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The Effects of Parental History of Hypertension, Menstrual Cycle Phase, and Stressor Type on Cardiovascular Response

F. Aurelio Sita

A Thesis
in
The Department
of
Psychology

Presented in Partial Fulfilment of the Requirements for the Degree of Masters of Arts at Concordia University Montreal, Quebec, Canada

August 1992

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Abstract

The Effects of Parental History of Hypertension, Menstrual Cycle Phase, and Stressor Type on Cardiovascular Response

F. Aurelio Sita

The present study was carried out to examine whether cardiovascular response to stress in female offspring of hypertensives (PH+) was a function of menstrual cycle phase and stressor type. Thirty healthy women (18-35 years), half of whom were offspring of hypertensives, were tested in both the follicular and luteal phases of the menstrual cycle. Order of testing was counterbalanced within parental history groups. Cycle phase was confirmed by measurement of serum levels of estradiol and progesterone. In each session, subjects were exposed to four tasks: the cold pressor test, an interpersonal speech task involving real-life scenarios, a shock-avoidance video game task, and a reading task. Analyses indicated that PH+ women had higher heart rate responses to the video game task, but only during the luteal phase. PH+ women also had higher diastolic blood pressure responses and higher self-reported anger to the speech task, but again only in the luteal phase. Menstrual cycle effects on baseline heart rate, cardiac output, pre-ejection period, and diastolic blood pressure levels were also observed for the entire sample. In the luteal phase women had a higher resting heart rate and cardiac output, a shorter pre-
ejection period, and a lower diastolic blood pressure. Results suggest that previous inconsistent findings for elevated cardiovascular response in PH+ women may be due to a lack of control for menstrual cycle phase.
Acknowledgements

The author would like to thank Cathy Giannini, Rosetta Caruso, and Marianne Friese for their assistance in data collection and Dr. Reinhart B. Billiar, Ph.D., Director, Division of Research Laboratories, Obstetrics and Gynaecology, Royal Victoria Hospital for his collaboration in the analysis of sex hormone levels.
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The Effects of Parental History of Hypertension, Menstrual Cycle Phase, and Stressor Type on Cardiovascular Response

Hypertension, chronically elevated arterial pressure, is a major cause of heart failure, renal failure, and stroke, as well as an important risk factor for the development of atherosclerotic coronary artery disease (1984 Report of the Joint National Committee on the Detection, Evaluation, and Treatment of High Blood Pressure, 1984). According to the World Health Organization, hypertension is defined by a blood pressure greater than 160/95 mm Hg whereas borderline hypertension is defined as a systolic blood pressure between 140 and 160 or a diastolic blood pressure between 90 and 95 mm Hg. These cutoff points, though arbitrary, are based on values determined as necessitating treatment. In fact, however, even small elevations in blood pressure are associated with significant health risks (World Health Organization, 1983). In about 10 percent of cases hypertension is caused by known medical conditions (e.g. renovascular disease or coarctation of the aorta) and is termed secondary hypertension. In the vast majority of cases, however, its etiology remains unclear. Such hypertension is referred to as primary or essential hypertension. A number of etiological factors for essential hypertension have been proposed including genetic factors, psychological stress, and excessive salt intake.

Psychological stress has been hypothesized to play a
role in the development of hypertension. Early psychophysiological investigations demonstrated that mental challenges and emotionally arousing stimuli could elicit substantial increases in blood pressure, heart rate, and cardiac output (eg. Brod et al, 1959). Studies also showed that hypertensives demonstrated greater blood pressure and total peripheral resistance responsivity to laboratory stressors than normotensives (e.g. Stevenson et al, 1952; Schacter, 1957). This increased responsivity to stress in hypertensives, however, may be due to the presence of the disorder. Nevertheless, these early studies helped generate interest in the possible relationship between stress and hypertension.

Epidemiological studies have also pointed to a stress-related hypertension. Studies attempting to link occupational circumstances to the incidence of hypertension have found the prevalence of hypertension to be 2 to 4 times higher in air traffic controllers than in men of similar age in other occupations (Cobb & Rose, 1973). Additionally, amongst air traffic controllers, the age of onset for hypertension was earlier for those working in airports with high traffic density (Cobb & Rose, 1973). Epidemiological studies also suggest a positive relation between migration and elevation of blood pressure. A number of researchers in this area have hypothesized that the stress resulting from a breakdown in traditional values and norms brought about by
migration to more modern societies may, in some individuals, result in chronic autonomic arousal leading to an elevation in blood pressure (Henry & Cassel, 1969). Scotch (1963) found that rural Zulus who moved to urban communities had a higher prevalence of hypertension than Zulus still living in rural areas. Such epidemiological investigations are, however, replete with problems of confounding variables. For example, in addition to psychosocial factors, changes in diet and/or physical activity may often accompany migration to urban areas. Furthermore, correlations between migration and high blood pressure though significant are typically weak. In spite of the fact that these weaknesses exists, the idea that stress can contribute to the development of hypertension persists.

Given that blood pressure regulation depends on numerous intricate interactions between the circulatory, nervous, endocrine, and exocrine systems, it is unlikely that any single factor on its own can lead to hypertension. Researchers are currently of the belief that stress may induce hypertension only in individuals who are in some manner biologically and/or psychologically susceptible (Brody et al, 1987). One factor believed to raise susceptibility is a family history of the disorder. According to a recent study, hypertension before age 55 occurs 3.8 times more often among those individuals with two or more hypertensive first degree relatives (Hunt et al, 1986). Addi-
tionally, normotensive individuals with a parental history of hypertension (PH+), seem to exhibit enhanced cardiovascular responses to stress as compared to normotensive individuals without a parental history of hypertension (PH-) (Matthews and Rakaczky, 1986; Frederickson & Matthews, 1990). Thus, it may be that stress does play a role in essential hypertension, but only in those who are at higher risk for the disorder.

Although it is not clear whether the transient cardiovascular hyperreactivity to stress seen in PH+ individuals actually contributes to hypertension development, data from both animal and human research lend support for this notion. Much of the animal research has focused on the spontaneously hypertensive rat (SHR), a strain selectively bred for developing hypertension. SHRs, like PH+ individuals, exhibit increased heart rate and blood pressure reactivity to acute noxious stimuli before the onset of hypertension. A study by Hallbäck (1975) using SHRs demonstrated that aversive stimulation (i.e. a crowded environment) facilitated the development of hypertension whereas a low stimulation environment (i.e. social isolation) impeded it. Lawler et al. (1975) found that backcross offspring of SHRs eventually became hypertensive after being placed into a conflict situation and that the hypertension persisted when the rats were removed from the stressful situation. Rats not exposed to the stressful
situation did not develop hypertension. Furthermore, it was found in a longitudinal study with children of hypertensives that the blood pressure response to a ten minute laboratory task was a predictor of subsequent development of hypertension (Falkner, 1981).

Most of the studies examining the relation between parental history of hypertension and cardiovascular responses to stress have employed male subjects. This has been done, in part, due to the potential confound of sex differences in cardiovascular responsivity to stress. Men have been reported to exhibit greater resting systolic blood pressures and greater systolic blood pressure responses to laboratory stressors. However, women have been reported to have a higher resting heart rate and somewhat higher heart rates in response to stress (Stoney, Davis, & Matthews, 1987; Matthews & Stoney, 1988). Some studies have shown higher diastolic blood pressure responses to stress in men (Matthews & Stoney, 1988; Stoney et al, 1988), whereas others have found no sex differences (Stoney, Davis, & Matthews, 1987). There may also exist sex differences on less commonly measured cardiovascular variables. A study by Messerli et al, (1987) found hypertensive women to have a higher left ventricular ejection time and a lower total peripheral resistance than hypertensive men matched for age and mean arterial pressure. Therefore, it is unclear to what extent the results from studies involving male offspring of
hypertensives can be generalized to females. In fact, amongst the studies that have included women (eg. Anderson et al, 1986, 1989; Jorgensen & Houston, 1981; Lane et al, 1984; Lawler et al, 1991; Polefrone & Manuck, 1988; Stoney & Matthews, 1988), only a few have found offspring of hypertensives to be cardiovascularly hyperreactive as compared to offspring of normotensives (Jorgensen & Houston, 1981; Stoney & Matthews, 1988).

Inconsistent findings concerning cardiovascular hyperreactivity in female offspring of hypertensives may be related, in part, to a lack of control for menstrual cycle phase. Evidence suggests that menstrual cycle phase may influence cardiovascular activity at rest and in response to stress. A number of studies have reported a higher baseline heart rate during the luteal as compared to the follicular phase. (Little & Zahn, 1974; Polefrone & Manuck, 1988; Kaplan, Whitsett, & Robinson, 1990; Stoney, Langer, & Gelling, 1986). In addition, systolic blood pressure and heart rate responses to stress have been reported to be elevated in the luteal phase over those in the follicular phase (Hastrup & Light, 1984; Kaplan, Whitsett, & Robinson 1991; Tersman, Collins, & Eneroth, 1991). A number of studies, however, have either failed to find any effect of menstrual cycle phase on cardiovascular activity (Carroll, Turner, & Lee, 1984; Collins, Eneroth, & Landgren, 1985; Stoney et al, 1990) or have found elevated stress responses
during the follicular phase as opposed to the luteal phase (Polefrone & Manuck, 1988).

Three factors may account for these discrepancies. The equivocal results may be related to unreliable confirmation of the targeted menstrual cycle phase. In many cases, researchers have relied on subject self-report of last menstrual period to confirm cycle phase. This method, however, leads to inaccuracies due to intra-subject cycle length variability (Speroff, Glass, & Kase, 1983) and its inability to detect anovulatory cycles. Only a few studies have used sex hormone levels to ascertain menstrual cycle phase. There is also a lack of consistency amongst studies with respect to the time of testing during the luteal and follicular phases. For example, some studies have tested women during days 17-21 of the luteal phase (Hastrup & Light, 1984; Stoney, Langer, & Gelling, 1988), when estrogen and progesterone levels are rising, whereas others have chosen days 20-22 (e.g. Stoney et al 1990), when both hormones are at their peak; still others have targeted days 24-26 (Tersman, Collins, & Eneroth, 1990), when estrogen and progesterone levels are decreasing. Similarly, there is variability among studies with respect to testing during the follicular phase; in some studies women were tested during days 7-11 (e.g. Hastrup & Light, 1984), when estrogen levels are peaking; in others during days 5-7 (e.g. Tersman, Collins, & Eneroth, 1990), when estrogen levels are
beginning to rise. Finally, studies have also differed in design. Though the majority have employed a within subjects design, a few have opted for a between subjects approach, testing different women in different phases of the cycle. Both have drawbacks; whereas the between design fails to control for intra-subject variability across phases, the within design is subject to habituation effects. Together, these factors render it difficult to compare results from different studies, and may explain the discrepancies in this literature.

An additional reason for inconsistent findings in regards to cardiovascular response to stress in PH+ and PH- women may be the choice of laboratory stressors. In general, with the possible exception of the heart rate response, the majority of commonly employed psychological stressors (e.g. mental arithmetic, reaction time) appear to elicit less cardiovascular reactivity in women than in men (Matthews & Stoney, 1988; Stoney, Davis, & Matthews, 1987; Stoney & Matthews, 1988). It may be more difficult, therefore, to observe parental history differences in reactivity given the potentially smaller range of cardiovascular responses to these stressors in women. There are, however, stressors to which women respond similarly to men. Hastrup and Light (1984) have reported similar cardiovascular responses in women and men to the cold pressor test. Frankenhaeuser et al (1978) have investigated sex differences in neuroendocrine
response to stress using a number of stressors. They showed that in response to either venipuncture or a conflict word task males exhibited significant increases in norepinephrine, whereas women did not show increases to either task. Both men and women, however, showed significant increases in norepinephrine in response to a matriculation exam. The authors concluded that when faced with real-life stress situations, women and men may show similar neuroendocrine responses.

In the present study, the role of both menstrual cycle phase and stressor type on cardiovascular response to stress were examined in female offspring of hypertensives and normotensive parents. Self-reported anger and anxiety states were also examined. Women were tested in both the follicular and luteal phases, and serum estradiol and progesterone levels were obtained to accurately ascertain menstrual cycle phase. To the author's knowledge, the only previous study to control for menstrual cycle phase when examining parental history differences in cardiovascular response to stress, failed to find elevated responses in offspring of hypertensives (Polefrone & Manuck, 1988). However, in that study sex hormone levels were not used to confirm menstrual cycle phase. Because of the previous work showing increased responsiveness to stress in the luteal phase it was hypothesized that offspring of hypertensives would exhibit greater cardiovascular responses to stress.
than offspring of normotensives in the luteal as compared to the follicular phase of the menstrual cycle.

Subjects were exposed to four tasks, including the cold pressor test, an interpersonal speech task, a shock-avoidance video game task, and a neutral reading task. The interpersonal speech task was used because it involved scenarios of real-life situations. The cold-pressor test was chosen because women and men tend to respond similarly on this task. The shock-avoidance video game task has been used frequently in the laboratory with male subjects and was chosen because it has been found to elicit increased cardiovascular responses in PH+ men (Ditto & Miller, 1989; Miller & Ditto, 1991). A neutral reading task was used to control for the non-specific effects of speaking on blood pressure and heart rate (Lynch et al., 1981).
Method

Subjects

Forty-four women recruited from the Concordia University undergraduate student population completed the study. Subjects were between the ages of 18 and 35, were not taking any medication, and had not used oral contraceptives in the six months prior to participating in the study. All subjects were clearly normotensive with none exhibiting a resting blood pressure greater than 130/80 mm Hg. Calendar records of subjects' menstrual cycles for the previous 3 to 6 cycles were obtained for the purpose of subject scheduling. Participants had menstrual cycle lengths that were in the range of 24 to 34 days with a variability of five days or less. The hypertensive status of the parents was carefully verified by obtaining, from the parents, information concerning the history of the disorder, medication names and usage, and the frequency of physician contact. All of the parents stated that they visited their physician at least once a year. Examination of sex hormone levels revealed that fourteen women were not tested in one or both of the targeted phases. These women were consequently dropped from the study. The inclusion criteria employed were as follows: for the follicular phase, serum estradiol levels had to be between 20-120 pg/ml and progesterone levels had to be less than 0.5 ng/ml; for the
luteal phase, estradiol levels had to be between 40-300 pg/ml and progesterone levels had to be no less than 2 ng/ml.

Thus, thirty healthy females, 15 with at least one hypertensive parent (PH+), and 15 with no parental history of hypertension (PH−) comprised the final sample. The two groups did not differ significantly in age (PH+ 24.9 ± 5.6, PH− 21.2 ± 6.4 years), height (PH+ 1.62 ± 0.06, PH− 1.60 ± 0.06 meters), weight (PH+ 63.86 ± 9.97, PH− 58.97 ± 8.69 kg), or menstrual cycle length (PH+ 27.9 ± 2.32, PH− 29.0 ± 1.91 days).

Measures and Apparatus

Measurements of systolic (SBP) and diastolic blood pressure (DBP) (in mmHg) were obtained at one minute intervals using an IBS Model SD-700A automatic blood pressure monitor and a thigh cuff on the left leg. Blood pressure values were corrected for distance from heart level according to the manufacturer's recommendation. Heart rate (HR: in bpm), cardiac output (CO: in l/min), stroke volume (SV: in ml), and pre-ejection period (PEP: in msec) were recorded non-invasively by way of impedance cardiography using a Minnesota Impedance Cardiograph (Model 304B), the Cardiac Output Program (C.O.P.) developed by Bio-Impedance Technology Chapel Hill, North Carolina, an IBM AT computer
and a tetrapolar band-electrode configuration. The inner two recording electrode bands were positioned around the base of the neck and around the thorax over the tip of the xiphoid process. The outer two current electrode bands were positioned around the neck and thorax at least 3 cm away from each of the inner recording electrodes. The EKG was recorded using 2 recording spot electrodes placed on opposite sides of the rib cage at the level of the seventh rib approximately and a ground spot electrode placed on the right hip bone. The EKG signal was filtered through a Coulbourn bypass filter and then routed to the Minnesota Impedance Cardiograph. Recordings for these cardiac measures were obtained during the first 30 seconds of each minute. Ensemble averaged values of HR, CO, SV, and PEP were obtained from these recordings by the C.O.P for each minute.

Pre-ejection period was used as an index of myocardial sympathetic activity since, in addition to being highly sensitive to changes in myocardial beta-adrenergic activity, it is, unlike heart rate, relatively unaffected by parasympathetic activity (Obrist et al, 1987). Decreases in PEP have been found to occur concomitantly with the administration of beta-adrenergic as well as positive inotropic agents (Weissler, 1974).

Blood samples were centrifuged immediately after collection in a Damon centrifuge at 3000 rpm for 15 minutes. Serum samples were then stored at ~70 degrees Celsius. Serum
estradiol and progesterone levels were determined using a solid phase radioimmunoassay procedure. All assays were performed in duplicate.

Measures of self-report state anger and state anxiety were collected using a 7 point Likert scale with 0 being least angry (anxious), and 7 being most angry (anxious).

Procedure

Each subject participated in two 1-hour sessions, one during the follicular phase and the other during the luteal phase. Based on a 28 day cycle, testing took place between days 7-11 for the follicular phase and days 17-21 for the luteal phase. The testing range for each phase was adjusted according to the subject's average cycle length. Order of testing was counterbalanced between subjects. Subjects were asked to refrain from smoking and drinking caffeine for three hours prior to coming to the laboratory and not to drink alcohol for a 24 hour period preceding the experimental sessions.

At the beginning of each session the subject was comfortably seated in a semi-reclining chair, and connected to the cardiovascular recording equipment. The subject was then requested to sit quietly for 10 minutes. Following this rest period the subject engaged in four tasks in the presence of a female experimenter. The tasks were
administered in counterbalanced order and a ten minute rest period preceded each task. The tasks included:

Cold Pressor Task: For the cold pressor task the subject was required to immerse her right hand in 4 degrees Celsius water for 2 minutes. The subject was told to keep her hand motionless for the entire period.

Interpersonal Speech Task: For this task the subject was presented with one of two hypothetical conflict situations. In the first session the situation involved being falsely accused of shop-lifting. The second scenario involved being falsely accused of cheating on an exam. The subject was asked to imagine herself in the situation, and requested to prepare a defense for herself. The subject was given 3 minutes to prepare her scenario during which she had the opportunity to make notes. The subject was then given 3 minutes in which to deliver her speech. She was informed that it would be tape recorded and replayed to the laboratory staff in order to judge her on poise, articulation, style, and content.

Reading Task: The subject was required to read a 500 word article aloud for 3 minutes. In order to make this task analogous to the speech task, 3 minutes of preparation time were given to the subject during which she was asked to silently rehearse the article. During the first experimental session, the subject was presented with an article discussing margarine, whereas during the second session the
subject received an article on how paper is made. These articles were chosen because they were high in informative content but low in affective content.

Shock-Avoidance Video Game Task: The subject engaged in a 3 minute shock-avoidance procedure, with avoidance made contingent on video game performance. Prior to the beginning of the task a shock electrode was placed on the ulnar side of the dominant elbow. To minimize extraneous motor activity, the subject's dominant arm was also lightly tied at the wrist to the arm of the chair. The video game employed was auto racing (Enduro by Atari). Before beginning play, the subject was informed of the score necessary to avoid the possibility of receiving an electric shock. The subject was told that upon failure to meet the criterion a computer would randomly determine whether or not she would receive a shock. In fact, no shock was ever administered. The criterion score was selected to reflect performance that was difficult, but not impossible, for subjects with this background (i.e. undergraduate university students) to attain. Similar criteria previously employed in this laboratory resulted in performance levels in which subjects experienced neither total failure nor total success on the shock avoidance task (Miller & Ditto, 1989; Miller & Ditto, 1991). To deal with practice effects, the criterion score was adjusted for the second session according to the subject's prior performance.
Midway through each task the subject was asked to rate her level of state-anger and anxiety. At the end of each session an 8ml blood sample was taken. Upon completion of the second session the subject was debriefed and paid for her participation.

**Data Reduction**

Cardiovascular data were collected and reduced in the following manner. Data were collected from minutes 6 to 8 of the 10-minute rest periods preceding each task, and for the full length of time of each stressor task. For each subject, a mean value was then calculated for each rest and task period for each cardiovascular measure. The mean values for each rest period served as baseline comparisons. Analyses of cardiovascular and affective responses to the tasks were carried out on baseline - stress change scores. These change scores were obtained by subtracting the task mean and the mean of the immediately preceding baseline for each measure.
Results

Estradiol and Progesterone Levels

Means and standard errors of serum estradiol and progesterone levels for each menstrual cycle phase and parental history group are presented in Table 1. The observed means are within the expected ranges for each menstrual cycle phase (see Speroff, Glass, & Kase, 1983). Separate 2 (PH+ vs PH-) x 2 (follicular vs luteal) repeated measures ANOVAs were conducted for each sex hormone. The ANOVAs revealed a significant main effect of menstrual cycle phase on estradiol ($F(1, 28)=10.12, p<.01$), and progesterone ($F(1, 28)=24.38, p<.01$). Subjects had higher levels of serum estradiol and progesterone during the luteal phase than during the follicular phase. No other significant main effects or interactions were observed.

Speech vs reading task comparison

The purpose of the reading task was to ensure that cardiovascular changes observed during the speech task could not be accounted for simply by the influence of speaking. Therefore, speech and reading task change scores were compared to each other for each cardiovascular measure. Separate 2 (follicular vs luteal) x 2 (speech vs reading) repeated measures ANCOVAs on baseline - stress change scores using the scores for the rest periods preceding each task as
Table 1

Means and standard errors of serum estradiol and progesterone levels by parental history and menstrual cycle phase

<table>
<thead>
<tr>
<th>Variables</th>
<th>Estradiol (pg/ml)</th>
<th>Progesterone (ng/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Follicular</td>
<td>93.2 (9.9)</td>
<td>0.28 (0.05)</td>
</tr>
<tr>
<td>PH+</td>
<td>101.3 (16.6)</td>
<td>0.23 (0.05)</td>
</tr>
<tr>
<td>PH-</td>
<td>85.1 (11.0)</td>
<td>0.34 (0.09)</td>
</tr>
<tr>
<td>Luteal</td>
<td>137.9 (11.0)</td>
<td>5.25 (0.92)</td>
</tr>
<tr>
<td>PH+</td>
<td>125.6 (11.1)</td>
<td>4.99 (1.68)</td>
</tr>
<tr>
<td>PH-</td>
<td>146.9 (17.3)</td>
<td>5.45 (1.26)</td>
</tr>
</tbody>
</table>
covariates were conducted for each cardiovascular measure. The ANCOVAs revealed that, with the exception of stroke volume, the speech task elicited greater cardiovascular responses than the reading task during each menstrual cycle phase (see Table 2). Therefore, the cardiovascular responses elicited by the speech task cannot be explained merely by the act of speaking. Because the reading task served solely as a control for the speech task, the data from this task and the rest period preceding it were not subjected to further analyses.

Baseline

2 x 2 x ... repeated measures ANOVAs for parental history (PH+ vs PH−), menstrual cycle phase (follicular vs luteal), and task (cold pressor vs video game vs speech task) were conducted on the baseline scores for each cardiovascular and affective measure. Means and standard errors of the resting cardiovascular measures by menstrual cycle phase and task are presented in Table 3.

The ANOVAs carried out on the baseline measures revealed a significant main effect of menstrual cycle phase on cardiac output ($F(1,28)=7.55$, $p<.01$), heart rate ($F(1,28)=9.83$, $p<.01$), PEP ($F(1,28)=13.13$, $p<.01$), and diastolic blood pressure ($F(1,28)=5.02$, $p<.05$). It can be seen that women exhibited higher cardiac outputs and heart
Table 2

Means and standard errors of cardiovascular change scores for speech and reading tasks by menstrual cycle phase

<table>
<thead>
<tr>
<th>Variables</th>
<th>HR (bpm)</th>
<th>PEP (msec)</th>
<th>CO (l/min)</th>
<th>SV (ml)</th>
<th>SBP (mmHg)</th>
<th>DBP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Follicular</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading</td>
<td>9.2(1.5)</td>
<td>-0.7(1.5)</td>
<td>0.29(0.11)</td>
<td>-4.8(1.4)</td>
<td>5.9(0.9)</td>
<td>4.9(1.2)</td>
</tr>
<tr>
<td>Speech</td>
<td>19.2(2.2)</td>
<td>-6.9(1.4)</td>
<td>0.92(0.18)</td>
<td>-5.9(1.9)</td>
<td>11.7(1.4)</td>
<td>9.7(1.4)</td>
</tr>
<tr>
<td><strong>Luteal</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading</td>
<td>8.1(1.6)</td>
<td>-2.6(1.2)</td>
<td>0.23(0.12)</td>
<td>-6.1(1.3)</td>
<td>5.5(1.2)</td>
<td>4.3(1.5)</td>
</tr>
<tr>
<td>Speech</td>
<td>19.9(2.2)</td>
<td>-3.3(1.4)</td>
<td>0.76(0.18)</td>
<td>-8.8(1.7)</td>
<td>11.4(1.1)</td>
<td>7.8(1.6)</td>
</tr>
</tbody>
</table>
rates, shorter PEPs, and lower diastolic blood pressures
during the luteal than the follicular phase (see Table 3).
No other significant phase effects or parental history
effects were observed. For all measures the three baselines
were not significantly different from one another.

Cardiovascular Responses to Stress

To examine the effect of parental history, menstrual
cycle phase, and task on cardiovascular responses and self-
reported affective states, 2(PH+ vs PH-) x 2(follicular vs
luteal) x 3(cold pressor vs video game vs speech task)
repeated measures ANCOVAs were conducted on the baseline –
stress change scores for each of these measures using the
respective baseline values as covariates. In order to
address the problem of homogeneity of covariance,
significance levels were determined using Greenhouse-Geisser
corrected degrees of freedom where appropriate (Greenhouse &
Geisser, 1959). For post-hoc comparisons, tests of simple
main effects were conducted using the appropriate pooled
error terms suggested by Winer (1971). Means and standard
errors of the task cardiovascular and affective measures by
menstrual cycle phase and task are presented in Table 4.
Table 3

Means and standard errors of cardiovascular and affective baseline scores by menstrual cycle phase

<table>
<thead>
<tr>
<th>Variables</th>
<th>HR (bpm)</th>
<th>PEP (msec)</th>
<th>CO (l/min)</th>
<th>SV (ml)</th>
<th>SBP (mmHg)</th>
<th>DBP (mmHg)</th>
<th>ANXIETY (units)</th>
<th>ANGER (units)</th>
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<tr>
<td>Baseline</td>
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<tr>
<td>Follicular</td>
<td>64.8(1.8)</td>
<td>108.6(3.0)</td>
<td>6.9(0.3)</td>
<td>108.2(5.7)</td>
<td>111.4(2.8)</td>
<td>64.8(2.0)</td>
<td>2.7(0.4)</td>
<td>1.7(0.3)</td>
</tr>
<tr>
<td>Luteal</td>
<td>69.1(2.0)</td>
<td>103.0(3.5)</td>
<td>7.6(0.4)</td>
<td>110.8(5.2)</td>
<td>110.2(2.9)</td>
<td>62.6(2.2)</td>
<td>3.1(0.4)</td>
<td>1.9(0.3)</td>
</tr>
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</table>
Table 4

Means and standard errors of cardiovascular and affective change scores by parental history, menstrual cycle phase, and task

<table>
<thead>
<tr>
<th>Variables</th>
<th>HR (bpm)</th>
<th>PEP (msec)</th>
<th>CO (l/min)</th>
<th>SV (ml)</th>
<th>SBP (mmHg)</th>
<th>DBP (mmHg)</th>
<th>ANXIETY (units)</th>
<th>ANGER (units)</th>
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<tr>
<td><strong>Cold Pressor</strong></td>
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<tr>
<td><strong>Follicular</strong></td>
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</tr>
<tr>
<td>PH+</td>
<td>3.6(1.7)</td>
<td>3.9(2.6)</td>
<td>-0.22(0.18)</td>
<td>-9.1(2.6)</td>
<td>15.4(2.4)</td>
<td>14.1(2.6)</td>
<td>2.8(0.5)</td>
<td>2.4(0.6)</td>
</tr>
<tr>
<td>PH-</td>
<td>4.1(1.2)</td>
<td>0.7(1.7)</td>
<td>-0.14(0.10)</td>
<td>-7.6(1.9)</td>
<td>14.7(2.2)</td>
<td>14.3(2.7)</td>
<td>1.7(0.5)</td>
<td>1.3(0.7)</td>
</tr>
<tr>
<td>Luteal</td>
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<tr>
<td>PH+</td>
<td>3.2(1.1)</td>
<td>0.7(1.9)</td>
<td>-0.30(0.26)</td>
<td>-8.7(2.4)</td>
<td>15.1(1.7)</td>
<td>17.8(2.6)</td>
<td>2.3(0.6)</td>
<td>2.0(0.6)</td>
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<tr>
<td>PH-</td>
<td>5.3(1.6)</td>
<td>0.7(1.8)</td>
<td>-0.05(0.20)</td>
<td>-9.7(2.7)</td>
<td>14.1(2.1)</td>
<td>18.1(3.0)</td>
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<td><strong>Speech</strong></td>
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<tr>
<td><strong>Follicular</strong></td>
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<tr>
<td>PH+</td>
<td>12.5(2.1)</td>
<td>-4.7(2.0)</td>
<td>0.89(0.23)</td>
<td>-7.1(3.2)</td>
<td>12.6(2.4)</td>
<td>8.7(2.4)</td>
<td>1.0(0.5)</td>
<td>1.0(0.3)</td>
</tr>
<tr>
<td>PH-</td>
<td>11.7(1.5)</td>
<td>-9.1(1.7)</td>
<td>0.96(0.27)</td>
<td>-4.9(2.2)</td>
<td>12.8(1.6)</td>
<td>10.7(1.6)</td>
<td>1.5(0.2)</td>
<td>0.8(0.3)</td>
</tr>
<tr>
<td>Luteal</td>
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<tr>
<td>PH+</td>
<td>12.9(2.3)</td>
<td>-2.6(1.5)</td>
<td>0.85(0.31)</td>
<td>-7.3(2.9)</td>
<td>10.7(2.1)</td>
<td>9.1(2.3)</td>
<td>1.5(0.4)</td>
<td>1.5(0.4)</td>
</tr>
<tr>
<td>PH-</td>
<td>14.1(1.9)</td>
<td>-4.0(2.4)</td>
<td>0.67(0.19)</td>
<td>-10.1(2.6)</td>
<td>14.4(1.5)</td>
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<td>0.1(0.3)</td>
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<tr>
<td><strong>Video Game</strong></td>
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<tr>
<td><strong>Follicular</strong></td>
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</tr>
<tr>
<td>PH+</td>
<td>9.4(1.3)</td>
<td>-7.4(3.2)</td>
<td>0.65(0.16)</td>
<td>-5.2(2.2)</td>
<td>12.5(1.7)</td>
<td>10.5(3.3)</td>
<td>2.9(0.5)</td>
<td>0.7(0.2)</td>
</tr>
<tr>
<td>PH-</td>
<td>11.9(1.3)</td>
<td>-7.4(1.3)</td>
<td>0.74(0.21)</td>
<td>-8.2(2.3)</td>
<td>11.6(1.8)</td>
<td>9.3(1.8)</td>
<td>2.5(0.5)</td>
<td>1.0(0.3)</td>
</tr>
<tr>
<td>Luteal</td>
<td></td>
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</tr>
<tr>
<td>PH+</td>
<td>13.0(2.1)</td>
<td>-5.9(1.3)</td>
<td>0.85(0.31)</td>
<td>-7.9(1.8)</td>
<td>11.8(1.6)</td>
<td>8.5(2.6)</td>
<td>2.3(0.6)</td>
<td>0.3(0.3)</td>
</tr>
<tr>
<td>PH-</td>
<td>9.1(1.1)</td>
<td>-4.4(1.5)</td>
<td>0.67(0.19)</td>
<td>-5.4(2.6)</td>
<td>10.2(1.4)</td>
<td>11.9(2.3)</td>
<td>1.7(0.5)</td>
<td>0.3(0.4)</td>
</tr>
</tbody>
</table>
Heart rate: The main effect of Task ($F(2,55)=22.61$, $p < .01$) and the Parental History X Phase X Task interaction were significant ($F(2,55)=5.40$, $p<.01$). Analyses of simple main effects revealed that, in response to the video game task, PH+ women exhibited higher elevations of heart rate as compared to PH- women, but only during the luteal phase (see Figure 1).

**Figure 1.** Heart rate baseline-stress change score responses (adjusted for baseline values) to the video game task for offspring of hypertensives (PH+) and offspring of normotensives (PH-) as a function of menstrual cycle phase.
PEP: The ANOVA for PEP revealed a significant effect of Task ($F(2,55)=25.46, p < .01$). Comparisons revealed that subjects had shorter PEP responses during the video game and speech tasks than during the cold pressor task. Effects involving parental history and phase were not significant.

Cardiac output: A significant effect of Task ($F(2,55)=21.69, p < .01$) was observed. Cardiac output was higher during the speech and video game tasks as compared to the cold pressor task. Responses to the speech and video game did not differ. No effects involving parental history or phase were observed.

Stroke volume: No significant main or interaction effects of stroke volume were observed. However, the Parental History X Phase X Task interaction approached significance ($F(2,55)=2.32, p < .1$). In the luteal phase PH+ women tended to have higher stroke volume responses to the speech task than PH- women.

Diastolic blood pressure: The ANCOVA revealed a significant main effect of Task ($F(2,55)=10.81, p < .01$), a significant Phase by Task interaction ($F(2,55)=4.39, p < .05$) and a significant Parental History X Phase X Task interaction ($F(2,55)=3.96, p < .05$). Analyses of simple main effects revealed that PH+ women exhibited higher diastolic
blood pressure responses than PH- women to the speech task during the luteal phase, but not during the follicular phase (see Figure 2).

**Figure 2.** Diastolic blood pressure baseline-stress change score responses (adjusted for baseline values) to the speech task for offspring of hypertensives (PH+) and offspring of normotensives (PH-) as a function of menstrual cycle phase.
Systolic blood pressure: No significant main effects or interactions were observed for systolic blood pressure.

Self-Reported Affective States

State-anger: The ANCOVA revealed a significant Task main effect \( F(2,55) = 5.74, p < .01 \) and a significant PH x Phase x Task interaction \( F(2,55) = 5.72, p < .01 \). As shown in Figure 3, the speech task elicited greater increases in state-anger ratings in PH+ women than in PH- women, but only during the luteal phase. For the speech task, PH- women had lower increases in state-anger ratings during the luteal phase than in the follicular phase, whereas the state-anger change scores of the PH+ women were not significantly different in the two phases.

State-anxiety: No significant main effects or interactions were observed for state anxiety.
Figure 3. State anger baseline-stress change score responses (adjusted for baseline values) to the speech task for offspring of hypertensives (PH+) and offspring of normotensives (PH-) as a function of menstrual cycle phase.
Discussion

The results of the present study indicate that parental history differences in cardiovascular responses to stress in women can be observed when menstrual cycle phase is clearly established. As hypothesized, PH+ women exhibited greater cardiovascular responses to stress than PH- women in the luteal phase, whereas in the follicular phase, no significant differences were found. During the luteal phase, PH+ women had higher heart rate responses than PH- women to the video game task. A parental history difference in heart rate reactivity to the video game task has also been observed in previous studies from this laboratory using males (Miller & Ditto, 1989; 1991). Similar to the present study, these studies did not observe parental history differences in blood pressure responses to this task. The parental history difference in heart rate observed only in the luteal phase can be compared to that found in a study by Hastrup and Light (1984). They found that women tested on a mental arithmetic task exhibited heart rate responses similar to those of men during the luteal phase, but during the follicular phase women showed lower heart rate responses than men. PH+ women also displayed significantly higher diastolic blood pressure responses and larger increases in state-anger ratings to the speech task than PH- women during this phase. Interestingly, the patterns of response for

30
diastolic blood pressure and state-anger were very similar. In both cases parental differences during the luteal phase were the result of sustained elevations in the responses of PH+ women across both phases, whereas PH- women exhibited a decreased response in the luteal as compared to the follicular phase.

Contrary to expectation, no parental history differences were observed during the cold pressor task. It had been proposed that, because women may be as reactive to the cold pressor as men, parental history differences would likely be found in response to this task. However, although the cold pressor task has been found by some to predict the subsequent development of hypertension (Hines & Brown, 1936; Wood et al, 1984), it has for the most part been unable to elicit cardiovascular differences between PH+ and PH- men (e.g. Hastrup et al, 1982).

Differences in baseline values across menstrual cycle phase were also observed for the entire sample. At rest, women displayed a higher heart rate and cardiac output, and a lower PEP and diastolic blood pressure in the luteal phase as compared to the follicular phase. It could be argued, however, that these menstrual cycle phase effects were due to order effects since order of testing with respect to menstrual cycle phase was not fully counterbalanced. More specifically, in the entire sample there were two more women tested first in the luteal phase than in the follicular
phase. Nonetheless, except for the baseline diastolic blood pressure values, the baseline differences in this study are consistent with results from other studies that have found elevated resting heart rate (Little & Zahn, 1974; Polefrone & Manuck, 1988; Kaplan, Whitsett, & Robinson, 1990; Stoney, Langer, & Gelling, 1986) and shorter PEP (Re et al, 1986) in the luteal phase. Furthermore, in terms of the diastolic blood pressure result, if it had been due to an order effect, one would have expected to find a higher diastolic blood pressure upon first testing (i.e. in the luteal phase).

To the author's knowledge, this is the first study to observe that the effect of a parental history of hypertension on women's cardiovascular stress responses varies as a function of menstrual cycle phase. The only other group to examine the effects of parental history of hypertension and menstrual cycle phase on women's cardiovascular responses to stress found neither an interaction between parental history and menstrual cycle phase nor any significant effects with respect to parental history (Polefrone & Manuck, 1988). Methodological differences between the two studies may explain these inconsistent findings. In the present study the range of average cycle lengths for the entire sample varied from 25 to 34 days. Based on subsequent analyses of serum estradiol and progesterone levels, 14 of 44 women were found not to
have been tested in the appropriate phase and had to be removed from the analyses (see Method section for criteria). This, despite the fact that women were monitored for at least three consecutive cycles before participating in the study and only women with cycles of 5 days variability or less were tested. In the Polefrone & Manuck study, subjects had a wider range of average cycle lengths (i.e. 22 to 46 days) and verification of menstrual cycle phase was limited to counting the number of days between consecutive menstrual periods. This method, however, does not control for the occurrence of anovulatory cycles which, depending on the nature of the sample, can occur in 2 to 25 percent of cycles (Friedman & Meares, 1979; Marshall 1963). Thus, their failure to find any cardiovascular differences between PH+ and PH- women may, in part, be due to incorrect targeting of menstrual cycle phase. Another potential limitation of the Polefrone & Manuck study was their use of a between-subjects design in which different women were tested in the follicular and luteal phases. The present study tested the same women in both phases, thus affording better control of individual differences in hormonal levels and reactivity.

It would be interesting to speculate on the mechanisms underlying the parental history and menstrual cycle differences observed in this study. The higher heart rate response to the video game task seen in PH+ women during the luteal phase may be due to increased sympathetic beta-
adrenergic activity in this phase. The shock-avoidance video game task has previously been shown to elicit a higher heart rate response in PH+ males than in PH- males (Miller & Ditto, 1989; 1991), but not during blockade with metoprolol, a beta-adrenergic antagonist (Miller & Ditto, 1991). Increased sympathetic beta-adrenergic reactivity to the video game task in the luteal phase is also consistent with results suggesting urinary epinephrine output during stress to be higher in the luteal phase than in the follicular phase (Collins, Eneroeth, & Landgren, 1985). The reason why PEP responses to the video game task were not significantly different during the two phases is, however, unclear given that PEP has been found to be sensitive to changes in myocardial beta-adrenergic activity (Weissler, 1974). In addition, estrogen and progesterone may be exerting a direct effect on the cardiovascular system. In the rat, for example, the myocardium has been found to contain receptors for estradiol (Stumpf, Sar, & Aumüller, 1977). Furthermore, in the guinea pig brain and spinal cord, nuclear receptors for progesterone have been localized in regions associated with cardiovascular regulation (Stumpf, 1990). The higher resting heart rate and cardiac output and decreased resting PEP observed in the luteal phase may similarly be due to increased sympathetic beta-adrenergic activity on the heart.

The lower luteal phase diastolic blood pressure response to the speech task seen in PH- women is consistent
with the findings of von Eiff et al (1971) who observed a significant negative correlation between progesterone levels and diastolic blood pressure during a mental arithmetic task. One way progesterone may lower diastolic blood pressure is by lowering blood volume via its natriuretic properties and inhibition of aldosterone induced salt reabsorption (Oparil et al, 1975). That a decrease in blood volume may, in part, underlie the lower luteal phase diastolic blood pressure levels observed in PH- women is supported by the present study's results indicating a trend for stroke volume and cardiac output to be lower during the luteal phase in this group of women. Alternatively, the decreased diastolic blood pressure response in PH- women to the speech task may be due to reduced peripheral vascular tone in the luteal phase since both estrogen and progesterone may inhibit vascular contraction (Eccles & Leathard, 1984; Sarrel, 1990). A decrease in blood volume and/or inhibition of vascular contraction may also underlie the lower luteal phase diastolic blood pressure observed in both groups of women at rest.

Of greater interest is that, unlike the PH- women, the PH+ women did not exhibit a decreased diastolic blood pressure response to the speech task during the luteal phase. Two possible mechanisms, both of which implicate elevated sympathetic nervous system activity in PH+ women during the luteal phase, may underlie this result. It is
possible that increased renal sympathetic activity in PH+ women during the luteal phase offset progesterone induced salt excretion, which in turn, impeded a decrease in blood volume. The fact that cardiac output and stroke volume remained unchanged in PH+ women across the two phases suggests that this group of women did not experience a decrease in blood volume during the luteal phase. A decreased propensity to excrete salt when stressed has been observed in PH+ males (Light et al., 1983) and pre-hypertensive SHRs (Lundin & Thoren, 1982; Koepke & DiBona, 1985). Increased renal sympathetic activity may underlie the decrease in salt excretion as suggested by studies showing 1) increased renal sympathetic activity in pre-hypertensive SHRs (Lundin & Thoren, 1982) and 2) near elimination of the decreased sodium excretion in pre-hypertensive SHRs during stress by renal denervation (Lundin & Thoren, 1982; Koepke & DiBona, 1985).

Additionally, it is possible that increased alpha-adrenergic activity under stress in PH+ women during the luteal phase impeded an attenuation in peripheral vascular tone. Increased alpha-adrenergic activity has been linked to increased vascular resistance in PH+ males during stress (Miller & Ditt, 1991). Both of these hypotheses, that of decreased salt excretion and that of attenuated reduction in peripheral vascular tone in PH+ women during the luteal phase, are consistent with increased sympathetic nervous
system activity in PH+ women during the luteal phase
overriding a decrease in diastolic blood pressure during
this phase.

The parental history differences in diastolic blood
pressure response to the speech task may also be related to
the group differences in state-anger ratings. Anger could
have played a role in the group differences in diastolic
blood pressure responses by increasing the sympathetic
response during the luteal phase for PH+ as compared to PH-
women. Research has suggested a relationship between anger
and increased cardiovascular reactivity to stress in PH+
males (Johnson, 1989; Manuck, Proietti, Rader, & Polefrone,
1985). Furthermore, anger has been found to elicit greater
increases in diastolic blood pressure than other affective
states (Ax, 1953; Schwartz et al, 1981). It is unclear,
however, why PH- women had lower increases in state-anger
ratings to this task during the luteal phase as compared to
the follicular phase. Studies investigating affective
changes during the menstrual cycle have yielded equivocal
results, however, those that have found menstrual cycle
changes in affect usually report increased as opposed to
decreased negative affect and irritability during the luteal
phase (Dennerstein & Abraham, 1982).

Given the speculations about the parental history
differences in diastolic blood pressure and anger ratings to
the speech task, future research should examine parental
history differences in renal function and peripheral vascular activity during the luteal phase under affectively neutral and anger-provoking tasks. In addition to the cardiovascular variables recorded in the present study, measures such as sodium excretion and blood volume responses to these tasks could be investigated during the luteal phase in these two groups. If the above speculations are correct, one may be more likely to observe group differences in diastolic blood pressure and renal function in the luteal phase under an anger-provoking task. Additionally, a study examining the effects of beta-adrenergic blockers on cardiovascular responses to stress in PH+ and PH- women during the luteal phase would help elucidate whether the parental history difference in heart rate was due to a difference in myocardial beta-adrenergic activity. If the parental history difference in heart rate was due to increased beta-adrenergic activity in PH+ women, the heart rate responses of PH+ and PH- women to the video game would be expected to differ under placebo, but not under beta-adrenergic blockade.

In conclusion, the major findings of the present study suggest that menstrual cycle phase does affect cardiovascular activity and that previous negative findings with respect to parental history effects on cardiovascular reactivity in women may have been due to a failure to control for menstrual cycle phase. Based on the results of
the present study, future investigations of the effects of parental history on cardiovascular responses in women should consider menstrual cycle phase.
References


von Eiff, A.W., Plotz, E.J., Beck, K.J., & Czernik, A.
Appendix A

INFORMED CONSENT FORM

RESEARCH STUDY CONDUCTED AT CONCORDIA UNIVERSITY, DEPARTMENT OF PSYCHOLOGY ON BEHALF OF DR. SYDNEY MILLER

We would like you to participate in a study investigating the possible effects of menstrual cycle phase on cardiovascular reactivity to stress. In this study, changes such as increases in heart rate and blood pressure will occur. These increases will be only temporary, returning to normal after the experiment and causing no adverse effects.

Your participation in the study will require you to come for two sessions, each lasting approximately one and a half hour. During each session you will engage in four different tasks. One of these tasks will consist of playing a challenging video game for 3 minutes. Your score on this task will be compared to a criterion level of which you will be informed prior to playing. If you score below the criterion level, there is a possibility that you will receive a mild electric shock upon completion of the task. This shock is harmless and produces no more discomfort than what is occasionally experienced in normal every day life. In another task you will be required to immerse your hand in cold (approximately 4°C) water for 3 minutes. There will also be two speech tasks. For one of these tasks you will be asked to read an article aloud for 3 minutes. You will be given 3 minutes of preparation time during which you can look at the article. For the other speech task you will be presented with a hypothetical situation around which you will have to construct a scenario. You will be given 3 minutes to prepare your scenario and 3 minutes to tell your story.

During these tasks we will obtain various physiological measures (heart rate, blood pressure, forearm blood flow, cardiac output). These physiological recordings are safe, painless, and non-invasive (no needles are involved) and only require the placement of various transducers on the skin.

At the end of each session we will take a 5 millilitre blood sample from your arm. The blood sample is needed in order to verify the targeted menstrual cycle phase. In between the two sessions you will be asked to fill out several questionnaires and to return them at the second session. You will be paid $40 for your participation at the end of the second session. Payment will be contingent upon
completion of the two sessions and the return of the completed questionnaires.

All the information we obtain about you is completely confidential, and will not be seen by anyone who is not a member of the research team. Ultimately, all data will be coded using a subject number rather than your name. You are free to withdraw from the experiment at any time.

Once you have carefully studied and understood this form, you may sign it in indication of your free consent and agreement to participate in the study.

NAME (please print):

........................................

SIGNATURE:

........................................

WITNESS SIGNATURE:

........................................

DATE:

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Appendix B

Speech Task

1. Imagine that you are in a crowded department store during the Christmas season. Just as you are about to leave the store, a plainclothes detective grabs your arm and arrests you for shop-lifting.

   Prepare and deliver a speech detailing the emotions you would feel, what you would say in your defense, how the detective would react to your story and what the outcome will be.

   N.B. Your speech will be tape-recorded and then judged by the laboratory staff on poise, articulation, style and content.

2. Imagine that you are writing a final exam in your final year of your undergraduate program. The room in which you are writing this exam is small and crowded. Midway through the exam, an invigilator comes to your seat, takes your exam away from you and asks you to follow him. Once outside the room, he accuses you of cheating.

   Prepare and deliver a speech detailing the emotions you would feel, what you would say, how the invigilator would react to your story and what the outcome would be.

   N.B. Your speech will be tape-recorded and then judged by the laboratory staff on poise, articulation, style and content.
Appendix C

Reading Task

1. Margarine is a butterlike food product made from vegetable oils or animal fats, or both. Many people cook with margarine and use it on bread and other foods. Some bakeries also use it.

In the United States, people use more than twice as much margarine as butter. Margarine usually costs less than butter, and it can be processed so that it has the same food value. Margarine also contains much less of a fatty substance called cholesterol. Many doctors warn that too much cholesterol in the blood can lead to a heart attack. As a result, large numbers of people eat margarine and other low-cholesterol foods.

The U.S. government requires that margarine contain at least 80% fat. The fat must be emulsified in milk, water, or a type of milk made from soybeans. Manufacturers add preservatives to prevent decay. Most margarine also contains butterlike flavouring, salt, vitamins A and D, and yellow colouring. One or more vegetable oils provide the fat content of most margarine. Soybean oil is by far the most commonly used oil. But processors also may use corn, cottonseed, palm, peanut, and safflower oil. Some margarine is produced with animal fats.

To make margarine, manufacturers emulsify melted oils with milk or water and chill the resulting substance until it hardens. In modern margarine plants, machines produce the product and then shape, wrap, and package it in one continuous operation. Most household margarine is made in sticks or is packaged in small tubs. Manufacturers also make large blocks of hard margarine for commercial bakeries, and fluid household margarine in plastic squeeze bottles.

Butter on the other hand comes from butterfat, which is present in milk and cream in the form of tiny droplets. Butter is churned from cream because cream contains about 10 times as much butterfat as milk does. When cream is mixed rapidly at a certain temperature, droplets of butterfat form particles called butter granules. Churning turns these particles into butter. Creameries make butter in a process that has three steps: (1) pasteurization, (2) churning, and (3) packaging.

Butter gets its food value primarily from the butterfat it contains. Standards set by the United States Department of Agriculture require that butter consist of at least 80 per cent butterfat. Most butter averages about 80.5 per cent butterfat. Butter contains much cholesterol, a fatty substance that makes up a part of all animal tissue. Some scientists believe too much cholesterol in the blood may
cause arteriosclerosis in human beings. This disease can lead to a heart attack. Many physicians advise people with a high level of cholesterol to avoid butter and other foods rich in cholesterol.

2. Paper is often called the handmaiden of civilization. It ranks as one of the most valuable materials in the recording and spreading of information and knowledge. Books, magazines and newspapers are printed on paper, and education, government, and industry could not operate without it.

Paper consumption per person is often considered a reliable index to the standard of living. The higher the standard of living and the greater the national wealth, the greater the amount of paper used. The United States consumes about 640 pounds (290 kilograms) of paper and paperboard per person every year. It produces about 62 million short tons (56 million metric tons) of paper and paperboard each year.

Chemical engineers have found many ways of treating paper to make it strong, fireproof, and resistant to liquids and acids. As a result, it can replace such materials as cloth, metal, and wood. For example, specially treated paper is used to make clothing, including disposable diapers and surgical gowns.

All paper is formed into sheets from cellulose fibers. Cellulose is a substance that is found in most plants. Plants that are especially used for papermaking include various kinds of trees, cotton plants, rice and wheat straws, cornstalks, hemp, jute, and esparto. About 75 percent of the paper produced in the United States comes from wood pulp obtained from trees and waste materials of lumbering operations. The remainder is made chiefly from pulp recycled from waste paper.

There are about 7,000 kinds of paper. The type of finished paper depends entirely on the manufacturing and chemical processes that it has passed through. For centuries, rags were the principal raw material for paper. Today, they have been largely replaced by wood pulp. But rug paper is still used for most high-grade writing paper and for documents that must be kept for many years.

Wood pulp comes from fir, hemlock, pine, poplar, spruce, tamarack, and from hardwood trees. Canada and the United States supply most of this wood. Canada produces more than a sixth of the world's pulp. Europe produces most paper pulp coming from esparto, hemp, and straw.