (FHR) is thought to be a good indicator of the health of the foetus. Gagnon et al.[15] employed visual analogue scales (VAS) to elicit FHR acceleration from a gestational age of 26 weeks to term in order to demonstrate adequate foetal neurodevelopment. According to prior research done by Ingemarsson et al., the current study was able to classify three distinct patterns of responses to the VAS. [19] The FHR response following VAS indicated a largely type II pattern in pregnant women who had significant trait anxiety, as opposed to the anticipated usual type I response. As compared with the group with low levels of trait anxiety, the group with high levels of trait anxiety had a considerably lower mean maximum amplitude of the FHR's acceleration following the VAS. In contrast, there were no significant variations seen in the response pattern following the VAS for the foetuses of moms who suffered from state anxiety. Previous research has demonstrated that foetuses carried by mothers who have high levels of depression take longer to return to their baseline heart rates after exposure to the VAS[17]. The low response and oscillatory pattern in foetal heart rate (FHR) after the VAS that were observed in foetuses born to mothers with high anxiety levels in this study may be indicative of delayed neurodevelopmental maturation.

CONCLUSION

In conclusion, the findings of the current study indicate that changes in neurobehavioral functioning that are associated with inadequate mood regulation in the mother can be identified as early as the period when the mother is still in utero. When compared to other subjects, the FHR response to maternal challenge that is observed in the offspring of women with high anxiety may be a marker of altered neurobehavioral development related to a genetic predisposition. The effects of stress on FHR patterns after VAS in pregnant women who have high trait anxiety may be possible. It is also possible that the differences in FHR response patterns are associated with unmeasured variables, such as foetal movement. This hypothesis cannot be proven, but it is an intriguing possibility.

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