A Neuropsychological Investigation of Sex Differences in Cardiovascular Reactivity to Verbal and Spatial Fluency Tasks: Testing a New Model of Sex Differences in Cardiovascular Regulation and Disease

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(ABSTRACT)

One hundred twenty-six right-handed undergraduate men and women underwent physiological measurements of SBP, DBP, and HR before and after verbal and figural fluency tasks, used as stressors. Dynamic and functional cerebral regulation of cardiovascular reactivity was assessed, specifically, the role that the frontal lobes have in regulating SBP, DBP, and HR in men and women. Sex differences in the functional cerebral regulation of these cardiovascular factors were predicted. Hostility was assessed in these participants, using the Cook-Medley Hostility Inventory (6 total groups of 21 participants each: high-, mid-, and low-hostile participants were identified). Sex and group (hostility) differences were predicted, as well as task (fluency type) differences. Comparisons were also made from a time estimation task (30 and 180 seconds), and the effect that women's menstrual cycle had on fluency. The MCSDS and the STAI were administered.

The principal findings of the current investigation were that the verbal fluency task raised SBP across sex and group, that both stressors raised SBP or DBP in different patterns (no sex differences were found), while stressors interacted with both sex and group. High-hostile men performed better on the first trial of the verbal fluency test compared to low-hostile men, while high-hostile women performed worse on the first trial of the verbal fluency test, compared to low-hostile women. Men perseverated more on each trial of the verbal fluency test, while women perseverated less across trials. High-hostile men's time perception seems to be more rapid than low-hostile men, while for women it is the opposite. Women reported significantly more stress from the figural fluency task than men. Women in the luteal phase of menstruation did better on the verbal fluency test than women in the follicular phase of menstruation, and hostility and menstrual phase interact with verbal fluency.

This study encourages the consideration of neuropsychological sex differences in order to better understand cardiovascular regulation mechanisms and disease, leading to the development of improved prevention and behavioral management programs. Findings supporting this idea may bring about a new research focus, as some forms of cardiovascular disease may be more appropriately investigated as arising from neuropsychological problems.

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This project is dedicated to my wife, DeAnn, my sweetheart. Her love and support has endured. I am also grateful for the support of my parents, Dan and Diana Higgins, and my wife's parents, Robert and Raleine Allen. They made continued progress possible and always encouraged me. My children Aaron, Zachary, Kyra, Cyera, Drew, and Caleb provided inspiration to continue, despite the odds.

TABLE OF CONTENTS

TABLES AND FIGURES (in order of appearance)

A NEUROPSYCHOLOGICAL INVESTIGATION OF SEX DIFFERENCES IN CARDIOVASCULAR REACTIVITY TO VERBAL AND SPATIAL FLUENCY TASKS: TESTING A NEW MODEL OF SEX DIFFERENCES IN CARDIOVASCULAR REGULATION AND DISEASE

Overview

The literature review portion of this document details theories of sex differences in neuropsychological functioning and then critically evaluates assorted neuropsychological models of cardiovascular regulation (including a review of recent developments in the understanding of cortical control over cardiovascular factors). In addition, the mechanisms which exacerbate faulty cardiovascular regulation patterns and lead to types of cardiovascular disease are discussed. A new model detailing three principal functional units in cerebral regulation of cardiovascular factors is presented, and through this merging of theories a neuropsychological model of sex differences in cardiovascular regulation is developed. The integration of these literatures allows for a more complete investigation of the neuropsychological regulation of cardiovascular functioning and the importance of the role of neuropsychological sex differences in this model. Subsequently, the proposed model presents a new, neuropsychological conceptualization of sex differences in cardiovascular disease, and it is meant to aid in the reassessment or reinterpretation of previous research data, where applicable or useful, as possible implications and/or applications of this model are considered.

This document attempts to accomplish two goals: First and foremost, to advance neuropsychological theory through research by experimentally testing the models proposed in this paper. The experiment was designed to test the model of neuropsychological sex differences in cardiovascular regulation, and to test the concept of dissimilar neuropsychological mechanisms leading to sex differences in cardiovascular disease (the role that hostility, or neuropsychological dysfunction, plays in this disease state). Second, this document is intended to complement previous findings of neuropsychological sex differences in language, emotions, spatial processing, motor, and basic sensory tasks, and lend support to the idea of sex differences in yet another functional cerebral system. This document is constructed in a manner to provide an investigation of the significance of neuropsychological sex differences on cardiovascular regulation and disease. Thus, the aim is to provide a parsimonious portrait of the current state of neuropsychological research concerning both sex differences and cardiovascular regulation, then attempt to lay the foundation of a new model, fashioned from fundamental research findings and new ideas in a design formed to support the structure of the proposed model. Furthermore, it is hoped that these theories and experimental results contribute to a new, meaningful model of sex differences in cardiovascular regulation and a better appreciation for possible neuropsychological mechanisms leading to cardiovascular disease.

Introduction

Cardiovascular Disease Epidemiology

Every thirty-three seconds, someone in America dies from cardiovascular disease (CD), it claims 2,600 Americans each day. It is the No. 1 killer of men and women. The American Heart Association (A.H.A.) has reported that cardiovascular disease claimed a total of 953,110 lives in 1997 (41.2%, or 1 of every 2.4 deaths in America); moreover, it was approximately 60% of total mention mortality, which means that it was listed as a primary or contributing cause of death for over 1.4 million of the more than 2 million deaths from all causes in 1997. This is compared to 539,577 lives lost to all types of cancer, 95,644 lives lost to accidents, and 16,516 lives lost to AIDS in 1997 (A.H.A., 1999). In fact, cardiovascular disease claims more lives each year than the next seven leading causes of death combined. Current estimates suggest that 59.7 million Americans (1 in 5) have one or more of the following forms of cardiovascular disease: high blood pressure, 50 million; coronary heart disease, 12.2 million; myocardial infarction (acute heart attack), 7.2 million; angina pectoris (chest pain), 6.3 million; stroke, 4.4 million; congestive heart failure, 4.6 million, and rheumatic heart disease, 1.8 million (A.H.A., 1999). In industrialized countries, the incidence of CD is prognostic of expected longevity at the time of birth (Theorell & Haerenstam, 2000).

The American Heart Association reports that cardiovascular diseases will cost America 326.6 billion dollars in the year 2000 through medication costs, health professional services, and lost productivity from morbidity and mortality. Understanding the mechanisms contributing to heart disease risk can lead to lowering its incapacitating effect through more precisely tailored prevention programs and treatment protocols. If all forms of CD were eliminated, life expectancy would rise by almost 7 years, compared to a gain of 3 years through an elimination of all forms of cancer (1999).

Sex differences contribute to the disparity of heart disease mortality rates among men and women. One in three men can expect to develop some major form of CD before age 60, while one in ten women can expect the same. Each year men die of CD 40% more often than women (505 vs. 361 deaths per 100,000, respectively). Furthermore, men die of coronary heart disease 53% more often than women do: 238 vs. 155 deaths per 100,000, respectively (A.H.A., 1998). Moreover, recent research suggests that, at age 40, the *lifetime* risk of developing coronary heart disease is 48.6% for men and 31.7% for women (Lloyd-Jones, Larson, Beiser, & Levy, 1999). Even at age seventy, the lifetime risk is 34.9% for men and 24.2% for women. Consequently, population data from Framingham and elsewhere show that women have an average of 10 to 15 years more CD-free life expectancy than men, due to a later onset of disease (A.H.A., 1999; Castelli, 1988; Mark, 2000).

However, *residual* lifetime risk of developing hypertension by middle age (55 and older), commonly viewed as one of the milder forms of CD, is 90% for both men and women (Vasan et al., 2002). Furthermore, as men and women age, the epidemiological disparity of more serious forms of cardiovascular disease diminishes, especially after menopause in women (see Figure 1). In fact, in terms of total deaths (which is different than mortality rates), more women than men have died from CD every year since 1984 (see Figure 2). Cardiovascular diseases claim the lives of more than half a million women every year, more lives than the next 14 causes of death combined, about a death a minute (A.H.A., 1999). Unfortunately, many women are more afraid of breast cancer than of cardiovascular diseases, even though 1 in 28 women die from breast cancer while almost half die from cardiovascular disease (A.H.A., 1999).

The misconception, that cardiovascular disease in women is not a widespread problem, has contributed to sex discrimination in cardiovascular disease treatment. For example, research data shows that following a finding of an abnormal stress test, women were referred far less often than men for cardiac catheterization and coronary artery bypass graft surgery (Tobin et al., 1987). Others have confirmed (Ayanian & Epstein, 1991; Steingart et al., 1991) that sex-based differences exist in many characteristics of care for cardiovascular disease. However, as women frequently have different disease symptoms than men (Framingham data demonstrate that women with CD most commonly present with angina pectoris initially whereas men often first present with myocardial infarction (Lerner & Kannel, 1986)) and often get heart disease later in life, it may be more difficult for physicians to diagnose serious heart disease in women than men (Mark, 2000). Thus, more research is needed to clarify sex differences in cardiovascular disease causes and symptoms, and to establish effective prevention and treatment protocols.

What is it that attenuates cardiovascular disease risk in women compared to men? Endogenous estrogen has often been credited as a protective agent in women. However, when other factors are weighed when analyzing sex differences in coronary heart disease mortality, this effect cannot explain these differences; thus, environmental, and/or other factors also contribute (Lawlor, Ebrahim, and Smith, 2001). There is an urgent need to better understand the underlying neuropsychological mechanisms contributing to this epidemiological sex difference and to develop promising interventions and management programs based on this new understanding. The brain is currently the new frontier under study, and as its conduits of influence over health variables continue to be discovered, a greater emphasis on differences in cerebral functioning (dynamic, lateralized, compartmentalized, priming and inhibitory effects, system effects and interference, etc.) will become more salient in the literature across many areas of study.

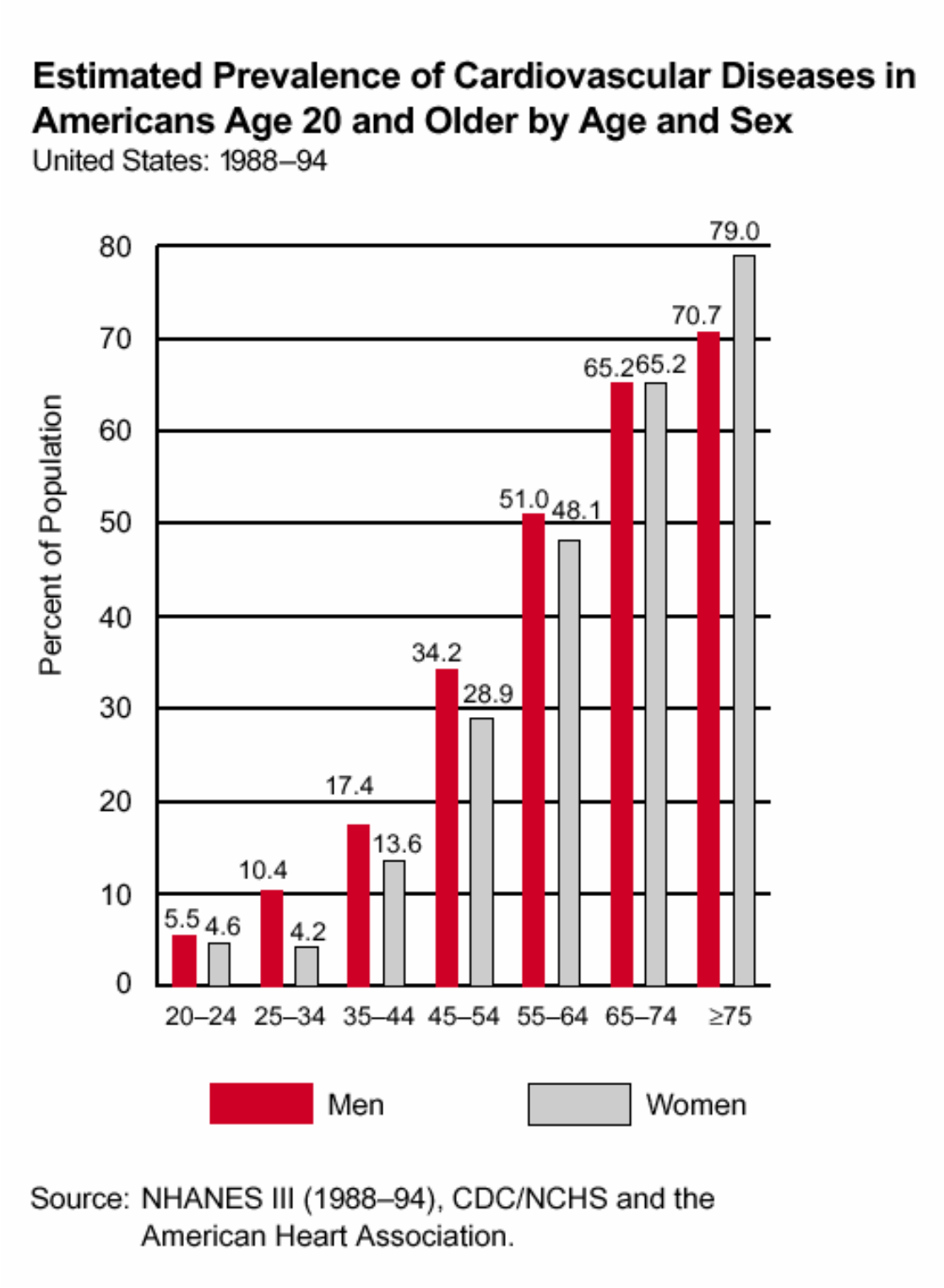


Figure 1. Prevalence of CD in American Men and Women at Different Ages.

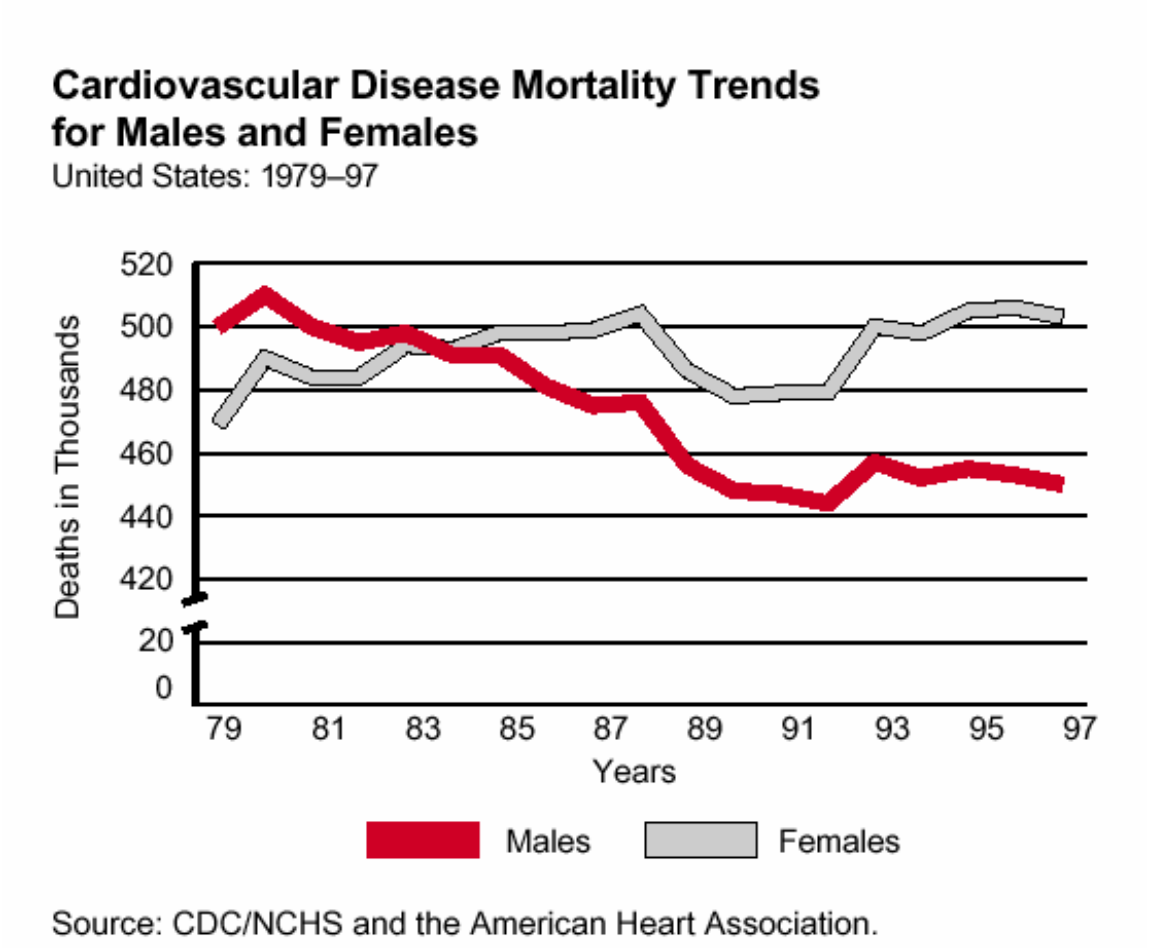


Figure 2. CD Mortality Trends in Men and Women.

Investigating Neuropsychological Mechanisms

The brain directs activity in all bodily systems and the cardiovascular system is one of the most important under its control. Some forms of cardiovascular disease can arise from cerebral dysfunction and can thus be conceptualized as brain disorders that produce cardiovascular disease. In addition, as neuropsychology is a field which focuses on relationships between brain structures and behavior (Pribram, 1964), cognition, and emotion, it has contributed enormously to the understanding of the underlying cardiovascular reactivity patterns of emotional states. Through a neuropsychological perspective, emotions and cardiovascular reactivity can be understood as functions of relative cerebral activation patterns and corresponding cardiovascular reactions. An exploration of relevant neuropsychological research data about sex differences in these patterns is herewith undertaken, and the present investigation is meant to complement the current understanding of neuropsychological mechanisms underlying sex differences in cardiovascular regulation and disease. This document proposes that this route of research is needed, as merging these literatures augments the current understanding of cardiovascular regulation and may improve treatment of cardiovascular disease. Also, as these literatures are critically evaluated, the significant effect of neuropsychological sex differences on cardiovascular regulation and disease can be better appreciated and considered more often when conducting future research. Furthermore, dynamic changes in cerebral laterality and functionality have been found, and these shifting forces need to be accounted for and integrated when discussing any comprehensive neuropsychological model. Findings of basic, fundamental sex differences in neuropsychological functioning lay the groundwork of the model under examination.

The Neuropsychology of Sex Differences

Neuropsychological research provides a valuable perspective of sex differences. Researchers have continued to be fascinated by sex differences, as this has been a persistent topic of study in neuropsychology. Sex, as a factor, is discussed throughout this text, rather than gender (Gentile, 1993), to emphasize basic differences between neural structure and function in men and women. Through the history of the neuropsychology of sex differences, an emphasis has been placed on cerebral laterality. Nevertheless, sex differences in reactivity and corresponding patterns of dynamic functional cerebral laterality are also beginning to be explored. This section will describe the developmental influences on the design of functional cerebral laterality, with data presented about hormonal and sensory effects on sex differences in cerebral cytoarchitecture, laterality, and then functional systems. Next, sex differences will be described that have been found in various, basic indices of cerebral functioning, such as the visual, auditory, somatosensory (galvanic skin response), and motor systems. In addition, measures of cognitive functioning detailing sex differences in the functional asymmetry in language, spatial, and emotion processing will be reviewed. Also, the importance of functional cerebral sex differences to recovery and performance measures is highlighted. The dynamic changes that occur with age are then briefly discussed. Thus, indications of sex differences found in these processes lead to the question of whether functional cerebral sex differences exist in cardiovascular regulation as well, which may lead to related sex differences found in cardiovascular disease.

Sex differences in cerebral symmetry have been detailed throughout the history of brain research. For instance, well over a century ago it was noted that the "tendency to symmetry in the two halves of the cerebrum is stronger in women than men" (Chrichton-Browne, 1878). At that time, evidence for such a statement was deficient, but over the years corroboration of this observation has accumulated (Kimura, 1987). Nonetheless, although sex differences in brain functioning due to cerebral asymmetry have been documented, they continue to remain controversial (Hellige, 1990; Turkheimer & Farace, 1992). Structure is often more readily discussed than function and some researchers seem reluctant to ascribe functional differences to structural dissimilarities. Nevertheless, structure and function must be considered jointly, as these characteristics of the brain are undeniably related to one another. By and large, it is remarkable that men and women act in such similar manners, despite the number of cerebral variations that have been found (De Vries & Boyle, 1998). Although the typical approach to this line of

investigation has often focused on sex differences in cerebral laterality, characteristic generalizations about lateralization of function have begun to give way to a more comprehensive study of cerebral systems. Thus, as functional neuropsychological mechanisms were previously assigned to a right-left lateral axis, they are now often allocated to frontal-caudal and cortical-subcortical axes (Harrington, 1995), or, similarly, dorsal versus ventral divisions of corticolimbic architecture (Liotti & Tucker, 1995). Laterality, in itself, does not seem to allow enough latitude to depict complete systems in detail.

Nevertheless, historically, neuropsychological sex differences have been classified for the most part as differences in cerebral laterality. These differences have been found in spatial processing (Berlin & Languis, 1980; Casey, Pezaris, & Nuttall, 1992; Davidson, Cave, & Sellner, 2000; Howard, Fenwick, Brown, & Norton, 1992; Kimura & Harshman, 1984; Kingsberg, LaBarba, & Bowers, 1987; McGlone & Kertesz, 1973; Rippon, 1990; Witelson, 1991), emotion processing (Braun, Baribeau, Ethier, Guerette, & Proulx, 1988; Davidson & Schwartz, 1976; Davidson, Schwartz, Pugash, & Bromfield, 1976; Hanske-Petitpierre & Chen, 1985; Harrison, Gorelczenko, & Cook, 1990; Johnsen, 1993), and language processing (Kimura & Harshman, 1984; McGlone, 1977; Rippon, 1990), where men and women differ in the degree to which these processes are lateralized. Data often suggests that men are more lateralized in these abilities than women (Butler, 1984; Gur et al., 1999; Sundet, 1986); that is to say, these cerebral processes are more laterally positioned in men and located more bilaterally in women. Notwithstanding, some early studies have suggested that women are more functionally lateralized than men for some tasks (Davidson et al., 1976).

Sex differences in cerebral functional specialization and lateralized cerebral operations, or symmetry in brain systems develops through various mechanisms. The basis for the differential rates of neural development of the cerebral hemispheres should not be viewed as context-free (Turkewitz, 1993), as there is evidence that neuroanatomical structures can be shaped by experience. Structure and environment are co-acting systems which give rise to brain functionality. Some of the current theories on cerebral development follow.

Prenatal/Hormonal Influences on Laterality and Functional Differentiation

As some cerebral systems differ in men and women, especially when considering laterality differences, the neurochemical influences that help shape the formation of these neural networks should not be underestimated. Hormones may influence the development of cerebral (a)symmetry during gestational development in men and women, as well as dynamic laterality shifts later in life. That is to say, as experience shapes brain structure and function, sex has a significant impact on brain development, influencing both cerebral lateralization and cerebral system functionality. Differences in cerebral symmetry can later impact sex differences in emotional regulation and cardiovascular reactivity (which can lead to the development of cardiovascular disease), and, ultimately, differences in mortality rates in men and women.

Sex hormones have been found to influence lateralization of brain structure when exposure has occurred during prenatal (Hines & Shipley, 1984; Joslyn, 1973) and neonatal (Holman & Hutchison, 1991) development (Lewis & Diamond, 1995). Differing hormone levels have also been found to correspond with adult brain asymmetry, where higher testosterone has been found to be associated with higher degrees of lateralized function (Moffat & Hampson, 2000). Since lateralization and cerebral system functionality are partly developed through influential sex effects, this mechanism has begun to be studied more closely. Exposure to various levels of hormones during human gestation appears to play a primary role in the cytoarchitectural design of the central nervous system in men and women, and these gestational hormones help determine whether the brain will have structural features characteristic of the male or female. During the initial phase of fetal development, the brain and physical character of the body are female in appearance. Around the seventh week following conception, the Y chromosome, signifying a male genetic pattern, is activated. At this point, sex differences in the design of the brain begin to be evident as testosterone, believed to be largely responsible for anatomical sex differences, is released (Ehrhardt & Meyer-Bahlburg, 1981; Fishbein, 1992; Valzelli, 1981). Nevertheless, outside influences also are affecting cerebral development. As sensory stimuli has been found to shape cerebral functionality, these influences also merit further study.

Sensory Stimuli Influences on Laterality and Functional Differentiation

Research has supported the notion of dynamic cerebral development. For example, Turkewitz (1993) has suggested that auditory experience helps guide brain laterality. This idea was explained by Fred Previc in 1991. He theorized that cerebral lateralization in humans is a result of the asymmetric prenatal development of the ear and labyrinth (vestibular organ). The aural asymmetry gives rise to a right ear advantage, especially for sound in the mid-frequency range (i.e., speech). This advantage is a result of the fetal position that has been found to exist among the majority of developing babies. Typically, the predominant position of the fetus during the final trimester of pregnancy is a cephalic (head-down) one, with its right ear facing out- against the mother's anterior abdominal wall (Taylor, 1976). Thus, the bodily position of a developing fetus can be an important factor in the development of functional cerebral asymmetry, similar to the way a genetic program directs other facets of cerebral functionality. In addition, aspects of vestibular lateralization can be traced to the asymmetric positioning of the fetus *in utero* during the final trimester. As the baby's position combines with maternal locomotor patterns, a lateralized shearing effect favors the development of a left-otolithic advantage (Previc, 1991). This advantage gives rise to a reliance on the left side of the body for postural control and the right side for voluntary motor behavior. The right hemisphere also develops a specialization for most visuospatial operations. However, Walker (1994) has declared that alternative theories of lateralization of human brain function, such as the one proposed by Previc, find little support when considering the neural pathways involved in the process of asymmetric laryngeal innervation in language lateralization. He contends that the auditory system has much less contralateral and more ipsilateral neural projections than other sensory systems. It would be difficult to separate which pathways were responsible for specialized laterality (Walker, 1994). Nonetheless, findings are still inconclusive; thus, this area deserves further research consideration.

In addition to the auditory and vestibular stimulus-directed functional cerebral laterality theories above, researchers have theorized that visual stimuli seem to organize cerebral structural functionality during development as well, along with the space mapping within the structures (Knudsen, 1991; Knudsen & Brainard, 1991; Knudsen, Esterly, & Du Lac, 1991). New theories describe cerebral sensory and motor maps as dynamic, shaped by sensory experience (and by eye-head position signals) in developing animals. The superior colliculus (a small area at the "roof" of the brain stem and an integral part of the visual system) provides an ideal structure for examining the neural mechanisms underlying immediate modifications of structural cerebral functionality (information representation in the brain), as it integrates spatial information from various sensory systems (especially auditory) and higher brain centers into a common oculocentric (relative to the eye) frame of reference (Knudsen, 1991). In barn owls raised with their eyelids sutured closed, the precision and topography of the auditory space map and the motor map are severely disrupted. These owls typically overestimate the origin of a sound and then turn toward/past it (Knudsen & Brainard, 1991). In a study where prisms were mounted in front of owls' eyes during development, the auditory space map shifted "according to the optical displacement induced by the prisms" (Knudsen, 1991; Knudsen et al., 1991). These results indicate that visual experience during development shapes the collicular auditory map in a site-specific manner and dictates its alignment with the visual map. Consequently, research suggests that cerebral laterality and specialized functionality is developed through various sensory modalities, with auditory stimuli/systems aiding in laterality and visual stimuli/systems aiding in the design of cerebral system integration (of sensory information with motor production).

Accordingly, Péter Érdi (1993) has presented a Neurodynamic System Theory to attempt to account for the changing conceptualizations of neural organization. He suggests that generalized causal dynamic models can describe the self-organizing mechanism of the nervous system, connecting structural and functional aspects of neural organization. The brain can be considered as consisting of hierarchically organized self-organizing structures. Self-organization is considered as a mechanism for generating emergent neural structures. Thus, dynamic neural structures can be modified by environmental influences, both pre- and post-natally (Erdi, 1993), as others have noted, "both internal and external environmental components are continuously, directly or indirectly, altering the cerebral cortex" (Lewis &

Diamond, 1995); p. 31). Consequently, continuing influences on cerebral functionality should be investigated, especially as whole cerebral systems are affected by dysfunction in one area.

Assessing Sex Differences

A common way to assess lateralization of function is to present separate stimuli simultaneously to the left and right ears or to the left and right visual fields. Due to the lateralized organization of the auditory and visual perceptual systems, responses to lateralized stimuli are related to lateralized organization of the brain. The large literature on sex differences in language that uses lateralized stimuli presentation in visual and auditory modalities suggests that there is greater lateralization in men (Beaumont, 1982; Bryden, 1982; Crews & Harrison, 1994b; Davidson et al., 2000; Fairweather, 1982; Harrison & Gorelczenko, 1990; Hellige, 1990). As various functional cerebral systems have been tested, similar results have indicated that men are more lateralized than women for various sensory and motor modalities. In addition, interconnectivity between the cerebral hemispheres has been found to be greater among women, possibly leading to some of the heightened symmetrical effects (Dorion et al., 2000).

Neuropsychological Sex Differences in Verbal Processing

Right-handed men traditionally have shown more extensive functional cerebral lateralization of language at the left hemisphere (Hines, 1990), while women have been found to have more bilateral processing of language (Vikingstad, George, Johnson, & Cao, 2000). However, this issue has also been controversial, as some researchers have continued to support similar language processing (left hemisphere) for both sexes (Frost et al., 1999). Nevertheless, the preponderance of the data supports the view that there are significant neuropsychological sex differences in language tasks, with men evidencing more functional asymmetry than women.

Dual-task interference investigations have been used to demonstrate neuropsychological sex differences in language laterality. In one study, when right-handed subjects were asked to perform simultaneous reading and finger-tapping tasks, verbalizations disrupted right-handed performance in men but disrupted left-hand performance in women (Wolff & Cohen, 1980), another study found interference for simultaneous verbalizations in right-handed men and women, where it was found that the speech task disrupted right-hand performance in men but left-hand performance in women during a unimanual force production task (Elliott, Weeks, Lindley, & Jones, 1986). In a similar study, men and women solved a block design problem with one hand while either simultaneously finger tapping with the other hand or vocalizing words. Finger tapping had no differential effect on performance in either sex. However, vocalization disrupted women's performance using *either* hand, suggesting that verbal abilities are more bilateral in women (Kingsberg et al., 1987).

Dichotic listening tasks have also been used to assess language lateralization in the brain (Kimura, 1961; Kimura, 1967; Kimura & Harshman, 1984), which have found overall right ear advantages (Shankweiler & Studdert-Kennedy, 1967; Studdert-Kennedy & Shankweiler, 1970). Nevertheless, dichotic listening tasks have suggested that women are more able (than men) to perceive speech sounds in either ear, while men have difficulty with speech perception at the left ear (Higgins, 1999). In this experiment, women were able to focus on dichotic sounds at the left or right ear, significantly increasing accuracy at either ear. Conversely, men were unable to dynamically change their accuracy of dichotic speech sound identification as a function of focusing on sounds at each ear; men maintained their right ear advantage irrespective of requested focus (right, left, or no focus) condition.

Sex differences in lateralization of language function have also been studied with functional imaging techniques, the most common being electroencephalography (EEG). This method can offer a glimpse of brain function while the subject performs various tasks, providing relatively direct information about brain activity. Notwithstanding the imprecision of EEG (low spatial resolution), it is an adequate means of testing hypotheses about the activation of large structures, such as the cerebral hemispheres. In EEG studies of sex differences in lateralization of function, measurements are taken at sites in the left and right hemispheres while subjects complete tasks designed to selectively activate one hemisphere. Comparisons of ratios of left to right activation during the two types of tasks are used as an indication of lateralization of function. Most studies have shown that men have greater differential activation of the hemispheres between verbal and nonverbal conditions (Glass, Butler, & Carter, 1984; Ray, Morell,

Frediani, & Tucker, 1976; Trotman & Hammond, 1979). These studies are based on the power function of EEG spectra, which is related to the mean level of desynchronization during a task. When EEG data are analyzed for coherence, a measure of the correlation in desynchronization across frequencies at inter- or intrahemispheric sites, female subjects have been shown to have greater degrees of interhemispheric coherence on both verbal and nonverbal tasks (Beaumont, Mayes, & Rugg, 1978; Flor-Henry & Koles, 1982). This finding is consistent with the idea of greater interhemispheric cooperation in women, an indication of increased symmetry during verbal processing.

Other functional cerebral imaging studies have corroborated these findings. In a study of regional cerebral blood flow (rCBF) using statistical comparisons analogous to the analysis of coherence in EEG spectra, described above, correlations among the activation of eight left hemisphere and eight right hemisphere regions of interest obtained during a spelling task were analyzed (Wood, Flowers, & Naylor, 1991). There was a positive relationship between Broca's area (language production) and Wernicke's area (language reception) in men but not in women. Instead, "bitemporal coupling" was found in women, in which left temporal regions were activated in parallel with right temporal regions. The linkage of *intra*hemispheric regions in men and *inter*hemispheric regions in women suggests greater lateralized processing in men and bilateral processing in women. Functional magnetic resonance imaging studies have also uncovered similar sex differences in cerebral functioning during language tasks (Shaywitz et al., 1995). Sex differences have also been found in other neuropsychological tasks, such as spatial processing, emotional processing, and even motor functioning.

Neuropsychological Sex Differences in Spatial Processing

Similar to language processing, right-handed men have been found to process spatial information more exclusively in the right hemisphere, compared to women (Kingsberg et al., 1987). Thus, mean appear to have more pronounced cerebral lateral specialization in the right hemisphere for spatial information, compared to women, who appear to have more bilateral spatial processing abilities (Beaumont, 1982; Bradshaw & Nettleton, 1983b; Bryden, 1982; Fairweather, 1982; McGlone, 1980; Springer & Deutsch, 1989). The relationship between the right hemisphere's specialization for visuospatial processing and emotional regulation has been interpreted by Flor-Henry (1983) as indicating that males (referred to as "specialized females") have acquired a more lateralized and efficient right-hemisphere than females. The specialized functions include more efficient visuospatial processing, musicogenic ability (music composition), and emotional stability (Flor-Henry, 1983). Nevertheless, such a sweeping statement warrants further research to authenticate its veracity.

Related to the idea of spatial processing, basic visual processing sex differences have been found. Wada et al. (1996) found significant sex- related differences in EEG coherence tasks during photic stimulation (PS), with females having significantly higher coherence than males at electrodes F3-F4 and C3-C4, in the frequency band (4.5-5.5 Hz) corresponding to 5 Hz PS. In addition, sex differences were found during the change in interhemispheric coherence from rest to the PS stimulus condition, where females had significantly greater coherence reactivity for occipital electrodes O1 and O2 in EEG during PS at 5 and 15 Hz (Wada et al., 1996). Such sex- related differences in interhemispheric EEG coherence during PS offer more evidence that sex differences exist in the degree of lateralization of cerebral sensory function, even at the level of basic sensory processing.

Laterality versus Performance

It is interesting to note the relationship that laterality has with performance. Often times increased laterality translates into increased performance levels (Wendt & Risberg, 1994). Primary investigations found that men often outperformed women on tasks requiring spatial processing; nevertheless, investigations of sex differences in verbal-linguistic processing found women to have an advantage over men (Benbow & Stanley, 1980; McGlone, 1980; Springer & Deutsch, 1989). Consequently, although men show increased laterality for both verbal and spatial processing, they outperform women in spatial tasks, while women outperform men in verbal tasks. In fact, women's enduring verbal advantage seems to begin in early childhood (Kramer, Delis, Kaplan, O'Donnell, & Prifitera, 1997), and includes verbal fluency measures (Muniz, Garcia-Cueto, Garcia-Alcaniz, & Yela, 1985). Notwithstanding, men's superior spatial task performance is one of the most robust findings in the

literature about sex differences (Broverman, Klaiber, & Kobayashi, 1968; Dawson, Cheung, & Lau, 1975; Harris, 1978; Joseph, 1992; Kimura, 1993a; Kimura, 1993b; Levy & Heller, 1992). The largest spatial sex difference is found in mental rotation task performance (Collins & Kimura, 1997; Johnson & Meade, 1987; Moffat, Hampson, & Hatzipantelis, 1998; Shepard & Metzler, 1971; Silverman & Eals, 1992; Vandenberg & Kuse, 1978), with sex differences growing larger with increased task difficulty (Rich & Maki, 2000). These differences have been shown to exist despite the finding that women have shown decreased laterality (more functional cerebral symmetry) for language systems (Davidson et al., 2000; Hirst, 1982) and also spatial systems (Kingsberg et al., 1987). Thus, *symmetry* of verbal abilities in women gives rise to an advantage over men, while *asymmetry* of visuospatial abilities in men gives rise to augmented performance relative to women. Therefore, functional cerebral (a)symmetry differentially affects each sex, with symmetry (seemingly) aiding women in language operations and asymmetry aiding men in visuospatial performance. Accordingly, this relationship warrants further investigation. Correspondingly, neuropsychological research of emotions has increased recently, as findings impact so many other fields of psychological study.

A Brief History of Neuropsychological Research on Emotions

Early research on emotion focused on physiological changes in the autonomic nervous system. Later, mediating subcortical limbic system circuits were studied which are involved in specific emotional behaviors (Heilman & Bowers, 1990; Heilman, Bowers, & Valenstein, 1985; Heilman, Bowers, & Valenstein, 1993). Nevertheless, as the psychophysiology of emotions has been studied more closely, the key role of the cortex has become more evident in regulating emotional behavior (Davidson, 1993). Also, an important distinction between the cognitive and biological aspects of emotional responding has been made, as it is important to note the right hemisphere's specialized ability to think about an emotion. The experience of feeling an emotion, in contrast, involves cortical mechanisms in the control of arousal (Heller, 1990). As the cerebral cortex (and neuropsychological systems) have been studied more closely a better comprehension emotion has surfaced, and neuropsychological theories of emotion have been able to better explain normal (positive and negative) emotional reactivity patterns, various instabilities, and related emotional disorders.

Studies have consistently revealed a left visual field superiority in processing facial affective stimuli (Dimond, Farrington, & Johnson, 1976; Gainotti, 1972; McKeever, 1986; McKeever & Dixon, 1981; Schwartz, Davidson, & Maer, 1975; Wechsler, 1973; Wedding & Cyrus, 1986). These studies suggest that both positive and negative emotion are regulated more by the right hemisphere than the left hemisphere (Harrison & Gorelczenko, 1990). Furthermore, overall arousal, a precursor to emotional responding and reactivity has been shown to be regulated by the right hemisphere (Heilman & Van den Abell, 1979). However, further research indicates that the although the right hemisphere has been shown to regulate both positive and negative emotions, the left hemisphere has been shown to aid in the regulation of positive affect (Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Davidson & Fox, 1982; Gainotti, 1983; Harrison & Gorelczenko, 1990; Sackeim & Gur, 1978; Sackeim, Gur, & Saucy, 1978; Schwartz, Ahern, & Brown, 1979; Silberman & Weingartner, 1986; Tucker, 1981). However, care should be taken when assuming that research is concluded on the matter, as many emotion studies have used brain-damaged populations (Gainotti, 1984).

Functional imaging research using electroencephalography (EEG) has also demonstrated that right hemisphere EEG activation in the frontal region has been shown to elicit negative emotional experience, expression, and perception, while left hemisphere EEG activation in the frontal region has been shown to elicit positive emotional experience, expression, and perception. However, there is evidence that positive emotions can be perceived by the right hemisphere as well, while negative emotions cannot be perceived by the left hemisphere (Tucker, Stenslie, Roth, & Shearer, 1981). These differences seem to appear as early as during the first year of life (Davidson & Fox, 1982; Fox & Davidson, 1988). Tucker (1981) has proposed a "balance" model of cerebral activation, where reciprocal inhibition takes place between homologous brain tissue of the right and left cerebral hemispheres. That is to say, left hemisphere deactivation may correspond with homologous right hemisphere activation (Asenbaum, Lang, Egkher, Lindinger, & et al., 1992), while behaviors (and related emotions) associated

with the relatively more active area would become more salient (Tucker, 1981; Tucker & Williamson, 1984). Greater right than left cerebral hemisphere activation during a stress condition with emotional arousal was found prior to the publication of this hypothesis (Tucker, Roth, Arneson, & Buckingham, 1977), which may have contributed to the formulation of the "cerebral balance" theory.

Neuropsychological Sex Differences in Emotional Processing

Research has shown that the right hemisphere has been demonstrated to be superior in interpreting nonverbal emotional expression in the face and speech (Campbell, 1978; Gainotti, 1984; Ley & Bryden, 1979; Rinn, 1984; Rothbart, Taylor, & Tucker, 1989; Weber & Sackeim, 1984; Wedding & Cyrus, 1986), which can also be seen as a type of visuospatial task. Also, the right hemisphere has been shown to be more involved in emotional expression (Rinn, 1984), even in infants (Rothbart et al., 1989). Thus, overall emotion regulation seems to be more of a specialty of right hemisphere functioning (Weber & Sackeim, 1984), and emotion experienced through self-induced imagery of past emotional experiences has also found right hemisphere advantages (Crawford, Clarke, & Kitner-Triolo, 1996). Few studies have found exception to this (Stalans & Wedding, 1985).

Similar to language processing, right-handed men appear to have even more pronounced cerebral lateral specialization in the right hemisphere for emotional processing, compared to women, who appear to have more bilateral emotional processing ability (Beaumont, 1982; Bryden, 1982; Crews & Harrison, 1994b; Fairweather, 1982; Harrison & Gorelczenko, 1990; Harrison et al., 1990; Hellige, 1990; Pardo, Pardo, & Raichle, 1993; Stalans & Wedding, 1985; Wedding & Cyrus, 1986). Notwithstanding, some findings of greater symmetry in women have been detailed (Johnsen, 1993). Results have supported the hypothesis that men show heightened functional cerebral laterality when processing emotional faces; although, relative symmetry has been found for positive affect (Harrison et al., 1990). Nevertheless, overall, men have evidenced heightened functional cerebral laterality across valences, as they have been found to process affective facial information significantly faster than women when presented in the left visual field. Thus, it is possible that women process negative emotion across diverse cerebral regions within the left and right hemispheres. In contrast, men have a significant advantage when processing negative emotion presented to the right cerebral hemisphere, whereas the capacity to allocate left cerebral systems to the processing of negative affect may be diminished. These findings support fundamental research indicating that the right cerebrum has a larger involvement in processing emotion in men (Ekman, Levenson, & Friesen, 1983; Johnsen, 1993). Moreover, the idea of increased laterality of emotion in men has been supported by continuing research conducted at Virginia Tech (Demaree & Harrison, 1997b; Demaree, Harrison, & Rhodes, in press; Herridge, Harrison, & Demaree, 1997; Higgins, 1999). Thus, men may be more asymmetrically organized than women for verbal, spatial, and emotional processes (Beaumont et al., 1978; Bradshaw & Nettleton, 1981; Bradshaw & Nettleton, 1983a; Bradshaw & Nettleton, 1983b; Flor-Henry & Koles, 1982; Kingsberg et al., 1987; McGlone, 1980; Springer & Deutsch, 1989). Emerging evidence has extended these sex differences from sensory tasks to motor tasks.

Neuropsychological Sex Differences in Motor Functioning

Dynamic changes in motor functioning have been found with differing emotional states (Crews & Harrison, 1994a; Crews, Harrison, & Rhodes, 1999; Crews, Harrison, Rhodes, & Demaree, 1995). Recent research has found interesting sex differences in motor asymmetry (Figure 3). In a review of studies conducted at Virginia Tech that included the dynamometer for grip strength data, right-handed men consistently demonstrated asymmetry in motor functioning, while right-handed women (surprisingly) have evidenced symmetry in this aspect of motor performance (Higgins, Williamson, & Harrison, in preparation). However, in women, hand asymmetry on this task increased in the elderly, which corresponds to increases in language asymmetry in women with age (Alden, Harrison, Snyder, & Everhart, 1997).

Dynamic Changes in Neuropsychological Functioning

Various neuropsychological functions have been found to be dynamic in nature (e.g., changes in neurochemistry can influence proprioception, the ability to accurately perceive one's position in space). Hormonally-mediated changes in interhemispheric dynamics have been studied where it was found that women showed menstrual phase-related cognitive changes that suggested altered hemispheric activation

for specific tasks (McCourt, Mark, Radonovich, Willison, & Freeman, 1997). Women, during a space bisection task, were more likely to have asymmetric hemispheric activation (similar to men) during the luteal phase than during the menstrual phase. Similarly, Mead and Hampson (1996) found that during the midluteal phase, left visual field accuracy (right hemisphere functioning) was significantly lower for both verbal and nonverbal tasks than during the menstrual phase. Further, right ear performance on nonverbal dichotic test was significantly reduced at the midluteal phase. Thus, there was evidence for suppression of right hemisphere processing, as well as possible reductions in corpus callosum transfers, usually a strength in women (Mead & Hampson, 1996). Further, significant interhemispheric asymmetry of binding sites associated with serotonergic mechanisms has been found in the orbital frontal cortex of 6 women and 6 men who died of natural causes (Arato, Frecska, Tekes, & MacCrimmon, 1991). They found that women had significantly more binding sites in the right orbital cortex than men. However, there was not a significant sex difference at the left orbitocortical areas. Interestingly, many serotonin-related psychological disorders have different incidence rates in each sex. Thus, this finding begins to offer a possible explanation for these observed differences; of course, frontolimbic pathways associated with both positive and negative emotion reactivity are also implicated by this research finding.

Consequently, when considering the idea of dynamic hemispheric interplay, there is some evidence that women may have better connectivity between the two hemispheres, via a more dense corpus callosum (Johnson, Pinkston, Bigler, & Blatter, 1996). These fibers are presumed to interconnect homologous association cortices in the two hemispheres. Thus, sex differences in the efficiency of interhemispheric transfer are important to consider, especially for tasks involving higher-order processing tasks (Clarke & Zaidel, 1994). These have been described as of bilateral representation of language, functional interhemispheric inhibition, and the maintenance of hemispheric differences in arousal. Nevertheless, support for this mechanism is mixed, at best. Also, differences in functional lateralization in men and women are better appreciated when considering how they can affect health, recovery, and research validity. It is possible that enhanced functional cerebral symmetry in women has contributed to decreased cardiovascular disease incidence. For example, with men, one consequence of increased specialization may be a greater risk of cerebral system dysregulation when neighboring systems are activated.

Importance of Sex Differences in Recovery

Sex differences in brain structure are of interest to the extent that they provide clues to functional differences in the brain. The considerable interest generated by sex differences in brain anatomy is closely related to the fact that anatomical brain differences related to sex appear to be associated with some of the more substantial dimensions of human brain function: lateralization, verbal ability, spatial ability, and recovery from brain injury (Farace & Turkheimer, 1996). Recovery is directly related to the issue of asymmetry and cerebral functionality, since more symmetric and/or diffuse brain functionality is not as vulnerable to focal, unilateral damage, such as that caused by a cerebrovascular accident (CVA), like a stroke. Clearly, differences in cerebral functionality can play a role in recovery from brain injuries. Research has suggested that men and women are at unequal risk for stroke, have different recovery profiles, and experience differences in the effects of aging on stroke (Sundet, 1988). These differences are due, in a large part, to laterality differences. Other researchers have also noted sex differences in laterality that contribute to sex differences in recovery. Some have noted that language functions appear to transfer more readily to the right hemisphere in girls than boys when children sustain damage to the left hemisphere (Kelly, 1991), while others have found better recovery profiles for women from aphasia, suggesting more distributed bilateral cerebral organization of speech function in women, compared to men (Basso, Capitani, & Moraschini, 1982; Pizzamiglio, Mammucari, & Razzano, 1985). Moreover, when studying sex differences in the impact of brain injuries among neurological patients, a strong association was found between the side of the brain that was injured and the type of cognitive deficits produced (Inglis & Lawson, 1981). In men, verbal functions were disturbed by left hemisphere lesions while nonverbal functions were disturbed by right-hemisphere lesions. However, in women, this association was much weaker, again suggesting more functional symmetry in women.

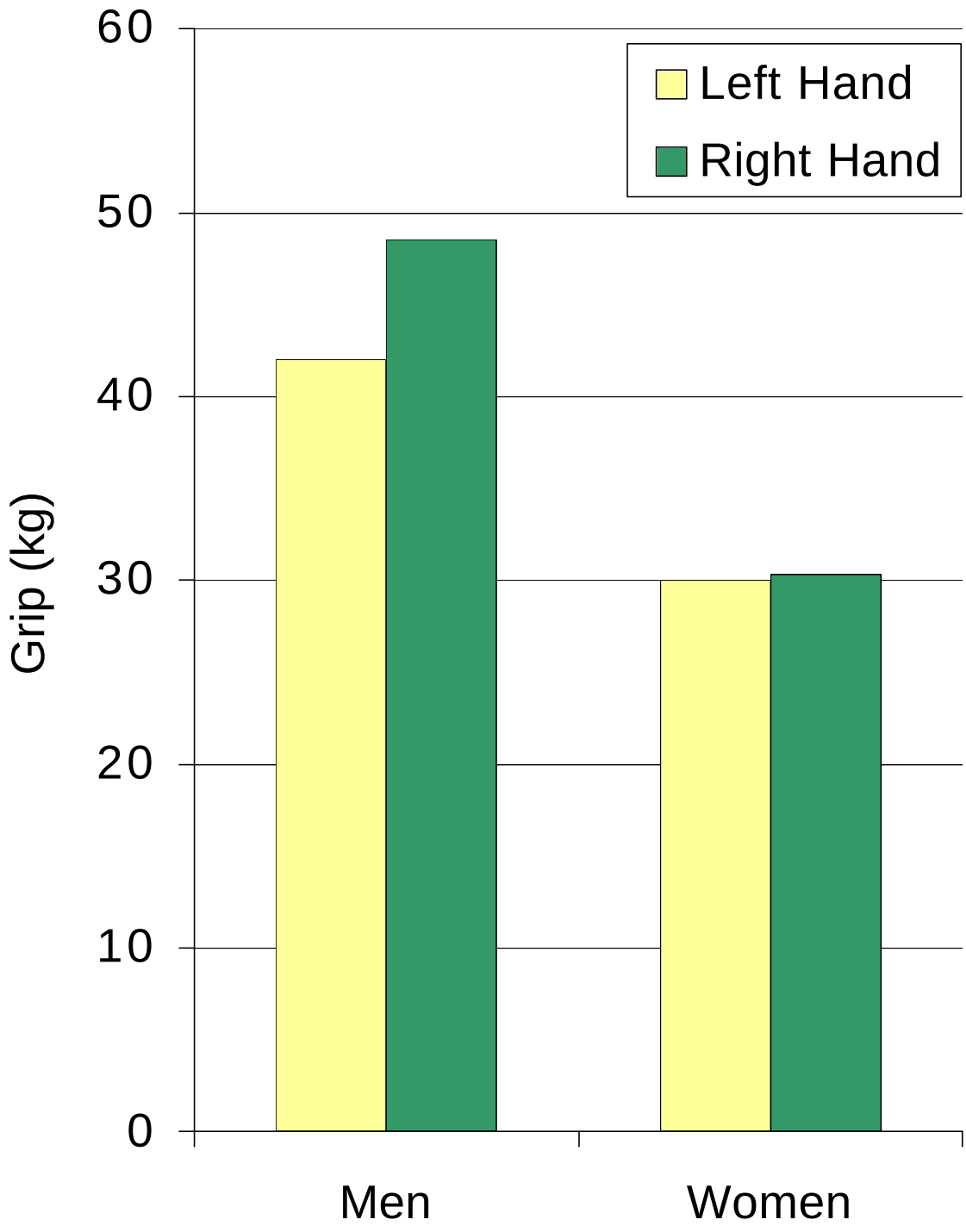


Figure 3. Sex differences in motor asymmetry for right-handed men and women (Sex by Hand Interaction Effect on Grip Strength).

The Age (and Aging) Factor

Dynamic age-related changes in cerebral functionality have also been found across sensory and motor modalities (Higgins et al., in preparation). In a study utilizing a dichotic listening procedure with younger (mean age = 20 years) and older (mean age = 72 years) women, younger women did not have difficulty switching their intention between left and right (ear) focus, measured through accuracy at each ear; whereas, older women had difficulty shifting their intention to the left but not right ear (Alden et al., 1997). This finding suggests increased lingual asymmetry, and it mimics the general pattern found with men, who have difficulty switching focus to the left ear (Higgins, 1999), notwithstanding some findings with hostile men who have been more able to switch focus (Demaree & Harrison, 1997b). In addition, as developmental factors have been studied in children, age has been found to be a factor in the development of lateralized (left) verbal fluency, as younger individuals have been found to use more bilateral processing, which becomes lateralized to the left hemisphere (frontal areas) as individuals age (Gaillard et al., 2000). Also, as testosterone levels change with age, it is interesting that a recent study found that a testosterone injection blocked the practice effect in verbal fluency in men, but did not affect spatial or verbal memory (Wolf et al., 2000). This finding partly supports the general idea that hormones may modulate performance in tests with known, robust sex differences. It also underscores the finding that women experience increases in cardiovascular reactivity and disease risk with age (and increased testosterone levels).

The functional asymmetry of the cerebral hemispheres has been a constant focus of research in neuropsychology- one of the most widely studied characteristics of the brain (Wittling, 1995). It is well accepted that the two cerebral hemispheres differ in various functions and basic cognitive processing (Trevarthen, 1990). Language, emotional, and spatial processing have been shown to be performed in specific areas of the brain, as heretofore discussed (with corresponding sex differences in these capacities). As significant sex differences for these cognitive and emotional processes and basic motor functions are considered, a new model for understanding sex differences in cardiovascular regulation may be contemplated. To be precise, it is possible that sex differences in cardiovascular regulation and disease are partly due to sex differences in cerebral asymmetry and functionality.

Neuropsychological Models of Cardiovascular Regulation

Neuropsychological models have proven useful when describing cardiovascular regulation and control, as neural pathways have been studied extensively between brain areas and corresponding cardiovascular responding. Consequently, neuropsychological studies of asymmetry and functional laterality have generated valuable models of cardiovascular regulation. Recent investigations into the roles of each cerebral hemisphere in cardiovascular regulation have highlighted the importance of appreciating the significance of neural control of cardiovascular functioning (and associated cardiovascular disease conditions). Through a neuropsychological model, one can conceptualize certain underpinnings of cardiovascular disease, both the possible biological vulnerabilities and, alternately, protective strengths. In this section, a brief history of neurological models of cardiovascular regulation will be portrayed, followed by a description of the direction of current research. Historically, cerebral laterality has been the emphasis in cardiovascular regulation- with right hemisphere regulation of autonomic, sympathetic cardiovascular indices being the main focus of investigation. Nevertheless, contemporary models will also be presented, which describe parasympathetic control by left cerebral systems. In addition, research will be reviewed that describes both right and left anterior temporal and orbito-frontal cerebral regions' influence on each other and on cardiovascular regulation, based on neuropsychological theories. This research will be explained under the designation of quadrant theory. Within this framework, the construct of hostility and mechanisms that lead from cardiovascular regulation to cardiovascular reactivity to cardiovascular disease will be detailed. Functional neuroanatomical systems will be described through information implicating dual-task effects (e.g., priming and interference) associated with dynamic factors and changes in cardiovascular regulation. Following this review, a new, neuropsychological model detailing *the three principal functional neuropsychological*

units of cardiovascular regulation will be presented. Information will describe elemental, functional neuroanatomical systems and the various, well-established theoretical bases of this model.

Recently, neuropsychology has provided more detailed theories of the neural regulation of cardiovascular functions; these models have proven to be both interesting and helpful in furthering understanding of cardiovascular regulation and disease. These theories began with a focus on cerebral laterality and have recently developed into models of cerebral systems involved in cardiovascular regulation. Cerebral management of cardiovascular functioning has been studied extensively through many methods of assessment. These have ranged from long-established psychophysiological methods of measurement to novel techniques of evaluation. Theories have not always been consistent when describing neural pathways controlling cardiovascular regulation; nevertheless, knowledge of essential mechanisms of cardiovascular control has increased with recent investigations. Early research suggested that the right hemisphere regulates general arousal levels, and may have greater control of the subcortical systems, which are largely responsible for arousal (Heilman & Van den Abell, 1979). This led to a model of right hemispheric involvement in various basic autonomic (heart rate and blood pressure) factors (Katkin & Reed, 1988; Yokoyama, Jennings, Ackles, Hood, & Boller, 1987), which was then narrowed to specify more sympathetic components (i.e., hearth rate and systolic blood pressure increases) of cardiovascular regulation (Caltagirone, Zoccolotti, Originale, Daniele, & Mammucari, 1989; Hachinski, Oppenheimer, Wilson, Guiraudon, & Cechetto, 1992; Hugdahl, Franzon, Andersson, & Walldebo, 1983; Lane, Novelly, Cornell, Zeitlin, & Schwartz, 1988; Lane & Schwartz, 1990; Rosen, Gur, Sussman, Gur, & Hurtig, 1982; Swartz, Abrams, Lane, Dubois, & Srinivasaraghavan, 1994; Walker & Sandman, 1979; Walker & Sandman, 1982; Yokoyama et al., 1987; Yoon, Morillo, Cechetto, & Hachinski, 1997). Thus, historically, the right hemisphere was associated with arousal, and a general model of right hemisphere control of autonomic responding developed from this literature base. Nonetheless, cerebral asymmetry has also been found in cardiovascular management, as new research data suggests that left hemisphere activation is associated with parasympathetic autonomic changes, suggesting bilateral cerebral control of cardiovascular regulation (Wittling, 1990; Wittling, 1995; Wittling, 1997; Wittling, Block, Genzel, & Schweiger, 1998a; Wittling, Block, Schweiger, & Genzel, 1998b; Zamrini et al., 1990; Zoccolotti, 1986). However, just as emotional networks include frontal inhibition of anterior cerebral activity, a cardiovascular regulatory system of frontal inhibition of anterior (sympathetic and parasympathetic) activity may account for certain patterns of data found in recent neuropsychological investigations of cardiovascular reactivity conducted at Virginia Tech. These results have implicated both anterior temporal areas and orbitofrontal areas in cardiovascular regulation. What's more, these principal models of right hemisphere control, cerebral laterality, and then fronto-temporal circuits of cardiovascular regulation all support a model of three principal functional units of cardiovascular regulation. Thus, assimilation of these lines of research (general arousal, lateralized sympathetic and parasympathetic factors, and frontal-temporal functional systems) can provide a new conceptualization, presented below, of three principal functional units of cardiovascular regulation.

Several investigative designs are later described to test this model, and, therefore, research the importance of neuropsychological sex differences in cardiovascular regulation. Furthermore, as emotion response patterns interfere with normal, healthy cardiovascular functioning, it is wise to explore these associations. Thus, mechanisms leading from dysfunctional cardiovascular regulation to cardiovascular disease, such as through hostility, remain important factors when discussing cardiovascular disease. In fact, this idea is discussed below and then later proposed to be tested in experiment two, described in the experimental design portion of this document. The progression in understanding cerebral control over cardiovascular factors begins with an analysis of right hemisphere studies.

The Right Hemisphere, Arousal, and Cardiovascular Regulation

The anterior right cerebrum has been found to be involved in the regulation of autonomic functioning in numerous studies (Caltagirone et al., 1989; Hachinski et al., 1992; Hugdahl et al., 1983; Lane et al., 1988; Lane & Schwartz, 1990; Rosen et al., 1982; Swartz et al., 1994; Walker & Sandman, 1979; Walker & Sandman, 1982; Yokoyama et al., 1987; Yoon et al., 1997). Heart rate has been found to change more in response to emotional and nonemotional stimuli presented to the right cerebral

hemisphere than the same stimuli presented to the left hemisphere (Hugdahl et al., 1983), suggesting right hemisphere regulation of heart rate. For example, Hachinski et al. (1992) studied cardiac effects associated with each cerebral hemisphere at the level of the cortex. These effects were measured following experimental stroke induced by left or right middle cerebral artery occlusion, involving the insula and portions of the perirhinal, frontolateral, motor, and sensory cortex. Effects upon mean arterial pressure and the QT interval of the ECG were greater if the right brain as opposed to the left brain was involved.

Thus, many studies examining the cardiac effects of unilateral brain damage in human subjects have been interested in investigating heart rate changes. Two influential studies were conducted by Caltagirone et al. (1989) and Zoccolotti et al. (1986). Caltagirone et al. (1989) found that left-brain damaged patients and control subjects experienced significantly greater cardiac decelerative responses to an emotionally negative film than a neutral film. However, right brain-damaged patients did not differ in their cardiac responses to the negative- or neutral-valenced films and experienced a smaller cardiac decelerative response to the emotional film than both the left-brain damaged patients and the control subjects. The researchers theorized that parasympathetic control of heart rate was impaired in right brain-damaged patients. Nevertheless, brain-damaged populations can yield various results, based upon the focus of damage in the groups.

Bilateral Cerebral Cardiovascular Regulation

Newer research has implicated both the right and left hemisphere in cardiac control. Research examining right and left hemisphere involvement in heart rate control has been conducted using techniques for unilateral hemispheric inactivation through intracarotid amobarbital injection. Rosen et al. (1982) found heart rate increases to be higher following left versus right hemisphere inactivation; similarly, Lane et al. (1988) found that heart rate increased during left hemisphere inactivation, "favoring patients with left hemisphere as opposed to right hemisphere damage" (Lane & Schwartz, 1990; Wittling, 1995). Further, Zamrini et al. (1990) studied 25 patients undergoing unilateral cerebral inactivation prior to epilepsy surgery. Heart rate was measured preceding amobarbital injection (1 minute) and after the injection (3 minutes). The researchers found a significant difference in heart rate changes following left vs. right intracarotid injection. "Mean heart rate increased up to 4 beats per minute (bpm) during left hemisphere inactivation and decreased up to 2.4 bpm during right hemisphere inactivation" (Wittling, 1995). Oppenheimer, Gelb, Girvin, and Hachinski (1992) found that stimulation of the left-insular region corresponded with decreased heart rate, while stimulation of the right-insular region produced increases in heart rate. Walker and Sandman (1979) found that spontaneous changes in heart rate were related to visual evoked potentials. Flashes of light were presented to subjects during fast, midrange, and slow heart beats. However, when looking at the visual evoked responses recorded over the left and right occipital lobes related to heart rate changes, the data were different for the two hemispheres. "Potentials recorded during high vs. slow heart rate could be differentiated from one another by waveform if recording was done over the right hemisphere. In contrast, potentials were not related to heart rate if recorded from the left hemisphere." Walker and Sandman (1982) then explored if the differences in the waveforms of visual evoked potentials could be found during periods of systolic vs. diastolic carotid pressure. They again found that "sensory evoked activity recorded from the right hemisphere differed significantly between systolic and diastolic pressure phases" (Wittling, 1995). While, Wittling, Block, Genzel, and Schweiger (1998) used lateralized film presentation for stimulation of the left and right hemispheres. They found that sympathetic activity seemed to be mainly controlled by the right hemisphere and parasympathetic activity by the left hemisphere (see also Wittling, 1997; Wittling, Block, Schweiger, & Genzel, 1998). Given these indications of the contribution of sex differences to emotional regulation and corresponding differential cardiovascular reactivity, several recent theories have proposed that functional cerebral asymmetry contributes to differences in emotional regulation in the right hemisphere (including aggression) in men and women, and that these differences may contribute to corresponding cardiovascular disease risks.

Left Hemisphere Involvement in Cardiovascular Regulation

Recent research has begun to lead investigators to develop a model of left hemisphere control over parasympathetic responding (Wittling, 1990; Wittling, 1995; Wittling, 1997; Wittling et al., 1998a; Wittling et al., 1998b; Zamrini et al., 1990; Zoccolotti, 1986). Evidence in early studies was not acknowledged, as it was typical to ascribe autonomic control to the right hemisphere, as seen in many of the studies detailed in the previous section. Nevertheless, the left hemisphere seems to be closely involved in overall health, especially when one considers the importance of the parasympathetic system when considering cardiovascular fitness. Cardiovascular disease can follow arterial injury sustained from either over-activation of sympathetic cardiovascular responding, or under-activation of parasympathetic cardiovascular control. Thus, it is important to continue to investigate the role that the left hemisphere plays in cardiovascular regulation, in combination with the right hemisphere. In addition, it is important to research sex differences in the functional cerebral systems of cardiovascular regulation (sympathetic and parasympathetic), as well as differences in the dynamic changes in functioning in these cerebral systems.

Mechanisms of Cardiovascular (Dys)Regulation Leading to Cardiovascular Disease

The severity of cardiovascular dysregulation can be understood in terms of real risks to an individual's health. The path paved with problems in cardiovascular regulation often leads to a life with cardiovascular disease. Many mechanisms can pave this path, as dysregulation of cardiovascular systems has been linked to cardiovascular disease through various mechanisms and interruptions of healthy regulatory patterns can arise from various sources. Emotional reactivity has been shown to play a key role.

Emotional Reactivity

Coronary heart disease (CHD), a type of cardiovascular disease, is the single leading cause of death in America today, causing 466,101 deaths in 1997. About every 29 seconds an American will suffer a coronary event, and about every minute someone will die from one (A.H.A., 1999). Research has shown that increased emotional reactivity to individual stressors leads to higher rates of coronary heart disease (CHD). For example, people in lower socioeconomic divisions and the elderly exhibit patterns of higher CHD incidence rates (Manuck & Krantz, 1986; Matthews et al., 1992; Rosenman et al., 1975; Rosenman, Brand, Sholtz, & Friedman, 1976; Rosenman et al., 1966; U.S.D.H.H.S., 1988). Also, many CHD prevention efforts have not equally benefited men, as African-American men of lower social class have not realized the same benefit from these programs as other men; thus, public health programs need to be better targeted to these men (Barnett, 2000). Some have also noted that along with stress, reactivity may contribute to increased rates of cardiovascular disease and CHD in men (Krantz & Raisen, 1988). Cardiovascular responsivity patterns (greater heart rate and SBP responses) have often been found in men more than women (Harbin, 1989), which corresponds with higher CHD incidence in men.

In studying the etiology of emotional reactivity and its relationship to cardiovascular disease, several mechanisms have been proposed. One possible mechanism is that repeated excessive cardiovascular reactions to behavioral stressors promote arterial "injury" through "hemodynamic forces such as turbulence and sheer stress" (Manuck, Kaplan, & Clarkson, 1983). Behavioral factors can influence the development of CHD act through the cardiovascular and/or endocrine correlates of "sympathetic-adrenal-medullary and pituitary-adrenal-cortical activation" (Manuck & Krantz, 1986), which can cause blood vessel sheer stress. Recent evidence supports the concept that individual differences in behaviorally-induced reactivity corresponds with a higher incidence of CHD (Manuck & Krantz, 1986). Emotional reactivity has been linked to cardiovascular disease in numerous studies of adults (Kubany, Gino, Denny, & Torigoe, 1994; Lee & Cameron, 1986-87) and children also (Matthews, Woodall, & Allen, 1993; Treiber et al., 1989). It has been found to be one of the most predictive measures for future cardiovascular problems.

Biochemical forces can also damage coronary arteries through the increased release of certain endocrine substances (such as the catecholamines and corticosteroids) to damaging levels during stressful events. If these physiologic mechanisms are partly responsible for the development of CHD, then the greatest damage would be incurred among highly reactive individuals, those who show the greatest

psychophysiologic responsivity (Manuck & Krantz, 1986). Recent research results lend support to the idea that certain cardiovascular reactive patterns are more likely with individuals who have particular neural networks *and* concurring circumstances. That is to say, a reactive individual may differ from a non-reactive individual in cerebral systems involved, which may influence concurrent activation or inhibition of cardiovascular reactivity. Accordingly, Julius, Weder, and Hinderliter (1986) looked at the link between behaviorally induced blood pressure (in juveniles) and hypertension across studies and theorized that some forms of hypertension may result from "abnormal central processing of environmental stimuli" (Julius, Weder, & Hinderliter, 1986). As heightened reactivity can lead to high blood pressure and essential hypertension, this has been shown to be a risk factor centrally involved in the causation of atherosclerotic disease and its multiple clinical manifestations, especially coronary heart disease (Stamler, 1992). Therefore, given a specific circumstance (e.g., a perceived threat, such as confrontation or harassment), some individuals seem to overreact according to the functioning of their sensory-emotional regulatory system (neural network), both through observable behavior, cardiovascular reactivity patterns, and through excess release of damaging endocrine substances.

In a healthy cardiovascular regulatory pattern, heart rate and blood pressure are inversely related through the baroreceptor reflex (Rushmer, 1976). That is to say, as blood pressure increases as a function of a stressor, heart rate should decrease, compensating for some of the extra pressure effects in the vasculature. Needless to say, neural mechanisms involved in differential levels of systolic blood pressure and diastolic blood pressure should be considered with other risk factors (Guyton, Coleman, & Granger, 1972), especially when this mechanism fails (i.e., blood pressure rises as heart rate increases). Furthermore, when considering systolic blood pressure and diastolic blood pressure regulation, as mediated by adrenal-medullary and parasympathetic systems, they cannot be viewed as orthogonal systems, but rather as complementary, co-acting systems.

Hostility and (Over)Reactivity

Overall, sex differences in aggression, or hostility, have remained remarkably stable, when changes in methodology are controlled (Knight, Fabes, & Higgins, 1996). Men evidence more aggression than women, and these differences in aggression may be a manifestation of different underlying functional emotional cerebral networks in men and women (that then contribute to differential rates of coronary heart disease and other types of cardiac risk). Through a neuropsychological conceptualization, different (functional and dysfunctional) emotional regulatory patterns seen in men and women can be better appreciated, along with the health consequences of the corresponding cardiovascular reactivity patterns attributable to each sex.

Many of the measures that have been used in studies of emotional reactivity have been physiological measurements of cardiovascular reactivity, skin conductance, and skin temperature. Early research on emotion noted profound changes in cardiovascular reactivity during fight or flight responses in animals (Cannon, 1929; Malmo, 1959). Later, the idea of stimulus specificity of emotions, specifically cardiovascular specificity, was illustrated through a classic experiment where heart rate, finger temperature, or skin conductance distinguished between emotions (Ekman et al., 1983). Accordingly, substantial empirical work then directly examined central nervous system (CNS) activity associated with emotion, which led to a considerable literature on the CNS substrates of emotion. The increased attention to the central substrates of emotion in adults has been catalyzed by observations of the affective consequences of localized brain damage and also the studies performed during the past three decades on the effects of experimentally produced lesions in animals on affective behavior (Panksepp, 1992). These studies have emphasized that different brain systems regulate different emotions and they also highlight the specific role that different neurotransmitters play in the production and modulation of a range of emotional behavior. Nevertheless, multiple cerebral systems with complex neural processes are involved in emotion management; thus, researchers still rely on behavioral evidence to better understand these distinctive systems (Thayer, 1989). For this reason, emotions can be better understood when seen from a neuropsychological perspective.

The history of neuropsychological research into hostile emotional regulation and expression is significant. In 1937, Klüver and Bucy described a pattern of behaviors that resulted from the bilateral

removal of the temporal area in monkeys. After the operation, the monkeys showed changes in visual perception, behavior (sudden attacks of rage), and memory disturbances (Klüver & Bucy, 1937; Klüver & Bucy, 1938). At that same time, in 1937, Papez suggested that the medial zones of the temporal region are related to the primary mechanisms of emotion (Papez, 1995). Likewise, electrical stimulation of the amygdala was found to lead to vigorous attacks in monkeys, while lesioning the amygdala in previously aggressive monkeys lead to pacification and placidity (Rosvold, Mirsky, & Pribram, 1954). Research conducted on electrical stimulation of different brain areas and corresponding, hostile behavioral patterns was reviewed by Kalat (1995), who found that stimulation of the hypothalamus, amygdala, and brain stem was found to elicit an affective attack in the cat. As a result of such stimulation, the cat hissed, growled, arched its back, and bared its teeth. Often the cat would direct its attack toward a close target, but sometimes electrical stimulation elicited only the facial expressions without the remainder of the attack sequence (Delgado, 1981; Siegel & Pott, 1988). Also, lateralized effects have been found, as stimulation of the right amygdaloid area elicited rage behavior in the cat (Shaikh, Schubert, & Siegel, 1994; Shaikh, Steinberg, & Siegel, 1993). Similarly, as subjects were studied who had cortical tumors removed, ventral frontal or temporoparietal tumors showed significant negative mood changes postoperatively, and only frontal paralimbic lesions had negative effects on mood (Irle, Peper, Wowra, & Kunze, 1994).

Subsequently, the prefrontal cortex was implicated in emotional regulation as it has extensive connections with the limbic system (Luria, 1962). Consequently, the medial and orbital surfaces of the frontal lobes were described as part of a single "frontal-limbic" system (Pribram, 1969a; Pribram, 1969b; Pribram, 1975). Similarly, Nauta (1971) described this area of the prefrontal cortex as the "cortical modulator of the limbic system" (Nauta, 1971). Therefore, the frontal lobes were hypothesized to regulate overall limbic system activity (Pribram & Luria, 1973). More importantly, however, the orbitofrontal cortex was also seen as an inhibitor over anterior/temporal areas. In support of this idea, damage to the right orbitofrontal region yielded aggressive behavior, whereas stimulation of the right orbitofrontal region produced passive responses, often accompanied by flat affect expression. Activation of the frontal lobe, particularly the orbital frontal cortex, was been found to lower or inhibit the expression of hostility (Butter, Snyder, & McDonald, 1970). The right orbital-frontal cortex has extensive interconnections with the right anterior temporal area, especially the amygdala. Heilman, Bowers and Valenstein (1993) postulated that the right orbitofrontal region and the right anterior temporal region interact, resulting in emotional regulation and expression, which gives rise to *emotional learning*. A stable interaction yields more appropriate emotional regulation. However, if the interaction affords relative activation of either the orbital-frontal cortex or the anterior temporal area then emotional dysregulation is likely, as emotions and arousal are more prominent in the right hemisphere; (Heilman & Bowers, 1990). While testing this system using quantitative electroencephalographic (QEEG) techniques, Everhart and Harrison (1995) found decreased orbital-frontal activation with corresponding increased right temporal beta activation in a homicidal patient. Thus, negative, hostile emotions were found to be expressed following deactivation of the right orbital-frontal region with concurrent increased activation of the right temporal region (Everhart & Harrison, 1995, Fall). This finding has also been supported by previous research supporting the role of the orbital-frontal cortex in hostility inhibition (Krynicki, 1978), as well as other research using QEEG techniques with hostile individuals finding decreased beta activation of the right frontal region relative to the left temporal region (Demaree & Harrison, 1996). Negative health consequences often follow dysfunction in these emotional regulatory systems, especially as hostile reactivity leads to unhealthy cardiovascular responding (Kalat, 1995), while cerebral areas act in concert as a functional cerebral systems.

Neuropsychological Relationship of Hostility and Cardiovascular Regulation: Frontotemporal Systems

Various research projects Virginia Tech have investigated this very idea- of interacting dynamic functional cerebral systems which give rise to an unhealthy cardiovascular response (with an emphasis on hostility and laterality). Hence, possible neuroanatomical mechanisms of hostility and associated cardiovascular reactivity patterns have been assessed through various sensory modalities. Furthermore, investigating areas in the left and right hemisphere, specifically frontal areas vs. temporal areas, has helped shape the direction of research inquiry at Virginia Tech's lab (and the common theme uniting research projects). Furthermore, components, or individual parts of these systems have been found to be dynamic (i.e., subject to priming or interference from other areas). The effects of hostility and its interconnections with different systems throughout the brain have been studied, with an emphasis on possible health consequences and/or intervention strategies. Also, neuropsychological features of high-hostile individuals have been studied, as hostility has been analyzed as a dysfunctional sensory activity (altered negative emotional perception act) in right cerebral systems (Demaree & Harrison, 1996). The research has supported previous neuropsychological theories and has been instrumental in furthering the understanding of the possible functional neuroanatomical bases of emotional/hostile reactivity and cardiovascular regulation.

For example, high-hostile men have been found to be less accurate than low-hostile men in the assessment of happy, angry, and neutral faces presented tachistoscopically to their left visual field (right hemisphere) (Herridge, 1996). However, high-hostile men were also more accurate than low-hostile men when assessing happy and angry facial configurations presented to their right visual field (left hemisphere). Thus, high-hostile men were found to have more lateralized cerebral functionality. It is unclear whether this same pattern would be found between "normal" men and "normal" women, respectively, as women have traditionally shown less lateralized function than men. Next, greater right cerebral activation to stress was found among high-hostile men during a dichotic listening task, as enhanced intention to the left ear was found (Demaree & Harrison, 1997b). Physiological reactivity (SBP, DBP, and HR) corresponding to dichotic listening changes were also found. A significant finding, although not the primary finding, was that low-hostile individuals had significantly more left temporal cerebral activation to stress and also evidenced minimal cardiovascular reactivity to the cold pressor stressor; raising the possibility that both hemispheres are involved in cardiovascular regulation. Additional pronounced laterality effects among hostile men were found when Herridge, Harrison, and Demaree (1997) investigated skin conductance between low and high-hostile groups and found significant left hand skin conductance increases in men while making angry facial configurations. Making angry faces yielded slow habituation of skin conductance at the left hand in hostiles and slow habituation at the right hand in low hostiles, and making faces portraying happy affect increased conductance at the right hand (Herridge et al., 1997).

Cerebral Quadrant Theory and Cardiovascular Control

Thus, visual, auditory, and somatosensory differences were noted among high-hostile and low-hostile individuals. Hostility can be viewed to some extent as a dysfunctional sensory task that can enhance or interfere with normal sensory processing. Follow-up research extended previous research findings from a level of secondary implication of brain activation (skin response, heart rate, blood pressure, auditory processing, and visual processing) to a level of primary visualization of brain activation through the use of real-time, QEEG (topographical brain-mapping). Demaree, Harrison, and Rhodes found that low hostile men show decreased left frontal activation and increased left anterior temporal activation. They hypothesized that the left anterior temporal region of the brain may help regulate parasympathetic nervous system activation, just as the right anterior temporal region regulates sympathetic nervous system activation (Demaree et al., in press). Furthermore, just as the right orbito-frontal area inhibits the right anterior temporal area (sympathetic), it is possible that the left orbito-frontal area inhibits the left anterior temporal area (parasympathetic). Thus, low-hostile men may have under-inhibited parasympathetic nervous system activation (i.e., increased parasympathetic activation), which would decrease cardiovascular reactivity and CHD risk. This pattern (in low-hostile men) may be similar

to women, who normally show decreased cardiovascular reactivity, increased emotional regulation, and decreased CHD risk.

Thus, similar to findings of the neuropsychology of emotion, neural regulation of the cardiovascular system is better understood in terms of four quadrants. This idea follows laterality findings mentioned previously and also the research at Virginia Tech that has suggested evidence for cerebral systems within each hemisphere that may be involved in the activation and/or inhibition of sympathetic and parasympathetic activity. Specifically, activity in the right anterior temporal area is thought to produce sympathetic activation (while the right orbito-frontal area inhibits activity in this area), and activity in the left anterior temporal area is thought to trigger parasympathetic activation (while the left orbito-frontal area inhibits activity in this area). Thus, these cerebral areas may be differentially involved in cardiovascular regulation (Demaree & Harrison, 1997b; Demaree et al., in press; Emerson & Harrison, 1990; Higgins, 1999; Williamson, 1999).

A Proposed Model of Three Principal Functional Units of Cardiovascular Regulation

Consequently, a concept is proposed that takes the most well-established neuropsychological theories (laterality and systems theories) and reconceptualizes them through a merger and advancement of ideas (i.e., through the development of quadrant theory, briefly described above) to a proposed new level in understanding neuropsychological patterns of cardiovascular regulation. This new theory is described in terms of three principal functional units of cardiovascular regulation, similar in design to the *three principal functional units of the brain* first described by A. R. Luria during the formative years in neuropsychology (Luria, 1973). These basic, fundamental theories have been well supported since their introduction, reaching a general consensus of acceptance. It is argued that from the position of this foundation that neuropsychological sex differences in cardiovascular regulation can be better understood. These theories are well-established, but the application of them in a neuropsychological model of sex differences in cardiovascular regulation is a new development. Thus, these proposed three neuropsychological principal functional units (PFU; see Figure 4) of cardiovascular regulation may be able to better capture previous concepts of asymmetry in cardiovascular regulation and control, cerebral systems or quadrants, and newer research detailing inhibitory systems and mechanisms across areas. Furthermore, it is argued that through this model sex differences in cardiovascular regulation and disease can be better depicted understanding of some forms of cardiovascular disease etiology can be enhanced.

These three principal functional units of cardiovascular regulation are somewhat similar to a concept proposed by Derryberry and Tucker (1992), who proposed that neural mechanisms of emotion are distributed across diverse areas: the brainstem, limbic, paralimbic, and neocortical regions. Further, they postulated that connections among these levels are related to types of emotional processes, with ascending and descending influences allowing for a modulation of cortical pathways and flexibility of emotional responding (Derryberry & Tucker, 1992). Also, following the design of the quadrant model, above, it is likely that each hemisphere of the brain could be divided into three functional units (i.e., both the left and right hemisphere have three principal functional units). Nevertheless, these would differ in each hemisphere (e.g., left temporoparietal areas process language and right temporoparietal areas process spatial/emotional information). The concept of the hemispheres having different roles is similar to one proposed detailing the unique role each hemisphere in regulating emotion, as the right and left hemisphere operate on different principles of neurophysiological arousal (Tucker, Vannatta, & Rothlind, 1990). Furthermore, these right and left principal functional subunits would likely differ in men and women. Support for components of this model are well-established. There is extensive support that cerebellar regions are intimately involved in general tone and arousal, that sensory projections are involved in the processing of sensory and emotional stimuli, and that frontal systems are involved in the integration and organization of multiple tasks. Thus, the three principal functional units of cardiovascular regulation can be more fully described in these terms.

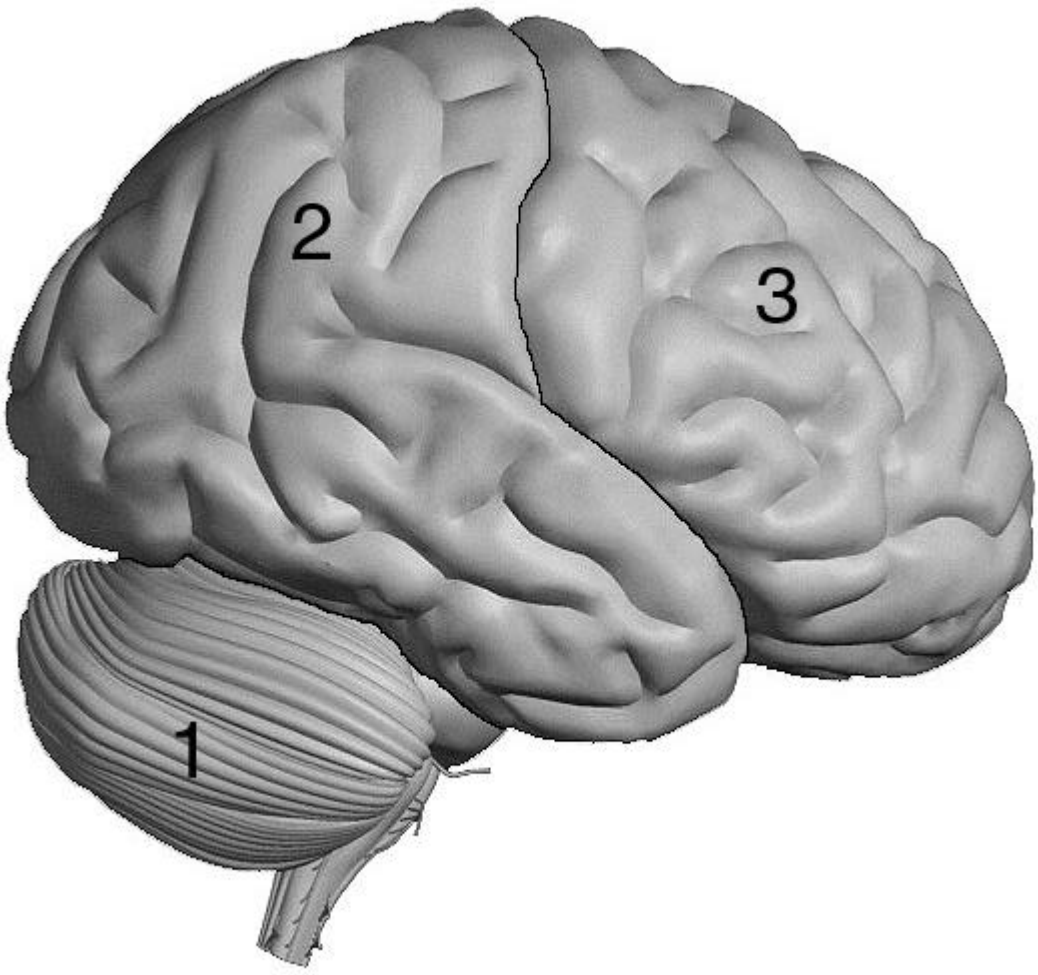


Figure 4. The three principal functional cerebral units proposed to be responsible for cardiovascular regulation.

Unit One: General Cardiovascular Tone and Arousal

Unit one is proposed to be responsible for maintaining general cardiovascular tone and arousal, through the involvement of the cerebellum and brain stem. A comparable system of emotional regulation where primitive emotional processes aid in regulating higher order functions has been proposed (Tucker et al., 1990), which can aid in understanding how primitive processes can have such significant, global effects. Components of unit one have been described by Porges, who has proposed a vagal circuit of emotion (Porges, Doussard-Roosevelt, & Maiti, 1994), where the emphasis is on lateralized (right) brainstem structures and the vagus in emotional regulation. In fact, his idea of measuring base levels of cardiac tone and reactivity work well in conceptualizing the nature of principal functional unit one. A vagal complex, such as the one he proposes, with basic levels of attention and emotion or arousal (Porges, 1995) can be incorporated into the design of this unit, nevertheless, it is estimated that there is more cortical involvement in regulating this unit (than he proposes). Dysfunction in this unit would likely produce stuporous cardiac activity, difficulties in maintaining cardiac tone in various bodily positions (orthostatic hypertension), and broad dysfunction in all areas of cardiac responding (creating the appearance of dysfunction in units two and three also).

Unit Two: Sensory Analysis and Cardiovascular Reactivity

Unit two is proposed to be responsible for the reception, comprehension, and analysis of sensory events, environmental threats, and emotional provocation/cues. It includes the parietal, temporal, and occipital lobes of the brain. It can be affected by priming and/or interference effects, and it responds to noise, visual events, and threatening or alerting cues. Dysfunction in these posterior brain regions may lead to comprehension errors, such as overestimating a threat, pain stimulus, or facial expression. Hostility, viewed within this framework, is seen as a dysfunctional interpretation of the actions of others.

Right hemisphere activation for autonomic responses, more than cognitive factors, suggests that the physiological response system, rather than perceptual/cognitive systems, is at the center of the right hemisphere's superiority for emotion (Spence, Shapiro, & Zaidel, 1996). Thus, the right hemisphere has often been found to be dominant for many emotional functions. In cortical areas involved in emotion regulation, "there are two important, complementary, sensorilimbic connective pathways: a dorsal system critical for surveillance, attention, and arousal and a ventral system specialized for stimulus identification, learning, and emotional response" (Bear, 1983). Through hemisphere specialization, lateral asymmetries in these sensorilimbic connections may arise. In fact, a neuropsychological paradigm may be a good explanatory model for abnormal psychology, as hemispheric asymmetries are often found between normal and psychopathological groups (Tucker, 1984). Also, arousal is an important issue to consider with many behavioral disorders, and recognition of (over- or under-) arousal deficits is key to the understanding of many behavioral problems. Consequently, it is argued that left-right asymmetrical function is not only related to perceptual and cognitive functions subserved by the cortical hemispheres, but also subcortical systems and peripheral measures of physiology, including autonomic and endocrine system events (Hugdahl, 1996). For example, hypertension is greatly influenced by posterior/sensory stimuli and corresponding psychological reactions to factors and can arise from "a state of abnormal central integration of autonomic cardiovascular control" (Julius et al., 1986).

Unit Three: Executive Organization of Complex Cardiovascular Responses

Unit three is proposed to be involved in the executive organization, control, and planning of cardiovascular response, as it responds to cardiovascular needs according to task demands and regulates cardiovascular functioning secondary to other areas. Dual tasks, or multi-tasking would likely recruit activity in this unit. It involves mainly frontal lobe functioning. Dysfunction in this unit may produce cardiac lability, non-fluency in cardiac transitions in response to stress, and perseverations in cardiac patterns. Furthermore, the executive action, preparation, planning, and organization of cardiac responses would be affected. Connections between frontal lobe regions, especially orbitofrontal areas, limbic structures, and sensorimotor areas are dense and often reciprocal (Figure 5). These systems can influence activity levels, through inhibitory or other mechanisms.

Units two and three are the primary units when considering sex differences, as they are proposed to be the most important units in understanding the proposed neuropsychological model of sex differences

in cardiovascular regulation. Neuropsychological sex differences have been found in the analysis of emotional cues (visual and auditory), verbal information, spatial tasks, motor tasks, and others, implicating core differences in neuropsychological functioning in occipital, parietal, temporal, and also frontal cerebral areas. Thus, a conceptualization of functional cardiovascular units two and three is key in order to understand their relationship with these cortical areas. That is to say, as sex differences have been found in various neuropsychological functional systems, it is important to discover the relationship between these systems and cardiovascular regulation to investigate the influence of sex effects.

Sex differences have also been found in cardiovascular reactivity through disparate increments in systolic blood pressure, diastolic blood pressure, and heart rate changes. As sex differences have been established in brain asymmetry and functional system design, corresponding to sex differences in sensory and cognitive factors, this experiment questions whether sex differences in neural system structural design and functionality contribute to sex differences in cardiovascular reactivity, regulation, and disease.

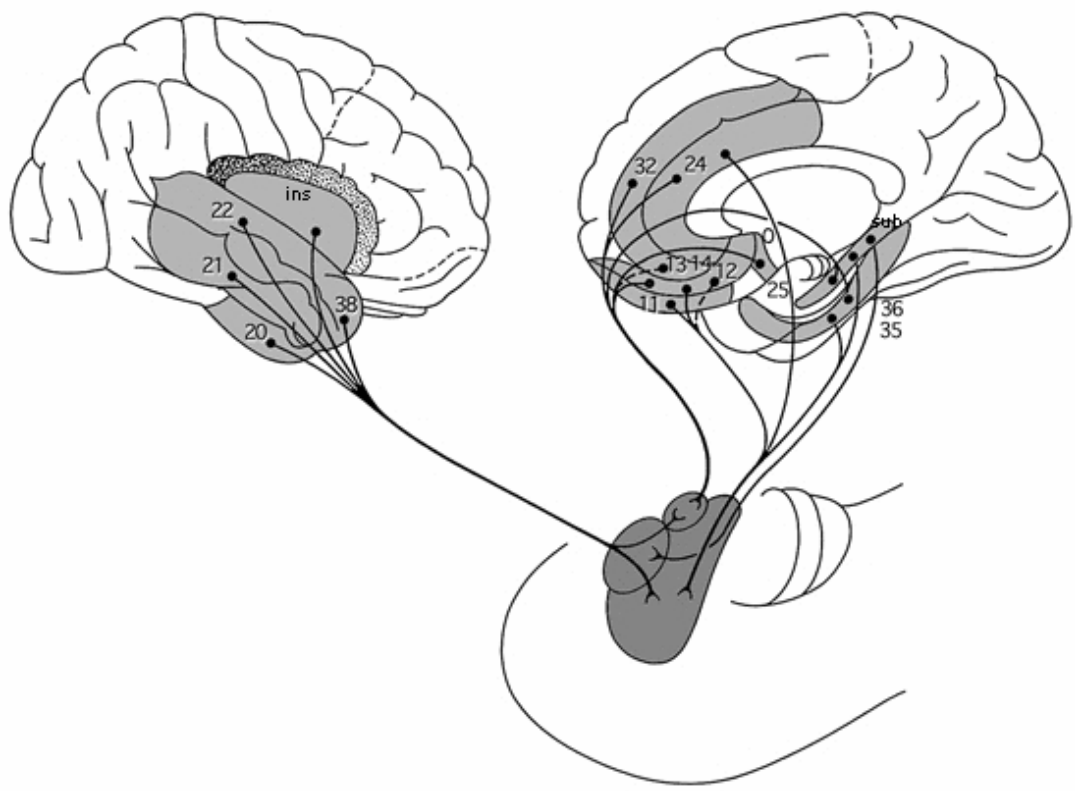
A Neuropsychological Model of Sex Differences in Cardiovascular Regulation and Disease

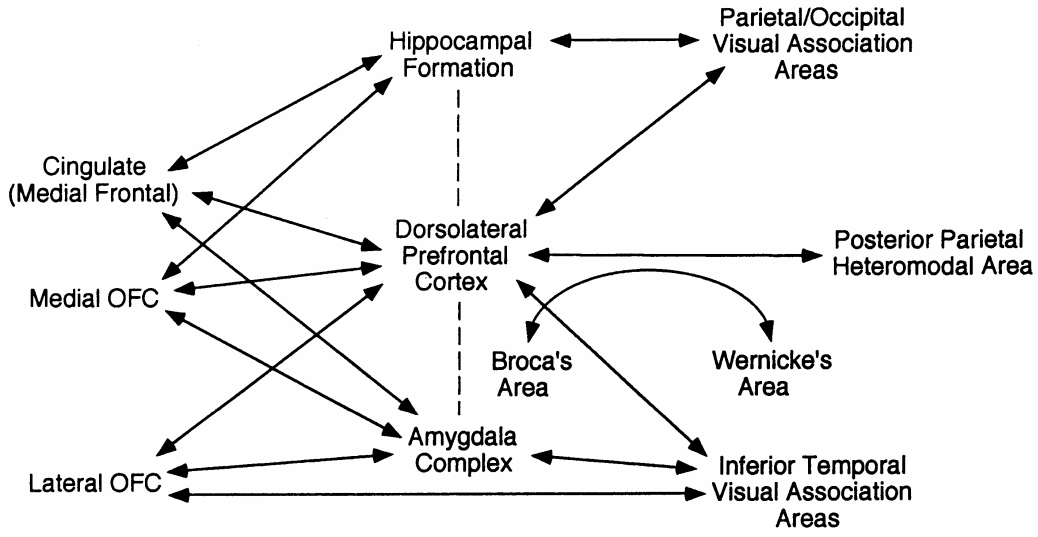
Some sex differences in cardiovascular regulation and reactivity can be reconceptualized, then, within a neuropsychological model, which might better account for some sex differences in cardiovascular disease. It is proposed that a direct function of sex differences in cerebral systems may account for sex differences in cardiovascular regulation and some cardiovascular disease forms. The research designs in this experiment were designed to test this model. If this model advances current understanding of cardiovascular disease, then classic findings in cardiovascular reactivity research may need to be revisited and reinterpreted, and future research directions may need to be carefully planned. To date, neuropsychological research on emotion with related cardiovascular indications has suggested that there are sex differences in emotion processing, cardiovascular reactivity, and related hostility levels. Consequently, there are sex differences in cardiovascular disease. If there is a common neuropsychological mechanism underlying these various concepts/differences, then discovery of the functional units involved would allow for increased precision in predicting cardiovascular disease risk. Furthermore, prevention efforts might allow for neurotherapy approaches to cardiovascular disease. Thus, a dynamic functional systems model might better account for sex differences in cardiac regulation to uncover dynamic factors in cardiovascular management.

Current Methods for Assessing Dynamic Functional Cerebral Systems

Routes to cortical processing are not as fixed as once thought. In fact, the dynamic nature of the brain is not completely understood. Even concepts as straightforward as laterality (left vs. right) are being redefined (as neither left nor right hemisphere areas act alone). It is not entirely accurate to simply say, for instance, that speech production occurs solely in one cortical area, as proximal areas can interfere with speech production. Thus, it is more prudent to say that speech production is a product of the interplay within a cerebral system composed of a primary production area and various accommodating or disruptive areas which aid or interfere with the production of speech. Another example of this might emphasize the inclusion of both right anterior temporal and frontal areas as responsible for cortico-autonomic control, as right temporal areas are inhibited by right frontal areas. Thus, the system should first be acknowledged, and then further research can aid in understanding individual areas' specific roles in the system. Nevertheless, systems can be quite complex, with various functions ascribed to the same structures which affect other areas via disparate pathways (Tucker, Luu, & Pribram, 1995).

Dual task priming and interference effects have been used to demonstrate the idea of dynamic cerebral functioning (Van Strien & Heijt, 1995) and has been helpful to find specific cerebral systems' influences on cardioregulatory dysfunction. This idea is also supported by fundamental research in arousal theory (Duffy, 1962; Easterbrooks, 1959; Harrison & Pavlik, 1983; Hebb, 1955; Lindsley, 1951), which suggests that increased arousal is associated with improved task proficiency up to an optimal level, at which point further increases in arousal may impair performance. This is similar to the idea of functional cerebral space, where enhancement or interference of one activity can arise by virtue of a concurrent activity (Filimonov, 1940; Kinsbourne, 1978; Kinsbourne & Cook, 1971).





OFC: Orbitofrontal Cortex.

Figure 5. Schematic diagrams of principal corticocortical connections within frontal lobe regions (including primary limbic connections) and between posterior sensory cortices and frontal lobe regions. Double-head arrows indicate reciprocity, and dashed lines indicate sparse connections (Kaufer & Lewis, 1999).

That is to say, functionally specialized cerebral areas are interconnected in varying degrees, and the level of interconnection dictates the level of influence over other areas (Kinsbourne, 1982).

The right and left hemispheres seem to have a dynamic relationship with one another, and each can influence the other's activity. Some studies on emotion have attempted to understand this relationships through the employment of a procedure known as the WADA test, in which sodium amytal is injected into the carotid artery. This is also done during neurosurgical procedures in order to aid the physician in identifying (and sparing) the language area in the brain. The sodium amytal temporarily arrests most functioning in the hemisphere of the brain to which the vascular supply travels (see Figure 6). Early studies of unilateral injection of sodium amytal into the left carotid artery (which supplies blood to the left hemisphere) produced severe emotional reactions (crying, pessimism, indignity or despair, fear, negative thoughts), often with the patient simultaneously reporting no apparent *reason* for such feelings (Fox, 1994). Unilateral injection into the right artery (to right hemisphere) elicited quite a different response (euphoria, smiling, optimism, an overall sense of well-being). Dynamic functional cerebral laterality is evidenced when considering the processes underlying these positive or negative emotional reactions. This research suggests that emotional changes are the result of the release of one cerebral hemisphere from the "contralateral inhibitory influences of the other." Thus, when not counterbalanced, the right cerebral hemisphere would produce dysphoria (negative mood) and the left cerebral hemisphere would produce euphoria, or positive mood (Fox, 1994). Viewed as such, emotions can be viewed as not just a result of the independent functioning or activity of one hemisphere, but rather, they arise from the dynamic interplay of the cerebral hemispheres. Such dynamic influences should be considered in order to appreciate sex differences in neural emotion networks and differing rates of emotional reactivity.

Thus, as cerebral areas interact with one another, these interacting areas can be thought of as functional cerebral systems. The interactions, themselves, support the concept of dynamic functional cerebral systems and the concept of dynamic functional cerebral laterality found in many recent research investigations, especially as the nature of the interactions change according to sex and other psychological variables (e.g., hostility, depression, or anxiety). Thus, this concept is elaborate, but can become elegant through research investigations and findings. That is to say, to correctly describe functional cerebral systems, all primary areas, accommodating areas and inhibitory areas should be included for maximum accuracy. For instance, the dynamic, sometimes bilateral (in women) functional cerebral systems for speech can be better understood within this framework. Also, cerebral regulation of cardiovascular function can be better explained after first considering this theoretical perspective. Consequently, is important to conceptualize cardiovascular regulation in terms of principal functional units, rather than solely in a left hemisphere, right hemisphere model.

Dynamic factors in changing laterality should be considered, as these factors often lead to sex differences in cerebral laterality (or asymmetry). These dynamic factors may help explain sex differences in cardiovascular regulation (i.e., functional cerebral laterality and cardiovascular regulation), specifically, dynamic neuropsychological sex differences in right and left anterior temporal and orbito-frontal cerebral regions may help explain sex differences in cardiovascular regulation. Thus, a new neuropsychological model explained through the three principal functional neuropsychological units of cardiovascular regulation introduced above can offer a better conceptualization of sex differences in cardiovascular regulation and disease. Therefore, a synthesis of the above neuropsychological theories that best captures the common components of each and weighs the significance of differing ideas, or a parsimonious model of how neuropsychological sex differences affect cardiovascular regulation, offers a new avenue and perspective for the investigation of cardiovascular regulation and disease differences in men and women.

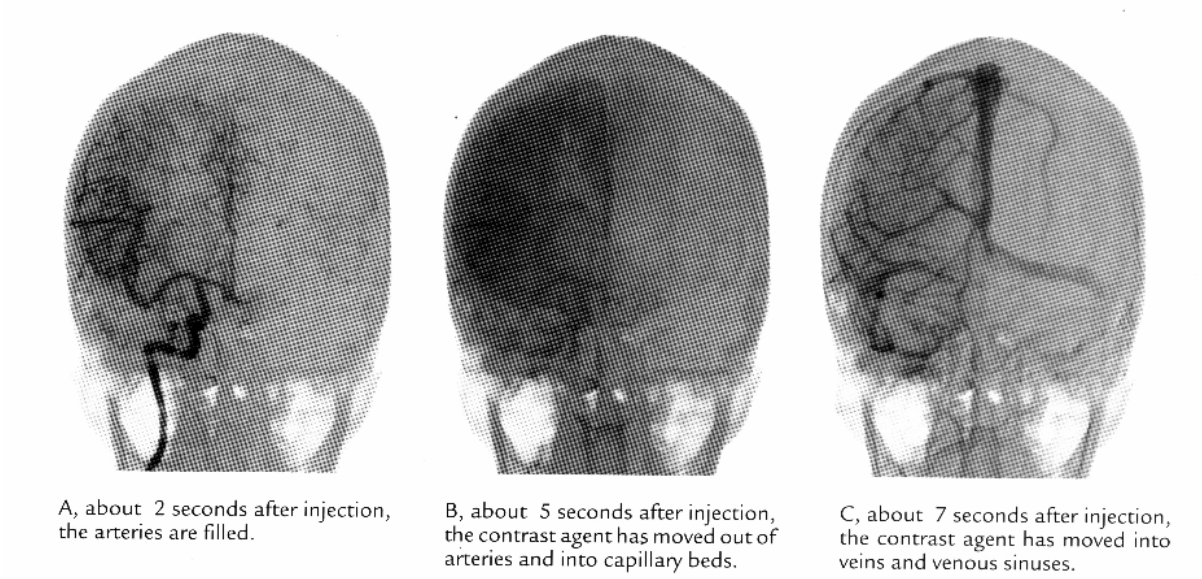


Figure 6. Asymmetry of cerebral vasculature is seen as movement of contrast material is shown, administered via injection of the right internal carotid artery (Nolte & Angevine, 1995).

Preliminary Dynamic Mechanisms Leading to Sex Differences in Cardiovascular Regulation

Some forms of dynamic cerebral functioning have been proposed to occur through the influence of sex hormones, acting as neurochemical mediators on cardiovascular variables. Khaw and Barrett-Connor (1988) investigated blood pressure and endogenous testosterone levels in men. They found that men with hypertension had significantly lower testosterone levels than non-hypertensives. This association was present throughout the range of blood pressures and testosterone levels, with a stepwise decrease in mean systolic blood pressure and diastolic blood pressure "per increasing quartile of testosterone" (Khaw & Barrett-Connor, 1988). Just as significant was the finding that no other hormone nor sex hormone-binding globulin showed a consistent relationship with blood pressure.

Another important consideration is the changing dynamic of neuropsychological mechanisms in men and women. For example, age has been found to be a primary risk factor in cardiovascular disease; however, aging impacts women much more than men, in regard to cardiovascular disease risk. As men and women age, the cardiovascular disease gap begins to close, with women experiencing "gains" in cardiovascular disease incidence. As women age, they begin to experience increased rates of cardiovascular disease incidence. Concurrently, research has shown that women begin to show increased laterality effects as they age. It would be helpful to find how these two developments are related. Thus, previous research assessing the right hemi-aging hypothesis may also be a valuable tool in understanding these age-related changes in laterality, especially when analyzing sex differences in hemi-aging. Neuropsychological research is able to investigate these underlying mechanisms. Research questions raised by this model include the investigation of the factors contributing to the changing incidence of cardiovascular disease according to sex across the lifespan. Also, the estimation of cardiovascular disease threat in men and women must be reassessed to ensure accuracy according to current neuropsychological models of laterality, aging, and dynamic changes across the lifespan of men and women.

Demonstrated dynamic effects of cognitive tasks on cardiovascular factors has been found as men, but not women, were found to experience significant decreases in systolic blood pressure (Figure 7) following a dichotic listening test (Higgins, 1999). This finding led to this follow-up investigation, as regulation of parasympathetic activity may be more asymmetrically organized in men than women, and that activation of the left hemisphere in men may increase regulation of the parasympathetic control of the heart (with diastolic blood pressure increases possibly mediated by parasympathetic mechanisms). Further, for both sexes, groups that focused on dichotic listening stimuli at the right ear (left hemisphere) experienced the lowest diastolic blood pressure, and for men only, the focus right group experienced the lowest systolic blood pressure. Finally, heart rate decreased significantly following the cold pressor for women only. A history of increased cardiovascular reactivity to stress has been shown in men, primarily measured through systolic blood pressure. However, as sex differences continue to be explored, future research should include measures of diastolic blood pressure and systolic blood pressure (Higgins, 1999; Lawler, Wilcox, & Anderson, 1995). Otherwise, sex differences in cardiovascular reactivity may be partial to men or women if considering only systolic blood pressure or diastolic blood pressure. A more detailed psychophysiological analysis of SBP versus DBP is encouraged.

Considering Hostility in the Current Model and Experiment

As sex differences have been shown to exist in various language and spatial processing tasks, these differences may represent fundamental differences in sensory processing in posterior cerebral areas (temporal, parietal, and occipital areas). Therefore, it is important to consider that many sex differences have already been shown to exist in principal functional unit two of cardiovascular regulation. Furthermore, as sensory processing tasks have uncovered sex differences in cerebral functionality, then differences in basic sensory processing might partially explain corresponding sex differences in hostility prevalence and related cardiovascular indices (leading to differences in cardiovascular regulation and disease). That is to say, hostility, viewed as a dysfunctional sensory task (overestimation of threat conditions, caustic interpretation of others' actions) may lead to these related cerebral processing conditions (as they are also functions of left and right posterior cerebral areas).

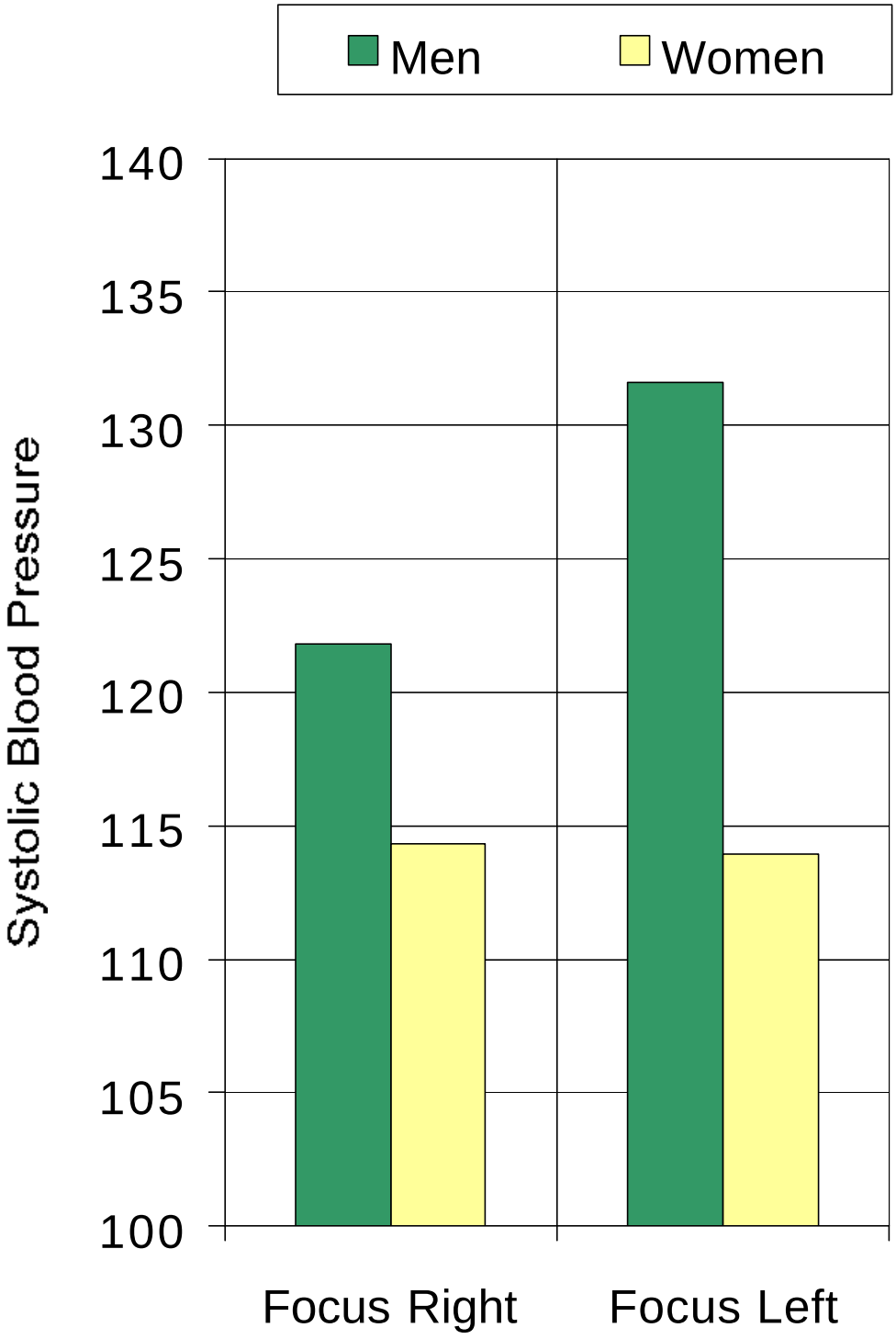


Figure 7. Sex differences in systolic blood pressure for focus (intention) groups to a dichotic listening task (Sex by Focus Interaction Effect on Systolic Blood Pressure).

Hostility, as a construct, "fits" into principal functional unit two, as it can be viewed as a sensory task gone awry. That is to say, although "hostility" has previously been used to describe negative affect, cognition, and behavior patterns, in this experiment (and others), this term is used to describe a construct with meaning more specific to cognitive factors. Miller (1996) described hostility as a cognitive phenomenon consisting of "negative beliefs about and attitudes toward others, including cynicism, mistrust, and denigration. Cynicism refers to the belief that others are motivated by selfish concerns, and mistrust is the often co-occurring expectation that others are likely to be provoking and hurtful" (p. 323). Smith (1994) previously stated that hostility is a general trait connoting a "devaluation of the worth and motives of others, an expectation that others are likely sources of wrongdoing, a relational view of being in opposition toward others" (p. 26). Thus, others are perceived as being untrustworthy, harmful, and even dangerous, notwithstanding the lack of evidence to support such a view. By including neuropsychological tasks that target these posterior sensory areas, priming and/or interference effects on frontal organization areas (unit three) and cardiovascular regulation can be investigated.

Summary and Rationale for Current Experiment

As men and women appear to differ in the degree to which traditional neuropsychological functions are lateralized (language functions, spatial abilities, emotional regulation, and motor functions), it seems important to consider a merging of the literature on neuropsychological sex differences (with an admitted emphasis on laterality) with the literature on the neuropsychology of cardiovascular regulation. Differences in functional cerebral systems must be considered when studying sex differences in cardiovascular disease risk, as sex differences may exist in the principal functional neuropsychological units cardiovascular regulation, and when cerebral dysfunction is present, some forms of *cardiovascular disease* may be better conceptualized as *neuropsychological disorders*. Thus, there may be disadvantages to having cerebral subsystems (such as emotion processing and cardiovascular sympathetic activity) lateralized to the same hemisphere, as they may be "crowded" in one hemisphere (Geschwind & Galaburda, 1987; Hellige, 1990; O'Boyle & Hellige, 1989) and they may be influenced by one another's activity (Kinsbourne, 1978). As such, these interconnected functional cerebral systems have varying levels of influence over other areas (Kinsbourne, 1982). Thus, it is important to test this model, to determine the level of interconnectedness, interference, and interplay between these systems and to discover if sex differences exist in such. If so, it should prompt reconsideration, reconceptualization, and reinterpretation of previous cardiovascular disease risk data. Studies of sex differences in cardiovascular disease have tended to focus on sex differences in stress, diet, hazardous health behaviors such as drinking and smoking, genetics/heritability, and other factors. However, if sex differences in cardiovascular disease can be explained in part through neuropsychological research data, then a new model of cardiovascular disease is in order. Furthermore, this should lead to a re-examination of any affected research data and conclusions.

Principal functional unit one of cardiovascular regulation seems to have extensive support in literature, whereas, principal functional unit two also has support through a level of implications from previous research (sensory tasks). Another important consideration is the role that emotions play in functional unit two. For, if unhealthy emotional reactions lead to unhealthy patterns of cardiovascular regulation, then basic emotional responding may arise partly from dysfunctional interpretation of sensory information. Unit three has less data of support, but a similar concept was proposed by Tucker, Luu, and Pribram (1995), where they suggested that neural system architectures of the frontal lobe reflect the dual limbic origins of frontal cortex. The two limbic-cortical pathways function differently- with dorsal limbic mechanisms being influenced by "hedonic evaluations, social attachments, and they may initiate a mode of motor control that is holistic and impulsive. In contrast, the ventral limbic pathway from the amygdala to orbital frontal cortex may implement a tight, restricted mode of motor control that reflects adaptive constraints of self-preservation" (Tucker et al., 1995).

Aggression, or hostility as heretofore described, is related to coronary heart disease (CHD) risk through complex associations. Nevertheless, although sex differences have been found in various components of these associations, research on its etiology has focused primarily on men. Thus, many

important theories and models of how aggression leads to CHD have not been sufficiently tested on women (Keltikangas-Järvinen, 1992). Thus, when studying the interplay of principal functional units two and three, this theory could be tested further, and support for this model could be enhanced. However, although principal functional unit three seems to have the least research substantiation so far, it also seems to be the most important unit in regulating complex cardiovascular responses and it seems to be the most important unit when considering the pathogenesis of cardiovascular disease. Thus, the purposes of the following experiments were to first test principal functional unit three, in particular, and then test principal functional unit two, as its activity is thoroughly tied to unit three's activity.

Methodologies used in this experiment have been demonstrated to be sensitive to the effect of neuropsychological sex differences. The proposed model was tested using these established methodologies, through a synthesis of the neuropsychological theories heretofore summarized. The hypotheses and expected outcomes that may lend support to the proposed model are described after the description of the experiment. Several research approaches were used to explore the hypothesized model of the contribution of neuropsychological sex differences on cardiovascular regulation (and ultimately, cardiovascular disease). Each approach targeted the measurement/analysis of neuropsychological functioning in specific brain regions, corresponding to proposed principal functional unit three and principal functional unit two of cardiovascular regulation. Thus, neuropsychological tests were used that target frontal areas (executive tasks, such as fluency), while cardiovascular reactivity was recorded. Furthermore, posterior areas (temporal areas) were targeted also, as hostility was investigated as a mechanism of cardiovascular regulation system dysfunction. Sex differences in the proposed principal functional units were thus examined with group (hostility) effects.

Experiment

Anterior/frontal tasks were designed to test principal functional unit three, while posterior/sensory measures were designed to test the contribution of principal functional unit two.

Variables

Men and women participated in this experiment, as sex differences were the main investigative focus. They were classified as low- mid- or high-hostile, according to a self-report measure, the CMHO. Results from this questionnaire were used to explore subject variables related to cardiovascular reactivity and disease. The dependent variables were the physiological/ cardiovascular measures (SBP, DBP, HR), verbal fluency scores, figural fluency scores, and perseveration scores from these fluency measures. A detailed description and analysis of these factors follows. Thus, results were analyzed by sex, group (hostility level), task (verbal vs. figural), condition (pre- and post-stressor, for cardio. measures), and trial (1-3 for fluency/perseveration measures).

Systolic and Diastolic Blood Pressure Factors

Neuropsychological regulation of cardiovascular dynamics (SBP, DBP, and HR) has been studied extensively, with newer research implicating both the right and the left hemisphere in sympathetic and parasympathetic factors, respectively. In addition, systolic and diastolic pressures are differentially related to heart and peripheral circulatory variables. Systolic blood pressure is influenced by stroke volume and heart rate and it is modulated by arterial distensibility (Steptoe, 1980); it is the measurement of the force of blood passing through the left brachial artery during ventricular contraction (Tortora & Anagnostakos, 1990). However, diastolic blood pressure is related to the rate and time involved for blood to flow out of the arterial tree, as well as peripheral resistance (Steptoe, 1980); it is the measurement of the force of blood passing through the left brachial artery during ventricular relaxation (Tortora & Anagnostakos, 1990). As such, systolic blood pressure provides information about the force of left ventricular contraction, while diastolic blood pressure provides information about the resistance level of the left brachial artery. Diastolic blood pressure can be seen as a measure of physiological resistance (Snyder, Harrison, & Shenal, 1998; Tortora & Anagnostakos, 1990), a function of neural innervation of the arterial walls via the peripheral nervous system. In contrast, systolic blood pressure has been associated with beta adrenergic responsivity of the heart, devoid of neural innervation of this structure. The only innervation of the heart is the vagus nerve with cholinergic, rather than adrenergic properties.

Therefore, systolic blood pressure responsivity may relate more directly to neural humoral systems with frontal lobe interactions with the adrenal gland through the pituitary axis. This latter system is a global response to a stressor which persists indefinitely, whereas heightened specificity of the response through direct neural innervation may be regulating diastolic blood pressure.

Verbal and Figural Fluency Tasks

Investigation of pre-motor cerebral regions allows for a more complete functional analysis of principal functional unit three, especially as a dual task challenge. Fluency is a neuropsychological construct measuring output, or generativity, of verbal or nonverbal material (Strub & Black, 1985; Strub & Black, 1988a; Strub & Black, 1988b). Verbal fluency tests require a listing of words that begin with a pre-specified letter or categorical item whereas nonverbal fluency tests require the generation of unnamable designs or figures (Lezak, 1995). Confrontational fluency tasks, where output must be provided under timed and/or limited search conditions, provide an elegant way to evaluate these anterior cerebral regions functionality and can help uncover their role in cardiovascular regulation, especially their possible role in inhibiting posterior cerebral systems (as cardiovascular indices are impacted by these fluency tasks).

An experimental design using fluency tasks as cerebral system stressors was used by Everhart (1997), when he used fluency as a dual task concurrent with cardiovascular regulation. He found that nonverbal fluency interfered with the sympathetic nervous system activity in anxious-depressed subjects, who showed increases in heart rate and blood pressure. Nevertheless, verbal fluency measures did not have this same effect (Everhart, 1997). In fact, this dual-task approach has been replicated with other populations with good results. Williamson studied high-hostile and low-hostile men and found that high-hostile men evidenced significantly higher systolic blood pressure responses during the nonverbal fluency task in comparison with low hostile men. Further, high-hostile men displayed more perseverative errors in nonverbal fluency than did the low-hostile men (Williamson, 1999).

Thus, these tasks were used as stressors (Everhart, 1997; Williamson, 1999), similar to the way that the cold pressor has been used in the past. As stressors can interfere with normal processing and summon a complex, organized cardiovascular response, the inclusion of a stressor (i.e., confrontational fluency tasks, in this experiment) is essential to cardiovascular research. In fact, laboratory measures looking at reactivity to a stressor have been found to be quite predictive of future cardiovascular problems. One study evaluating the relationship of reactivity to coronary heart disease found that the magnitude of the participants' diastolic blood pressure reactivity to the cold pressor test (a classic laboratory stressor) was associated significantly with development of CHD when a follow-up test was conducted 23 years later. In order to test the proposed model, specific patterns of investigations were undertaken to include stressors with frontal/anterior cerebral activation requirements.

Fluency task performance is dependent on the intentional processes of organization, problem-solving, strategy development and implementation, and the ability to quickly shift to other strategies or "cognitive flexibility." Without these capabilities, fluency output can be impacted. Individuals with frontal lobe damage have been demonstrated to produce reduced output on these tasks (Lezak, 1995; Williamson, 1999). Research has converged from various technologies, such as lesion studies of brain-damaged individuals and functional imaging studies of healthy individuals, to suggest that verbal fluency is associated with left frontal lobe functioning (Benton, 1968; Borokowski, Benton, & Spren, 1981; Butler, Rorsman, Hill, & Tuma, 1993; Demakis & Harrison, 1997; Miceli, Caltagirone, Gainotti, Masullo, & Silveri, 1981; Miller, 1984; Milner, 1964; Pendleton, Heaton, Lehman, & Hulihan, 1982; Perret, 1974; Ramier & Hecaen, 1970; Ruff, Evans, & Marshall, 1986; Schloesser et al., 1998; Tucha, Smely, & Lange, 1999). Likewise, spatial, or figural fluency has been linked to right frontal functioning (Demakis & Harrison, 1997; Jones-Gotman & Milner, 1977; Miceli et al., 1981; Ruff, Allen, Farrow, Niemann, & et al., 1994; Ruff et al., 1986; Ruff, Light, & Evans, 1987). Nevertheless, Tucha (1999) did not find differences in figural fluency between groups (individuals with tumors in the left brain, right brain, and controls). Verbal or figural fluency measures can also aid in the assessment of perseverative errors, which appear as repetitions of words or designs, respectively. Fluency performance in individuals with

frontal damage manifests through low performance scores and is often marked by consistent perseverative errors (Jones-Gotman & Milner, 1977).

Hostility and the CMHO

Typically, reactivity is assessed partially through its association with hostility. For example, Shapiro, Jamner, and Goldstein (1997) found negative moods with anger had high scores on trait hostility and consistently higher levels of blood pressure. The heuristic value of hostility and emotional reactivity can be measured in terms of predictable risks to the individual's health (Contrada, 1994), with evidence for increased morbidity and mortality rates in men and in women (Helmers et al., 1993). The Cook-Medley Hostility Inventory (CMHO; Cook & Medley, 1954) is the most frequently utilized measure of hostility, and it exhibits validity as a predictor of medical, psychological, and interpersonal outcomes (Contrada & Jussim, 1992). Consequently, the CMHO is an increasingly important measure in studies examining health consequences of hostility (Miller, Smith, Turner, Guijarro, & Hallet, 1996; Smith, 1994; Williams & Anderson, 1987). CMHO scores have been shown to be significantly associated with coronary heart disease and hostile (Type A) personality type (Barefoot, Dahlstrom, & Williams, 1983; Miller et al., 1996; Williams, 1984; Williams, 1986; Williams, 1987a; Williams, 1987b; Williams, 1996; Williams et al., 1988; Williams, Barefoot, & Shekelle, 1985; Williams et al., 1980; Williams, Suarez, Kuhn, Zimmerman, & Schanberg, 1991) as well as increased mortality rates (Miller et al., 1996; Shekelle, Gale, Ostfeld, & Paul, 1983; Smith, 1994). Williams, Barefoot, and Shekelle (1985) have contended that high-scoring individuals on the CMHO experience anger more frequently and intensively than other, low-hostile individuals (see also Harrison & Gorelczenko, 1990). The construct validity of the CMHO has been supported by research that found that high CMHO scores were associated with a greater level of aroused anger, suspicious thoughts, and the tendency to attribute hostile intent to others (Pope, Smith, & Rhodewalt, 1990). Further, individuals scoring high on the CMHO have shown significantly elevated cardiovascular responses when harassed, compared to low-hostiles who were harassed (Suarez & Williams, 1989). The discoveries from these and other investigations have been consonant with previous conceptual descriptions of cognitive, behavioral, and affective correlates of hostility (Pope, Smith & Rhodewalt, 1990), supporting the idea that the CMHO scale can be interpreted as reflecting future, potential health consequences of hostility.

Neuropsychological frontal/executive tasks for this experiment included a verbal fluency task and a figural fluency task to test the integrity of principal functional unit three: the executive organization of complex cardiovascular responses, mediated by neuropsychological processes. By including neuropsychological tasks that targeted these frontal areas, inhibition effects on posterior sensory areas (functional unit two) and corresponding changes in cardiovascular factors were investigated. In addition, hostility was added as a factor intended to test the dynamic functioning of principal functional units two and three, through assessing the interaction of the stressor with hostility, a possible mediating factor in these systems. Thus, the following hypotheses were predicted. These can be categorized under predicted sex differences and predicted group differences (high, middle, and low-hostile).

Hypotheses

1. A sex by condition interaction was predicted for cardiovascular reactivity to figural fluency tasks, as relative cardiovascular stability was predicted for women as a function of figural fluency tasks, while greater sympathetic cardiovascular changes (increases in SBP and HR) were predicted to figural fluency tasks in men.
2. Another sex by condition interaction was predicted for cardiovascular reactivity to verbal fluency tasks, as relative cardiovascular stability was predicted for women as a function of verbal fluency tasks, while greater parasympathetic cardiovascular effects/changes (relative stability or decreases in SBP and HR) were predicted to verbal fluency tasks in men.
3. Sex differences were predicted in verbal fluency and figural fluency performance. Women were expected to outperform men on the verbal fluency task, while men were expected to outperform women on the figural fluency task.

4. Men were predicted to perform better on figural compared to verbal fluency tasks; while women were expected to perform better on verbal compared to figural fluency tasks.
5. Prior to the stressors (fluency tasks), men were predicted to evidence higher levels of systolic blood pressure than women.
6. Men were predicted to evidence significantly more perseverative errors on figural fluency than women.
7. A sex by group by condition interaction was predicted for cardiovascular reactivity to figural fluency tasks, as greater sympathetic changes (increases in SBP and HR) were predicted for high-hostile men than for high-hostile women as a function of figural fluency tasks.
8. A sex by group by condition interaction was predicted for cardiovascular reactivity to verbal fluency tasks, as greater parasympathetic changes (relative stability or decreases in SBP and HR) were predicted for low-hostile men than for low-hostile women as a function of verbal fluency tasks.
9. Sex differences in overall cardiovascular stability were predicted for low-hostile women more than low-hostile men.
10. High-hostile men will evidence more perseverative errors on figural fluency tasks than low-hostile men.

Method

Participant Selection

One hundred ninety-eight participants participated in group-screenings, recruited from the Introductory Psychology course and other psychology courses at Virginia Polytechnic Institute and State University through the offering of extra credit. Of these participants, one hundred twenty-six (63.64% of those screened) right-handed undergraduate men (N = 63) and women (N = 63) underwent physiological measurements of systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) before and after verbal and figural fluency tasks, used as stressors (verbal: left frontal, and figural: right frontal). Only right-handed participants were used in the experiment, for enhanced data purity (Kertesz, Polk, Black, & Howell, 1992) and to ensure maximum homogeneity within the experiment (so that observed differences are more likely due to sex differences than to handedness). These participants were assigned to one of six groups: low-hostile men, mid-hostile men, high-hostile men, low-hostile women, mid-hostile women, or high-hostile women (each group included 21 participants), according to participant responses on a self-report measure of hostility, the Cook-Medley Hostility Inventory (Cook & Medley, 1954). Functional cerebral regulation of cardiovascular reactivity was assessed, specifically, the role that the frontal lobes have in regulating SBP, DBP, and HR in men and women, and the interaction of hostility in this model.

Classification

All participants were required to sign a consent form prior to participation in Phase One of the experiment (Appendix A). All participants received a neurological screening questionnaire (Head Injury/Medical History Inventory, or HIMHI; Appendix B). This questionnaire is a general survey of present/past neurologically-related problems and other medical conditions which may interfere with neuropsychological test performance. Participants with a history of head injury, learning disabilities, any significant neurological history (stroke, seizure, paralysis, pain disorder, etc.), current (illicit) drug use, or any serious psychological or physical illness were excluded from Phase Two of the experiment. Twenty-three participants (11.62% of those screened) were disqualified from continuing to Phase Two for one or more of these reasons.

Participants were then given the Coren, Porac, and Duncan Laterality Questionnaire (1979; Appendix C) to determine lateral hemibody preference. This self-report assesses right (+1) and left (-1) hemibody preference based on reported preferred use of four types of lateral preference: eye, ear, arm, and leg (Coren, Porac, & Duncan, 1979). Scores range from (-13) to (+13), indicating extreme left and right hemibody preference, respectively. Average concordance between the self-report and the behavioral measures for this questionnaire is stated to be .90. The criterion for right-hemibody preference and

inclusion for continued participation in the experiment (Phase Two) was a score of +6 or more. To simplify matters, right-hemibody preference will be referred to as "right-handedness" elsewhere in this document. Twenty-one participants (10.60% of those screened) were disqualified from continuing to Phase Two due to a low score on this measure.

In addition, 12 participants (6.06% of those screened) who qualified for participation in Phase Two did not wish to continue in the experiment (for various reasons). 16 other participants (8.08% of those screened) who qualified for continued participation in Phase Two were not asked to continue, to keep cell sizes equal (they were categorized as mid-hostile).

For the purposes of replication of previous research findings and for collection of pilot data for future research projects, all participants were also asked to complete the Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1960; Appendix D). This scale consists of 33 items and is purported to identify individuals who depict themselves in a very favorable way (Crowne & Marlowe, 1960; Crowne & Marlowe, 1964). They can be understood as wanting to be seen in socially-desirable terms, and can be conceptualized as an index of "denial." Using the Kuder-Richardson formula 20 for this scale, the internal consistency coefficient is .88. Further, a test-retest correlation of .88 was obtained, indicating satisfactory reliability (Crowne & Marlowe, 1964). The normative mean score for men (published in 1964) is 15.06 with a standard deviation of 5.58. More recently, however (Emerson & Harrison, 1990), the mean score for men attending college was reported to be 18.82 with a standard deviation of 2.74, where individuals with a high denial profile actually demonstrated increased physiological reactivity to stress (Emerson & Harrison, 1990). Therefore, the cutoff scores for future planned comparisons were established at the raw scores of >22 (high-deniers) and <16 (low-deniers).

Furthermore, for the purposes of replication of previous research findings and to collect pilot data for future possible research projects, all participants were also asked to complete State-Trait Anxiety Inventory (Speilberger, 1968; Appendix E). This is a 40 item self-report questionnaire which assesses state and trait anxiety. Twenty questions are provided to evaluate the way a person feels "right now" (state anxiety), and 20 questions are provided to assess the way a person feels "in general" (trait anxiety). Scores on the STAI may range between 20 and 80 for the State Inventory and between 20 and 80 for the Trait Inventory. Test-retest reliability for the trait portion of the STAI ranges from .73 to .86, while test-retest reliability of the state portion ranges from .16 to .54. Participants with scores of 41 or higher were classified as having elevated anxiety (HI) levels, for future planned comparisons. This is approximately one-half standard deviation above the normed mean of 38 for male college students. Participants with scores of 33 or lower were classified as having low levels of anxiety (LO), for future planned comparisons. This score is approximately one-half standard deviation below the normed mean for male college students.

Hostility was assessed in these participants, using the Cook-Medley Hostility Inventory (CMHO; Cook & Medley, 1954; Appendix F). Men who scored 21 or below on the hostility scale and who met the above criteria were asked to continue with Phase Two of the experiment (testing of cardiovascular reactivity to verbal and figural fluency) as the low-hostile group; while men who scored in the range of 22 to 26 points were asked to continue as the mid-hostile group; and men who scored at 27 and above on this measure were asked to continue as the high-hostile group. However, women who scored 17 or below on the hostility scale and who met the above criteria were asked to continue with Phase Two of the experiment (testing of cardiovascular reactivity to verbal and figural fluency) as the low-hostile group; while women who scored in the range of 18 to 24 points were asked to continue as the mid-hostile group; and women who scored at 25 and above on this measure were asked to continue as the high-hostile group. Norms indicating reliable sex differences on this measure dictated that to classify a man and a woman at the same score as either low- mid- or high-hostile was erroneous. Thus, cut-offs were considered in light of other previous studies at Virginia Tech, where high- and low-hostile men and women were identified in the top and bottom one-third of samples. Other studies, testing hundreds of students also suggested setting specific values for men and women on this measure (Smith & Frohm, 1985; Smith, Cranford, & Mann, 2000).

Phase 2. All participants were required to sign a consent form prior to participation in phase two of the experiment (Appendix G). Participants were again administered the Cook-Medley Hostility Scale (CMHO) to ensure classification stability (i.e., that they were still classifiable as high- mid- or low-hostile). Any variation greater than one point disqualified them from participation in Phase Two.

Materials and Apparatus

Physiological Assessment Apparatus: A Korotkoff automated digital blood pressure unit (Model SD-700A, IBS Corporation) was used to measure systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR). A preset inflation pressure setting of 180 mm Hg was used for all subjects. The exhaust rate was 3 mm Hg/second, performed automatically following each reading. The accuracy of blood pressure measurement is reported to be ± 3 mm Hg of those auscultated, while the accuracy of heart rate readings is reported to be within 2% of those gauged (approx. 1 beat / min.). This procedure thus adhered to the basic requirements of the American National Standard Institute (ANSI), the Association for the Advancement of Medical Instrumentation (AAMI) standards, and the American Heart Association recommendations (Harrison & Edwards, 1988; Harrison, Gorelczenko, & Kelly, 1988). The left arm of the participants was partially extended, supported, and positioned at about the fourth intercostal space with the palmar surface facing upward. The cuff was be positioned on the left upper arm with the cuff connector located over the brachial artery about 2.5 cm above the antecubital space. Arterial location was determined by palpation. In the event of an error reading, the cuff was removed and replaced to assure correct placement over the brachial artery. Each measurement of cardiovascular factors (SBP, DBP, and HR) taken was immediately repeated in order to control for stability. A third reading was taken if the first two readings differed by 8 beats per minute (HR) or 20 mm Hg for either SBP or DBP. Therefore, physiological data for any given trial was the average of two separate measurements.

Testing Room

The screening room for Phase One was a standard classroom at a large university. The participant filled out the questionnaires, then was scheduled to participate in Phase Two, if he or she qualified for inclusion. The testing chamber for Phase Two was composed of a desk chair in a sound-attenuated testing room. The participant faced a flat white wall and sat in a chair surrounded by a flat white vinyl curtain. Physiological assessment equipment was located outside the testing chamber in an adjacent laboratory equipment room, connected by cables and tubes to the corresponding physiological sensors. Participants were monitored through an observation window.

Fluency Materials

Verbal Fluency. The Controlled Oral Word Association Test (COWAT) was used to assess verbal fluency, the production of oral or written words beginning with a designated letter (Benton & Hamsher, 1976). It consisted of three trials in which participants were instructed to write as many words as possible in one minute beginning with a specified letter. Proper names, numbers, and the same word with different endings were not permitted (Appendix H). The final score is the sum of all acceptable words produced across trials. Test-retest reliability (after one-year) for this measure is approximately .71. This is the most commonly used test of verbal fluency; nevertheless, similar findings (i.e., depressed verbal fluency with left anterior damage) have been obtained with protocol variants using either different letters or time limits (Lezak, 1995). The letters F, A, and S are the most frequently utilized; however, the present experiment used a variation of the protocol, specifically, using the letters F, S, and T. These letters were chosen based on the tendency of normals to produce an equal number of responses for each letter, approximately 11-12 words per minute (Everhart, 1997; Lezak, 1995). Furthermore, this was done to more accurately replicate previous research on fluency and cardiovascular measures using high- and low-hostile men (Williamson, 1999) and high- and low-anxious men (Everhart, 1997). For this experiment, each trial was analyzed separately in order to assess performance across time.

Figural Fluency. The Ruff Figural Fluency Test (RFFT) was used to assess spatial fluency (Ruff et al., 1987). It is a valid measure, and test-retest reliability is approximately .76. It consists of five parts, each containing different stimulus presentations (i.e., dot matrices). Each part has 35 identical dot matrices arranged in a 5 X 7 array (Figure 8). Also, three samples of the stimuli for each part are displayed for practice. Participants were instructed to connect the dots in as many unique ways that they can conceive

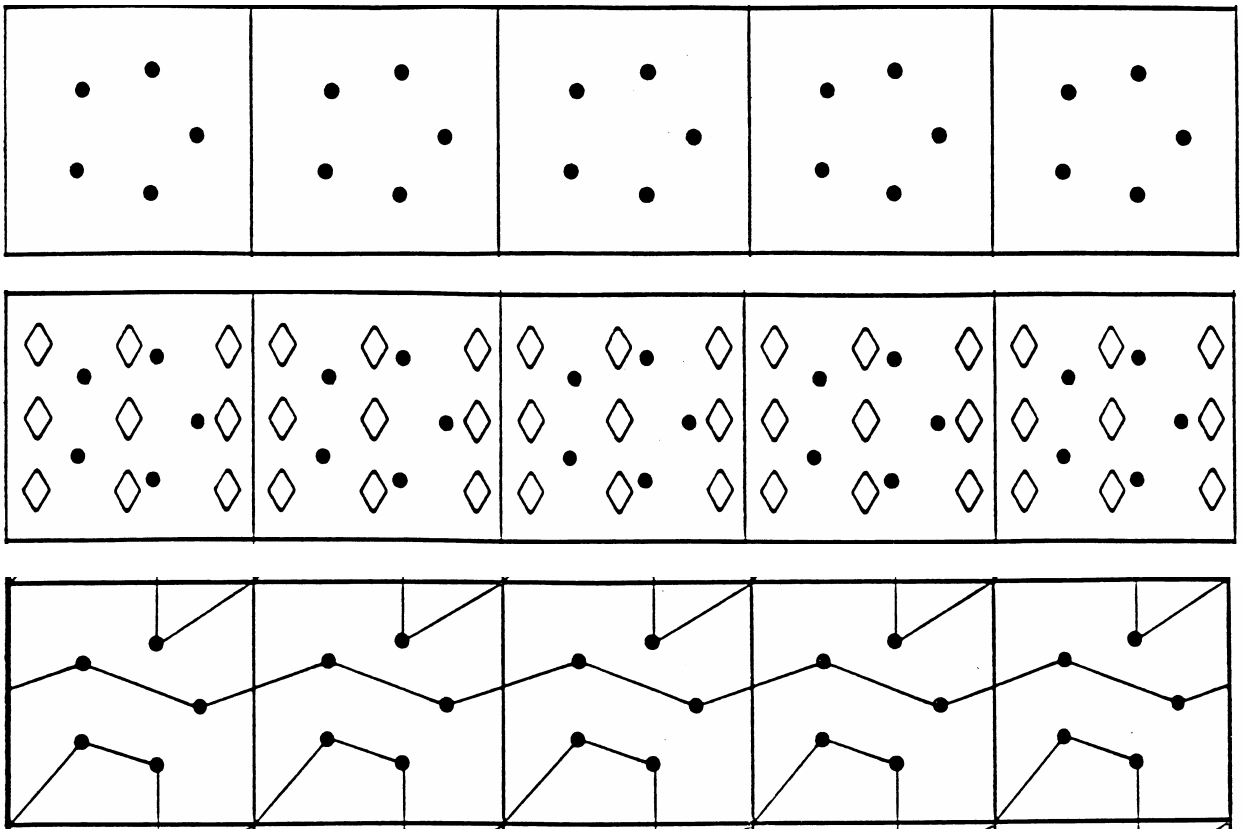


Figure 8. Sample items from the Ruff Figural Fluency Test (RFFT), parts one through three.

within a one minute period (Appendix I). It is scored by counting the number of unique designs and number of perseverations for each trial. A perseveration is any repetition of a previous design within the participant's responses on a single test sheet. Accuracy of scoring is ensured by checking each design against the remaining productions. Figural fluency is then considered to be the total number of unique designs generated across each test sheet minus the number of perseverations within each part. Three sheets, instead of the standard five, were used in this experiment in order to maintain consistency with the COWAT and with previous studies of fluency and cardiovascular reactivity (Everhart, 1997; Williamson, 1999). Scores on the RFFT were analyzed similar to the scores on the COWAT, with each trial analyzed separately to assess performance across time.

Experiment

Procedure: Phase One

Group Screenings. Participants in phase one were asked to sign a consent form and were asked to complete inventories assessing medical history (HIMHI), hemibody preference (CPD), hostility (CMHO), and other variables for future planned comparisons (STAI, MCSDS). Those who qualified according to criteria previously detailed were scheduled to return for further testing, in Phase Two.

Procedure: Phase Two

The experiment consisted of three "stressor" trials contained in two fluency tasks (verbal or figural, whichever was first; counterbalanced), while measurements of cardiovascular functioning were taken at pre-stressor and post-stressor points of measurement (condition: pre- or post-). Thus, cardiovascular indices were measured at baseline (after the participant was able to relax for 3 minutes) and then after three stressor trials (T1, T2, and T3). This experimental protocol (including a relaxation time and a 2nd baseline measurement) was then repeated for the other fluency type (see Figure 9). Following this testing, a time estimation task was conducted where the participant was asked to estimate the passage of 30 seconds and then 3 minutes of time.

Testing was conducted with one participant at a time. As each participant began the second phase of the experiment, he/she was assigned to one of two different groups to start: the verbal fluency first group or the figural fluency first group. Participants were assigned to these groups in a rotational fashion (verbal, figural, verbal, figural…) within each sex. This will be done until a total of 63 men and 63 women were tested.

Each participant had approximately 10 minutes after arriving to adjust to the testing room, as he/she filled out the consent form and the CMHO. The participant was then fitted with the cardiovascular measurement apparatus. The blood pressure monitor was attached to each participant's left upper-arm. The researcher then said the following, "Please make yourself comfortable, try to relax, and sit still in the chair." SBP, DBP, and HR data was collected after a 180 second baseline period. The researcher then read the instructions for the appropriate fluency task (see Appendices G and H), and then administered the practice trial of the verbal/figural fluency task. Three fluency trials (all verbal or figural) were then administered. After finishing these fluency trials, SBP, DBP, and HR data were again collected. This protocol was carefully repeated for the other fluency measure.

A simple time estimation task was also performed at the end of the experiment. Each participant was asked to estimate when 30 seconds had passed and when 3 minutes had passed. Responses were recorded on a Recording Form (Appendix J), which also contained all of the cardiovascular readings. The purpose of this task was to examine whether men, compared to women, have difficulty estimating time (e.g., they estimate that 3 minutes have passed when, in reality, only 2 ½ minutes have passed) and if hostility level had an effect on the perception of "time urgency" or "impatience," two constructs possibly associated with this task's data.

Using a 9-point Likert Scale, each participant will also be asked to rate the level of stress that they experienced during the experiment, how stressful the verbal and the figural fluency tasks were, and how anxious they felt during the experiment (Appendix K). Following the experiment, participants will be thoroughly debriefed and asked about their feelings of the experiment. If the participants exhibit negative verbal or nonverbal behavior then extra time will be spent to help them resolve these feelings.

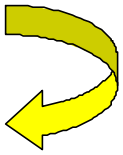
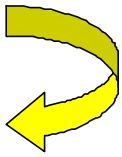
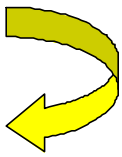
Groups	Rest Period	Cardio. Baseline	Fluency Trial 1	Fluency Trial 2	Fluency Trial 3	Cardo. Post	Repeat for Other Fluency Measure	time estima-tion task
High-Hostile Men	180 Seconds	SBP, DBP, HR	Verbal	Verbal	Verbal	SBP, DBP, HR		**30 sec. & 3 min.**
	180 Seconds	SBP, DBP, HR	Figural	Figural	Figural	SBP, DBP, HR		
Mid-Hostile Men	180 Seconds	SBP, DBP, HR	Verbal	Verbal	Verbal	SBP, DBP, HR		**30 sec. & 3 min.**
	180 Seconds	SBP, DBP, HR	Figural	Figural	Figural	SBP, DBP, HR		
Low-Hostile Men	180 Seconds	SBP, DBP, HR	Verbal	Verbal	Verbal	SBP, DBP, HR		**30 sec. & 3 min.**
	180 Seconds	SBP, DBP, HR	Figural	Figural	Figural	SBP, DBP, HR		
High-Hostile Women	180 Seconds	SBP, DBP, HR	Verbal	Verbal	Verbal	SBP, DBP, HR		**30 sec. & 3 min.**
	180 Seconds	SBP, DBP, HR	Figural	Figural	Figural	SBP, DBP, HR		
Mid-Hostile Women	180 Seconds	SBP, DBP, HR	Verbal	Verbal	Verbal	SBP, DBP, HR		**30 sec. & 3 min.**
	180 Seconds	SBP, DBP, HR	Figural	Figural	Figural	SBP, DBP, HR		
Low-Hostile Women	180 Seconds	SBP, DBP, HR	Verbal	Verbal	Verbal	SBP, DBP, HR		**30 sec. & 3 min.**
	180 Seconds	SBP, DBP, HR	Figural	Figural	Figural	SBP, DBP, HR		

Figure 9. Experimental Design.

Data Analyses

Self Report Scales/Descriptive Measures

Participants with a history of a past traumatic head injury, neurological damage/insult, or serious physical or psychiatric illness (according to the HIMHI) were excluded from the experiment, as well as any participants with a score of less than 6 on the Coren, Porac, and Duncan Laterality Questionnaire (CPD). T-tests were conducted to assess age, CPD, and/or CMHO differences between men and women.

Cardiovascular Measures

An analysis of variance (ANOVA) was performed on the physiological variables (SBP, DBP, MAP, and HR) to measure cardiovascular reactivity to the fluency stressors. Mean arterial pressure was computed using the standard formula: MAP = (1/3)(SBP-DBP) + DBP. The design was: Sex (2 levels) x Group (3 levels) x (Task [2 levels] x Condition [2 levels] x Participant). Thus, data was analyzed with a four-factor mixed design analysis of variance (ANOVA), with fixed factors of Sex (men and women) and Group (high-hostile, mid-hostile, and low-hostile), and repeated measures of Task (verbal vs. figural fluency) and Condition (baseline and post-stressor).

In addition, cardiovascular indices were analyzed within each task. To accomplish this, a design of Sex (2 levels) x Group (3 levels) x (Condition [2 levels] x Participant) was utilized. Thus, data was analyzed with a three-factor mixed design analysis of variance (ANOVA), with fixed factors of Sex (men and women) and Group (high-hostile, mid-hostile, and low-hostile), and repeated measures of Condition (baseline and post-stressor). Furthermore, refined analyses within each sex, group, and condition were performed.

Verbal Fluency and Perseveration

An analysis of variance (ANOVA) was performed on verbal fluency scores to analyze the number of unique words for each task, or fluency, and the number of perseverations. The design was: Sex (2 levels) x Group (3 levels) x (Trial [3 levels] x Participant). Thus, data for verbal fluency was analyzed with a three-factor mixed design analysis of variance (ANOVA), with fixed factors of Sex (men and women) and Group (high-hostile, mid-hostile, and low-hostile), and repeated measures of Trial (fluency score 1, 2, and 3). This statistical analysis was then repeated using verbal perseveration scores (with repeated measures of Trial as perseveration percentage 1, 2, and 3). In addition, refined analyses within each sex, group, and trial were performed.

Figural Fluency and Perseveration

An analysis of variance (ANOVA) was then performed on figural fluency scores to analyze the number of unique designs for each task, or fluency, and the number of perseverations. The design was: Sex (2 levels) x Group (3 levels) x (Trial [3 levels] x Participant). Thus, data for verbal fluency was analyzed with a three-factor mixed design analysis of variance (ANOVA), with fixed factors of Sex (men and women) and Group (high-hostile, mid-hostile, and low-hostile), and repeated measures of Trial (fluency score 1, 2, and 3). This statistical analysis was then repeated using figural perseveration scores (with the repeated measures of Trial as perseveration percentage 1, 2, and 3). In addition, refined analyses within each sex, group, and trial were performed.

Time Estimation Task

An analysis of variance (ANOVA) was performed on the Time Estimation Scores for 30 and 180 second time estimation values, to analyze this tasks relationship with each sex and group. The design was: Sex (2 levels) x Group (3 levels) x (Trial [2 levels] x Participant). Thus, data was analyzed with a three-factor mixed design analysis of variance (ANOVA), with fixed factors of Sex (men and women) and Group (high-hostile, mid-hostile, and low-hostile), and repeated measures of Trial (time_30 and time_180 score). In addition, refined analyses within each sex, group, and trial were performed.

Stress Scores

An analysis of variance (ANOVA) was performed on the Stress and Anxiety Scores, to analyze this tasks relationship with each sex and group on this self-reported level of distress descriptive measure. The design was: Sex (2 levels) x Group (3 levels) x (Trial [4 levels] x Participant). Thus, data was

analyzed with a three-factor mixed design analysis of variance (ANOVA), with fixed factors of Sex (men and women) and Group (high-hostile, mid-hostile, and low-hostile), and repeated measures of Trial (various stress/anxiety scores). In addition, refined analyses within each sex, group, and trial were performed.

Menstrual Phase

An analysis of variance (ANOVA) was also performed using the reported phase of menstruation (follicular versus luteal) in women as a factor, to analyze its effect on verbal and figural fluency performance, as well as verbal and figural perseveration. Also, its interaction with hostility was assessed. The design was: Phase (2 levels) x Group (3 levels) x (Trial [3 levels] x Participant). Thus, data for verbal fluency was analyzed with a three-factor mixed design analysis of variance (ANOVA), with fixed factors of Phase (follicular and luteal) and Group (high-hostile, mid-hostile, and low-hostile), and repeated measures of Trial (fluency score 1, 2, and 3; or perseveration percentage 1, 2, and 3). This statistical analysis was then repeated using figural fluency scores and then figural perseveration scores. In addition, refined analyses within each group and trial were performed.

Results

Descriptive Measures

In order to assess possible sex differences on the descriptive measures, t-tests were performed. There was no significant difference between the mean age of men (M = 20.28 years, SD = 1.61) and women (M = 19.93 years, SD = 1.06), $T(124) = 1.46$, $p < .15$. However, there was a significant difference between men's and women's mean score on the Coren, Porac, and Duncan Laterality Questionnaire, as women were found to have higher mean scores on this measure, compared to men, (men: M = 9.73, SD = 2.54; women: M = 10.73, SD = 2.41), $T(124) = -2.27$, $p < .03$. Nevertheless, men were found to have significantly higher scores on the Cook-Medley Hostility Inventory (CMHO) than women, (men: M = 23.38, SD = 7.74; women: M = 20.48, SD = 7.89), $T(124) = 2.09$, $p < .04$.

Cardiovascular Reactivity to Task

In order to assess cardiovascular reactivity to the cognitive tasks (verbal and figural fluency), independent analyses of variance (ANOVA) were performed on the following dependent variables of cardiovascular reactivity: systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR). Data were analyzed for overall sex, group, and (fluency) task effects/interactions across conditions first, and then refined ANOVAs were performed to test group, task, and condition effects within each sex, and task and condition effects within each group. Group means and standard deviations of SBP, DBP, MAP, and HR measures are presented in Tables 1, 2, 3, and 4 respectively. Independent ANOVA results of cardiovascular measures SBP and DBP are presented in Table 5, while ANOVA results of cardiovascular measures MAP and HR are presented in Table 6.

A main effect of sex was found for systolic blood pressure and diastolic blood pressure, as well as mean arterial pressure (derived from these two readings). Overall, men evidenced significantly higher SBP than women (M = 126.23 vs. 116.96, $F(1,120) = 33.04$, $p < 0.0001$; Figure 10). However, women evidenced significantly higher DBP than men (M = 84.03 vs. 75.46, $F(1,120) = 33.69$, $p < 0.0001$; Figure 11). In addition, women evidenced significantly higher MAP than men (M = 95.01 vs. 92.38, $F(1,120) = 4.41$, $p < 0.0378$; Figure 12). A main effect of sex was not found for HR.

A main effect of group was found only for SBP ($F(2,120) = 3.15$, $p < 0.0463$; Figure 13), as mid-hostile men and women had the highest SBP (M = 123.21), followed by low-hostile men and women (M = 122.84) and then high-hostile men and women (M = 118.74). However, despite the finding of a main effect, these mean scores were not significantly different from each other.

A main effect of condition was found for SBP ($F(1,120) = 7.75$, $p < 0.0062$), DBP ($F(1,120) = 14.66$, $p < 0.0002$), and MAP ($F(1,120) = 19.88$, $p < 0.0001$), with significant *increases* occurring between conditions 1 and 2 (pre-stressor and post-stressor).

A group by task interaction effect was evidenced for SBP ($F(2,120) = 3.63$, $p < 0.0294$), wherein low-hostile men and women experienced different cardiovascular effects (higher SBP across verbal

fluency tasks versus lower SBP across figural fluency tasks), compared to mid-hostile and high-hostile men and women, who did not evidence such group differences (Figure 14). A group by task interaction effect was also evidenced for MAP ($F(2,120) = 3.74$, $p < 0.0266$), wherein low-hostile men and women experienced different cardiovascular effects across verbal fluency tasks (higher SBP), compared to figural fluency tasks (lower SBP), and mid-hostile men and women experienced the opposite effects across verbal fluency tasks (lower SBP), compared to figural fluency tasks (higher SBP). Nevertheless, mid-hostile and high-hostile men and women did not evidence such differences across these tasks (Figure 15).

A task by condition interaction effect on SBP was found, as blood pressure increased significantly across groups after the verbal fluency task, but not the figural fluency task ($F(1,120) = 5.42$, $p < 0.0216$; Figure 16).

A sex by group by task interaction was evidenced for all cardiovascular measures. Thus, these factors were found to be significant in the proposed model. Systolic blood pressure was relatively stable in women across groups and tasks, while low-hostile men reacted differently (SBP) to the tasks, compared to mid-hostile and high-hostile men, who evidenced relatively stable response patterns (similar to women; $F(2,120) = 3.20$, $p < 0.0442$; Figure 17). Low-hostile men were found to have higher levels of systolic blood pressure across verbal fluency tasks and lower levels of systolic blood pressure across figural fluency tasks. Diastolic blood pressure differences were also found in both sexes, by group and task ($F(2,120) = 3.08$, $p < 0.0497$; Figure 18). Increased diastolic blood pressure levels were found across verbal fluency tasks (compared to figural fluency tasks) in low-hostile men, low-hostile women, and high-hostile women. Nevertheless the opposite pattern was found in mid-hostile men, high-hostile men, and mid-hostile women (higher diastolic blood pressure across figural fluency tasks compared to verbal fluency tasks). Mean arterial pressure differences were also found according to sex, group, and task ($F(2,120) = 3.99$, $p < 0.0211$; Figure 19). Higher mean arterial pressure was found across verbal fluency tasks in low-hostile men, and high-hostile women, relative to mean arterial pressure across figural fluency tasks. However, higher mean arterial pressure was found across figural fluency tasks in mid-hostile men, high-hostile men, and mid-hostile women, relative to map across verbal fluency tasks. Low-hostile women did not evidence mean arterial pressure differences across tasks. Heart rate was relatively stable in mid-hostile men across groups and tasks, but all other groups were found to respond differently to each task ($F(2,120) = 3.46$, $p < 0.0346$; Figure 20). Higher heart rate s were found across verbal fluency tasks in low-hostile and high-hostile women, compared to heart rates across figural fluency tasks. Nevertheless, all other groups (low-hostile men, high-hostile men, and mid-hostile women) were found to have higher heart rates across figural fluency tasks, relative to heart rates across verbal fluency tasks.

Table 1

Means and Standard Deviations of SBP by Sex, Group, and Task (verbal vs. figural fluency)

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Task 1 (verbal fluency)	131.1	10.0	117.3	9.5
Task 2 (figural fluency)	125.9	8.8	117.1	9.2
Mid-Hostile Group				
Task 1 (verbal fluency)	129.2	11.7	117.0	8.5
Task 2 (figural fluency)	129.2	11.3	117.5	9.4
High-Hostile Group				
Task 1 (verbal fluency)	121.0	9.0	116.6	9.1
Task 2 (figural fluency)	121.1	10.8	116.2	10.6

Table 2

Means and Standard Deviations of DBP by Sex, Group, and Task (verbal vs. figural fluency)

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Task 1 (verbal fluency)	75.6	10.4	84.2	8.5
Task 2 (figural fluency)	74.6	8.8	83.8	9.3
Mid-Hostile Group				
Task 1 (verbal fluency)	76.7	8.6	83.5	9.2
Task 2 (figural fluency)	78.2	10.1	84.9	9.0
High-Hostile Group				
Task 1 (verbal fluency)	72.6	9.6	84.8	7.4
Task 2 (figural fluency)	75.1	9.8	83.1	7.2

Table 3

Means and Standard Deviations of MAP by Sex, Group, and Task (verbal vs. figural fluency)

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Task 1 (verbal fluency)	94.1	8.3	95.2	7.5
Task 2 (figural fluency)	91.7	6.6	94.9	8.1
Mid-Hostile Group				
Task 1 (verbal fluency)	94.2	8.5	94.6	7.7
Task 2 (figural fluency)	95.2	9.6	95.8	7.5
High-Hostile Group				
Task 1 (verbal fluency)	88.7	6.2	95.4	6.8
Task 2 (figural fluency)	90.5	6.7	94.1	7.2

Table 4

Means and Standard Deviations of HR by Sex, Group, and Task (verbal vs. figural fluency)

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Task 1 (verbal fluency)	74.6	11.9	79.1	11.7
Task 2 (figural fluency)	75.6	10.9	78.2	12.4
Mid-Hostile Group				
Task 1 (verbal fluency)	76.0	13.1	76.4	13.7
Task 2 (figural fluency)	75.9	12.4	78.4	13.8
High-Hostile Group				
Task 1 (verbal fluency)	74.2	10.1	76.4	10.4
Task 2 (figural fluency)	74.8	10.4	74.1	11.0

Table 5

ANOVA Results for SBP and DBP Changes by Sex, Group, Task, and Condition

Source	DF	SS	MS	F Value	p
Systolic BP					
Sex	(1,120)	10827.17	10827.17	33.04	<0.0001**
Group	(2,120)	2066.97	1033.49	3.15	<0.0463*
Sex*Group	(2, 120)	1368.70	684.35	2.09	<0.1284
Task	(1,120)	89.17	89.17	3.02	<0.0846
Sex*Task	(1,120)	86.67	86.67	2.94	<0.0891
Group*Task	(2,120)	220.57	110.29	3.74	<0.0266*
Sex*Group*Task	(2,120)	188.87	94.44	3.20	<0.0442*
Condition	(1,120)	151.14	151.14	7.75	<0.0062**
Sex*Condition	(1,120)	32.00	32.00	1.64	<0.2026
Group*Condition	(2,120)	15.50	7.75	0.40	<0.6728
Sex*Group*Condition	(2,120)	2.84	1.42	0.07	<0.9298
Task*Condition	(1,120)	105.88	105.88	5.42	<0.0216*
Sex*Task*Condition	(1,120)	0.51	0.51	0.03	<0.8722
Group*Task*Condition	(2,120)	9.12	4.56	0.23	<0.7924
Sex*Group*Task*Cond.	(2,120)	69.38	34.69	1.77	<0.1739

Source	DF	SS	MS	F Value	p
Diastolic BP					
Sex	(1,120)	9252.86	9252.86	33.69	<0.0001**
Group	(2,120)	308.60	154.30	0.56	<0.5717
Sex*Group	(2, 120)	234.71	117.35	0.43	<0.6533
Task	(1,120)	18.10	18.10	0.77	<0.3823
Sex*Task	(1,120)	46.14	46.14	1.96	<0.1640
Group*Task	(2,120)	98.05	49.02	2.08	<0.1290
Sex*Group*Task	(2,120)	144.89	72.45	3.08	<0.0497*
Condition	(1,120)	245.14	245.14	14.66	<0.0002**
Sex*Condition	(1,120)	1.97	1.97	0.12	<0.7321
Group*Condition	(2,120)	12.78	6.39	0.38	<0.6832
Sex*Group*Condition	(2,120)	19.11	9.56	0.57	<0.5663
Task*Condition	(1,120)	1.73	1.73	0.10	<0.7511
Sex*Task*Condition	(1,120)	8.51	8.51	0.50	<0.4817
Group*Task*Condition	(2,120)	56.30	28.15	1.65	<0.1969
Sex*Group*Task*Cond.	(2,120)	48.74	24.37	1.43	<0.2443

* $p < .05$, ** $p < .01$

Table 6

ANOVA Results for MAP and HR Changes by Sex, Group, Task, and Condition

Source	DF	SS	MS	F Value	p
Mean Arterial Pressure					
Sex	(1,120)	866.91	866.91	4.41	<0.0378*
Group	(2,120)	654.40	327.20	1.66	<0.1937
Sex*Group	(2, 120)	463.53	231.76	1.18	<0.3112
Task	(1,120)	0.10	0.10	0.01	<0.9410
Sex*Task	(1,120)	2.03	2.03	0.12	<0.7350
Group*Task	(2,120)	128.27	64.13	3.63	<0.0294*
Sex*Group*Task	(2,120)	140.71	70.36	3.99	<0.0211*
Condition	(1,120)	211.30	211.30	19.88	<0.0001**
Sex*Condition	(1,120)	0.90	0.90	0.08	<0.7712
Group*Condition	(2,120)	13.64	6.82	0.64	<0.5283
Sex*Group*Condition	(2,120)	10.97	5.49	0.52	<0.5981
Task*Condition	(1,120)	6.52	6.52	0.62	<0.4343
Sex*Task*Condition	(1,120)	4.76	4.76	0.45	<0.5039
Group*Task*Condition	(2,120)	25.02	12.51	1.18	<0.3107
Sex*Group*Task*Cond.	(2,120)	3.58	1.79	0.17	<0.8449

Source	DF	SS	MS	F Value	p
Heart Rate					
Sex	(1,120)	462.88	462.88	0.89	<0.3481
Group	(2,120)	398.04	199.02	0.38	<0.6836
Sex*Group	(2, 120)	171.22	85.61	0.16	<0.8488
Task	(1,120)	0.29	0.29	0.01	<0.9067
Sex*Task	(1,120)	26.70	26.70	1.29	<0.2584
Group*Task	(2,120)	67.47	33.73	1.63	<0.2004
Sex*Group*Task	(2,120)	143.29	71.65	3.46	<0.0346*
Condition	(1,120)	38.89	38.89	2.06	<0.1538
Sex*Condition	(1,120)	5.79	5.79	0.31	<0.5809
Group*Condition	(2,120)	52.48	26.24	1.39	<0.2531
Sex*Group*Condition	(2,120)	32.77	16.39	0.87	<0.4224
Task*Condition	(1,120)	5.57	5.57	0.34	<0.5616
Sex*Task*Condition	(1,120)	13.02	13.02	0.79	<0.3754
Group*Task*Condition	(2,120)	44.59	22.30	1.36	<0.2617
Sex*Group*Task*Cond.	(2,120)	27.11	13.56	0.82	<0.4410

* $p < .05$, ** $p < .01$

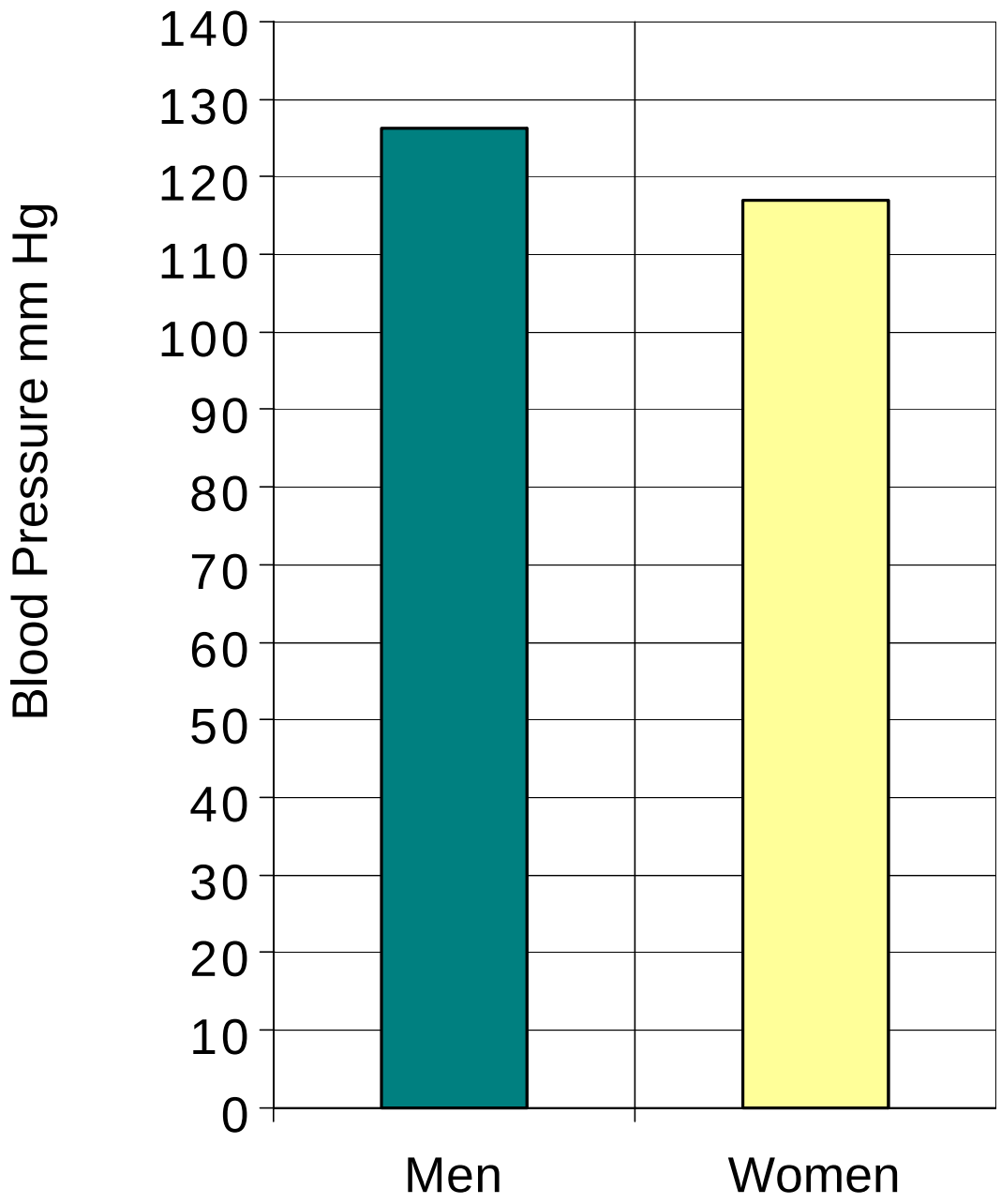


Figure 10. Main effect of Sex on Systolic Blood Pressure.

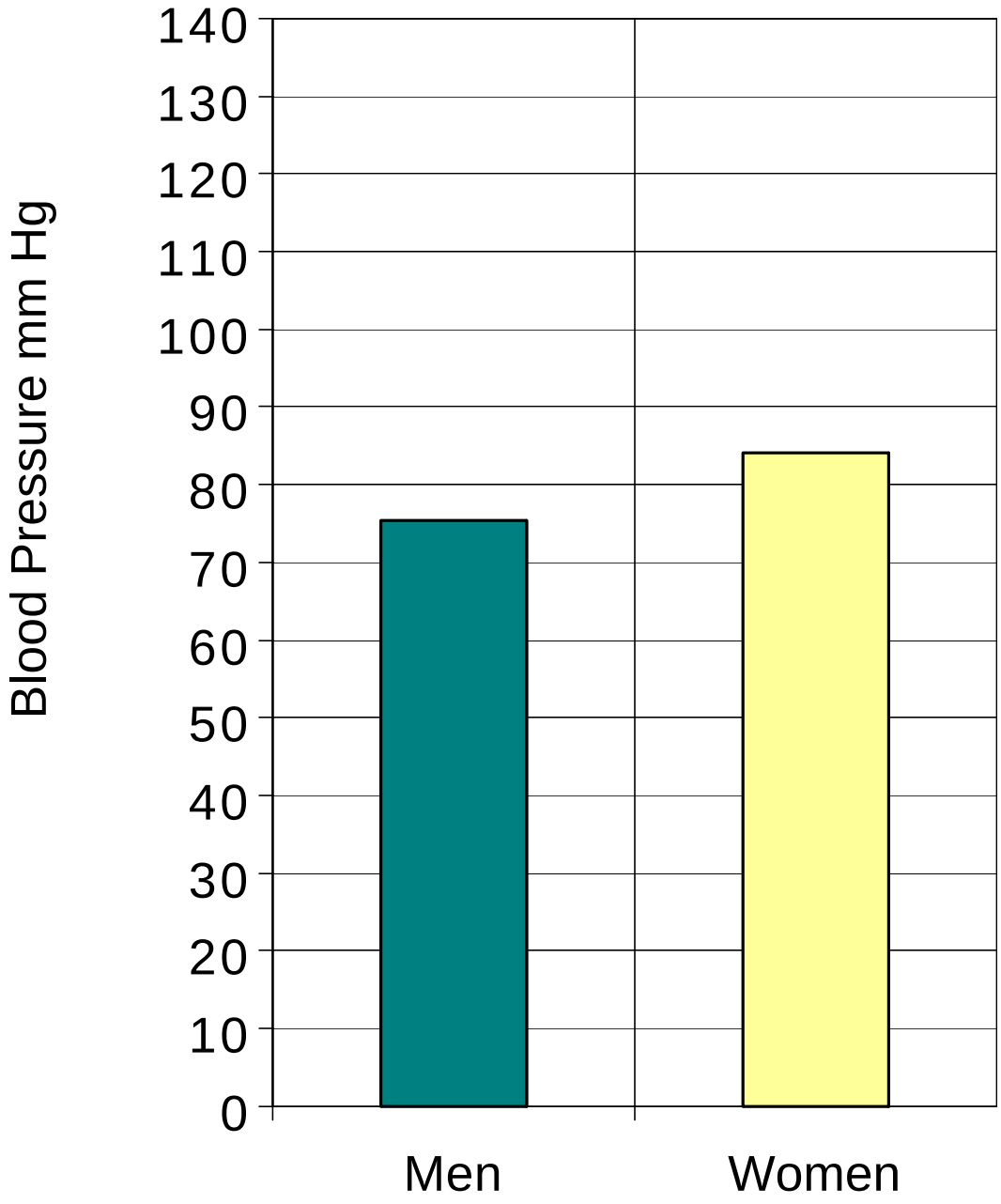


Figure 11. Main effect of Sex on Diastolic Blood Pressure.

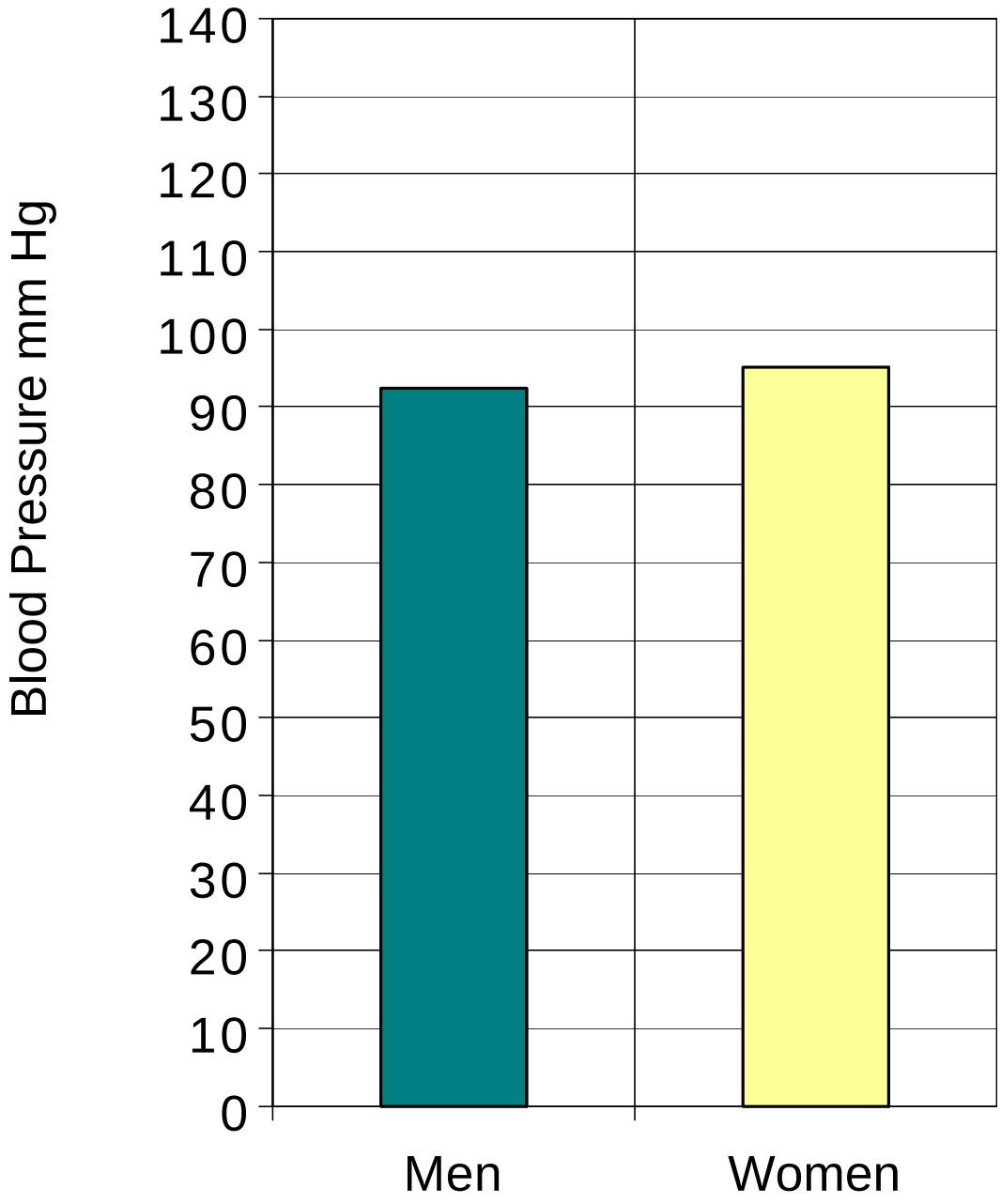


Figure 12. Main effect of Sex on Mean Arterial Pressure.

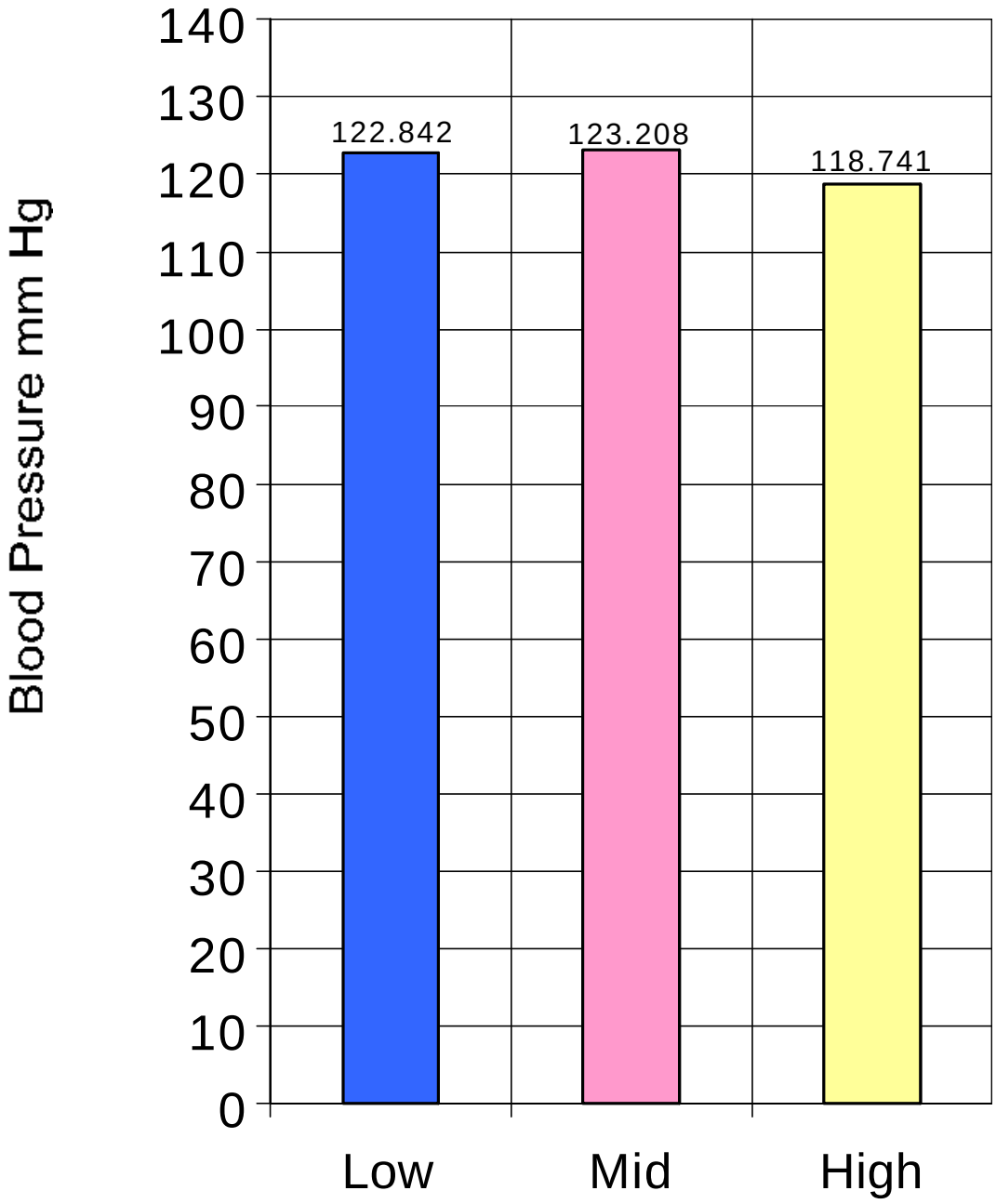


Figure 13. Main effect of Group on Systolic Blood Pressure.

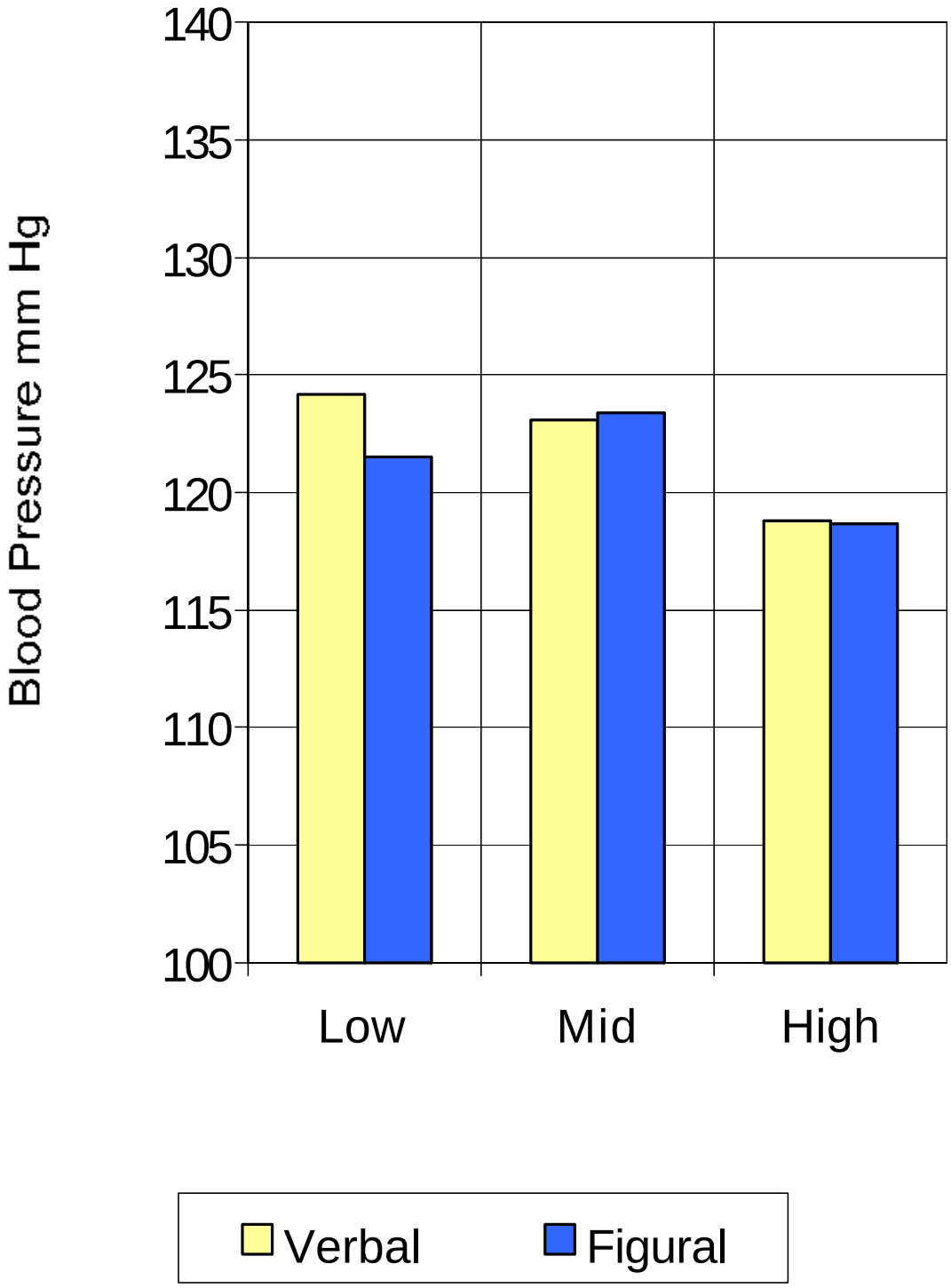


Figure 14. Group by Task Interaction Effect on Systolic Blood Pressure.

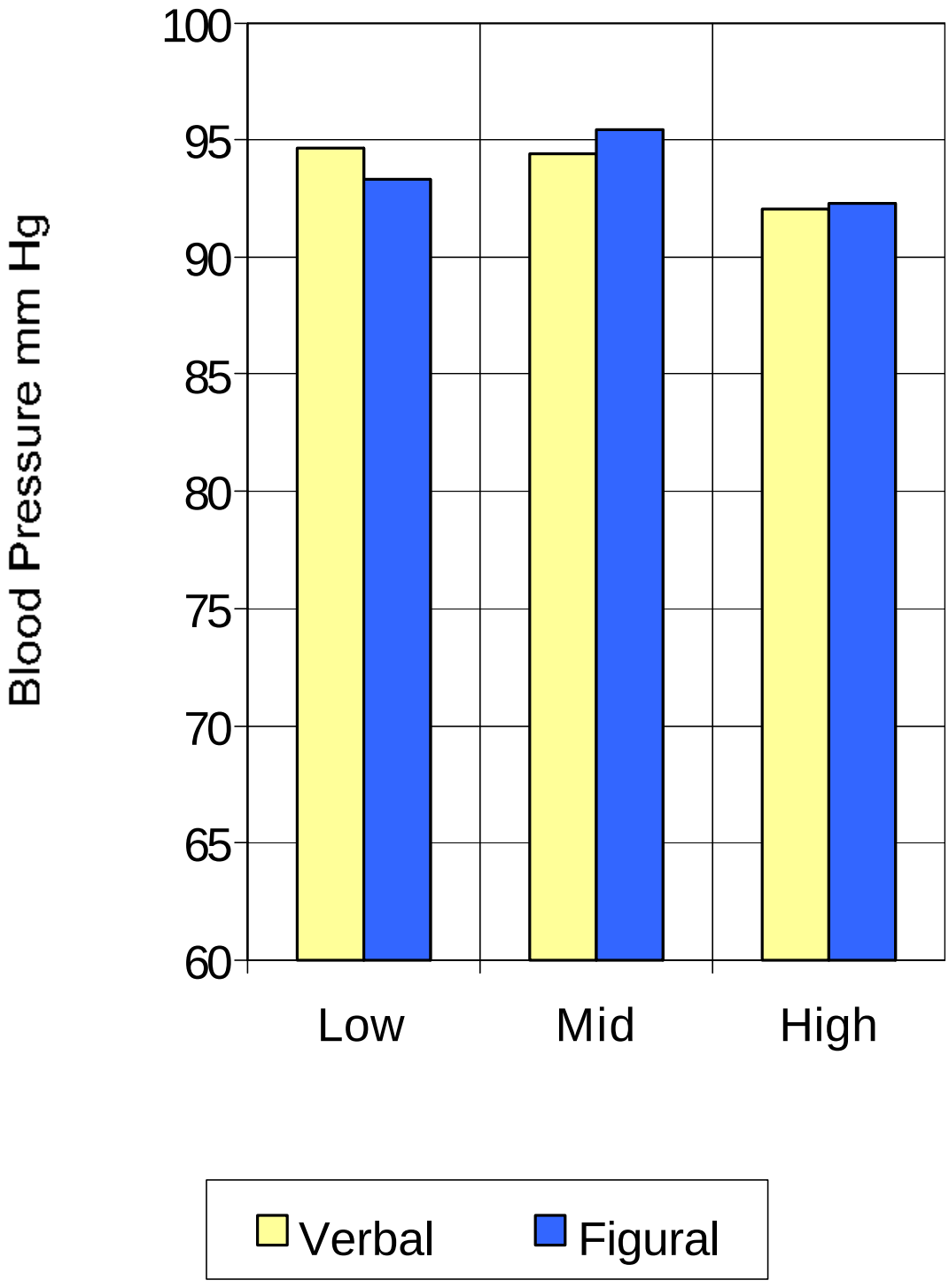


Figure 15. Group by Task Interaction Effect on Mean Arterial Pressure.

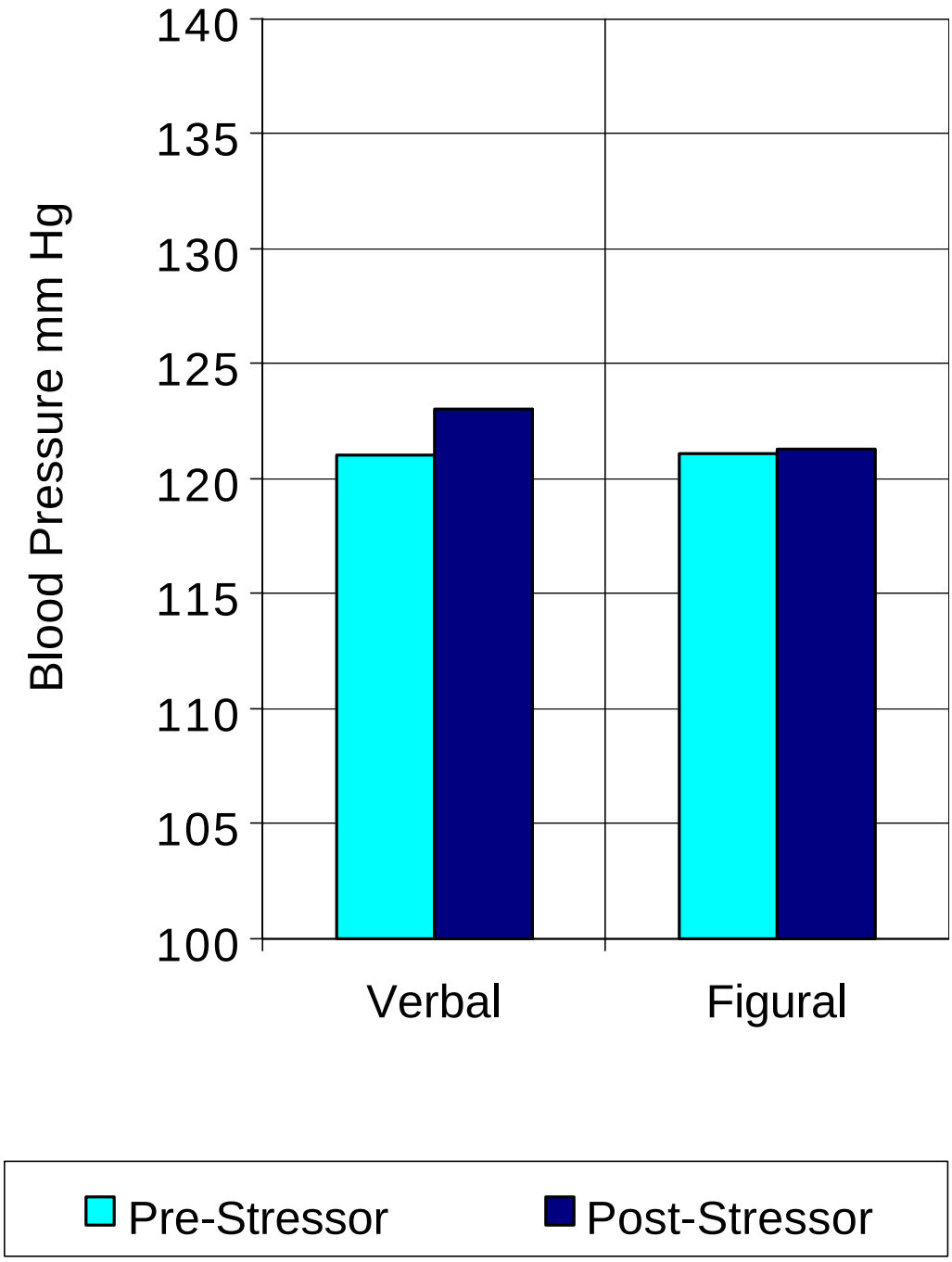


Figure 16. Task by Condition Interaction Effect on Systolic Blood Pressure.

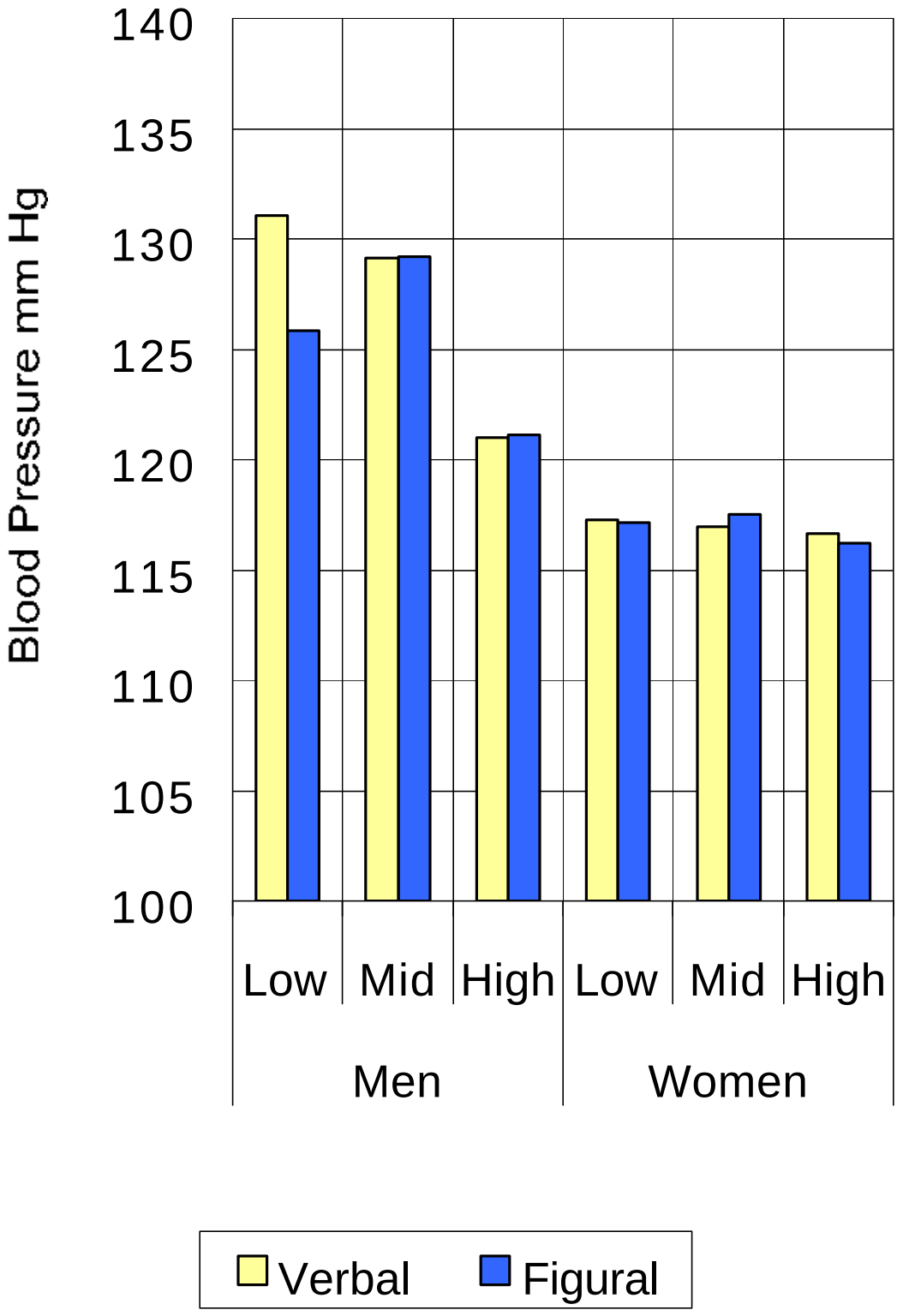


Figure 17. Sex by Group by Task Interaction Effect on Systolic Blood Pressure.

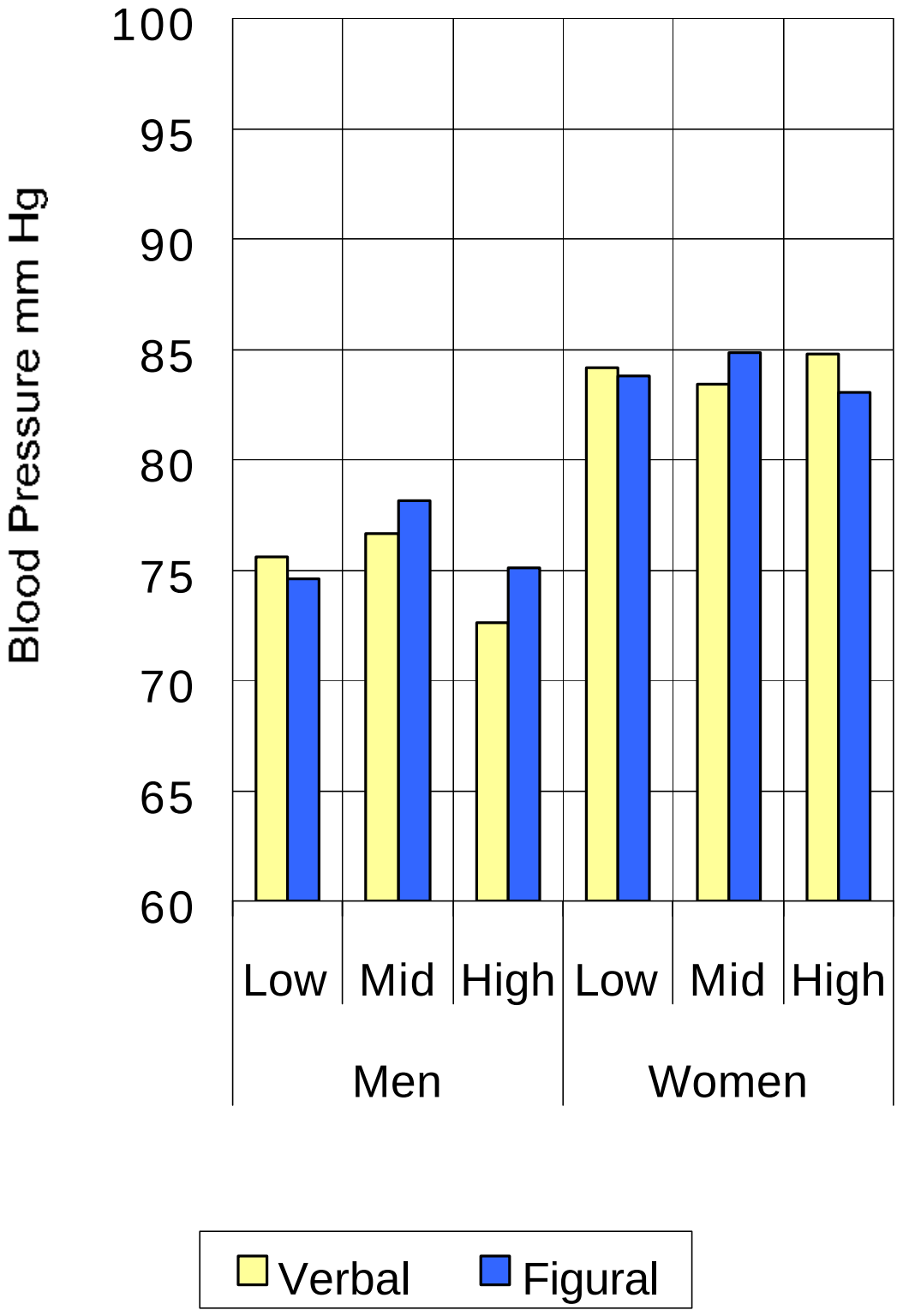


Figure 18. Sex by Group by Task Interaction Effect on Diastolic Blood Pressure.

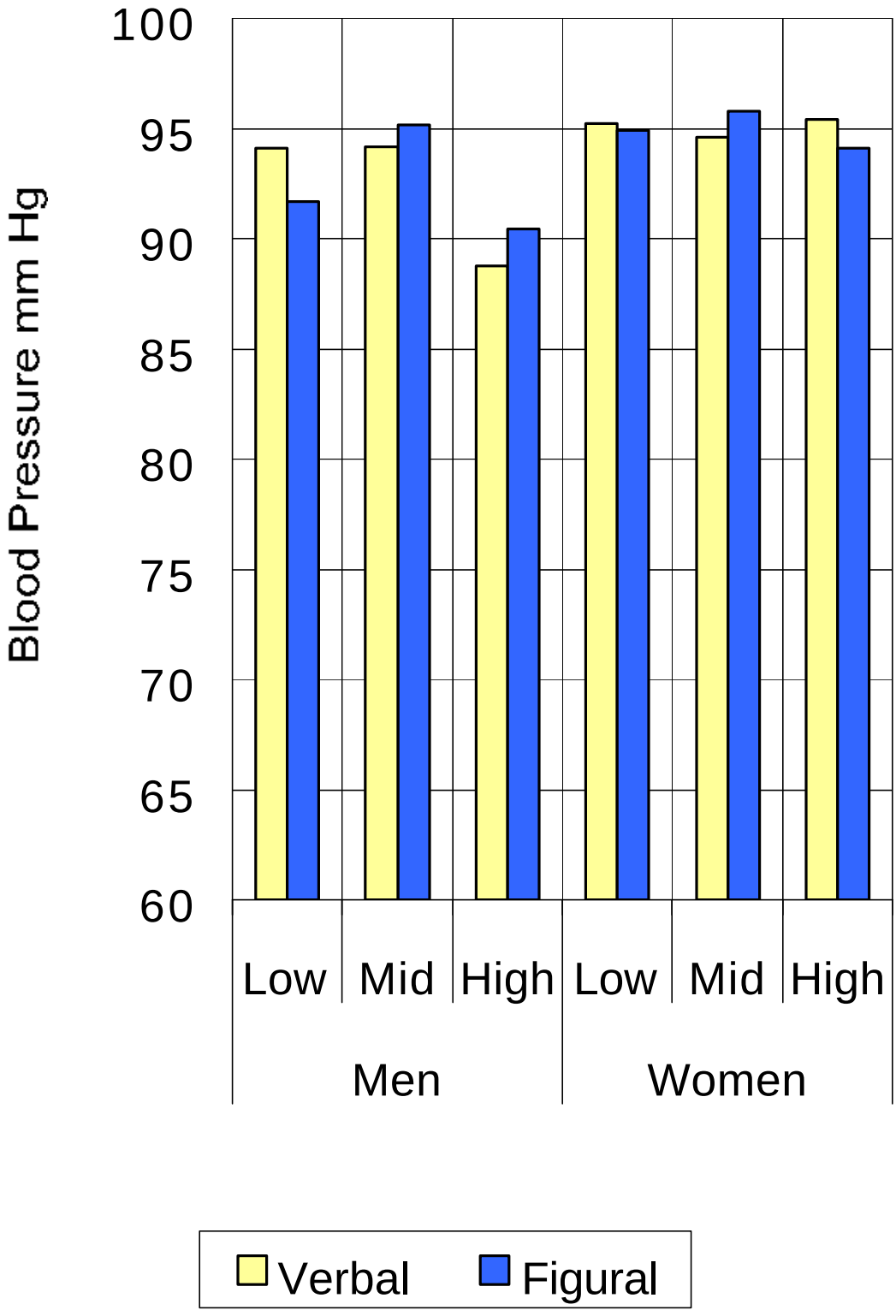


Figure 19. Sex by Group by Task Interaction Effect on Mean Arterial Pressure.

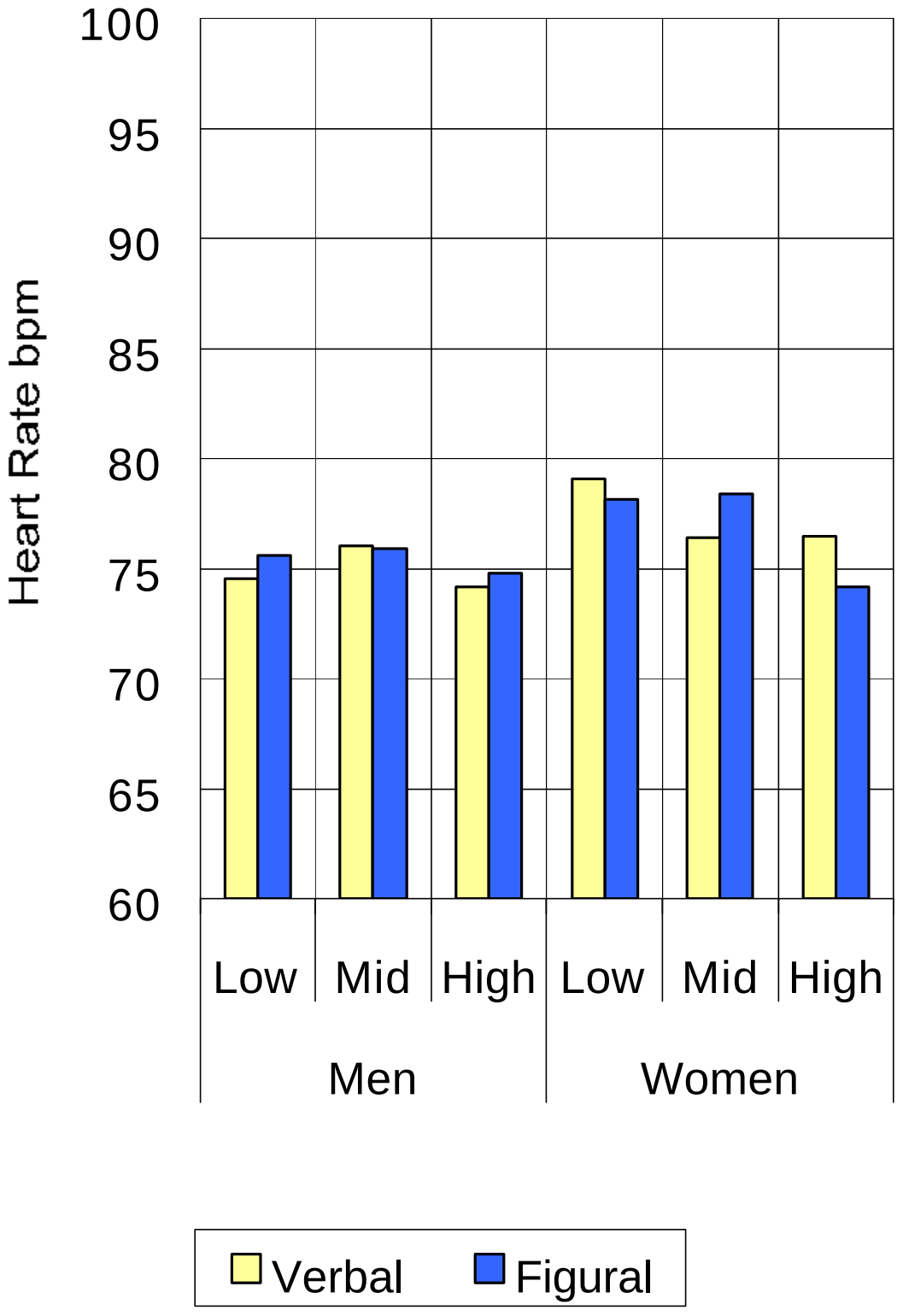


Figure 20. Sex by Group by Task Interaction Effect on Heart Rate.

Cardiovascular Reactivity to Verbal Fluency

In order to assess cardiovascular reactivity to the verbal fluency task, independent analyses of variance (ANOVA) were performed on the following dependent variables of cardiovascular reactivity: systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR). Data were analyzed for overall sex and group effects/interactions across conditions first, and then refined ANOVAs were performed to test group and condition effects within each sex and condition effects within each group. Group means and standard deviations of SBP, DBP, MAP, and HR measures are presented in Tables 7, 8, 9, and 10 respectively. Independent ANOVA results of these cardiovascular measures are presented in Table 11.

A main effect of sex was found for systolic blood pressure and diastolic blood pressure, as well as mean arterial pressure (derived from these two readings). Overall, men evidenced significantly higher SBP than women ($M = 127.07$ vs. 116.97, $F(1,120) = 37.74$, $p < 0.0001$). However, women evidenced significantly higher DBP than men ($M = 84.14$ vs. 74.97, $F(1,120) = 36.46$, $p < 0.0001$). In addition, women evidenced significantly higher MAP than men ($M = 95.09$ vs. 92.335, $F(1,120) = 4.54$, $p < 0.0351$). A main effect of sex was not found for HR.

A main effect of group was found only for SBP ($F(2,120) = 3.98$, $p < 0.0212$; Figure 21), where the low-hostile men and women had the highest SBP ($M = 124.19$), followed by the mid-hostile men and women ($M = 123.06$) and then high-hostile men and women ($M = 118.80$).

A main effect of condition was found for SBP ($F(1,120) = 13.44$, $p < 0.0004$), DBP ($F(1,120) = 5.56$, $p < 0.02$), and MAP ($F(1,120) = 14.36$, $p < 0.0002$), with significant *increases* occurring between conditions 1 and 2 (Figures 22, 23, and 24 respectively).

Also, a Sex by Group Interaction was evidenced for SBP only ($F(2,120) = 3.17$, $p < 0.0457$), wherein men's SBP was found to be at decreasing levels in increasingly hostile groups, while women's SBP remained relatively stable across groups (Table 12; Figure 25).

Table 7

Means and Standard Deviations of SBP by Sex, Group, and Condition (pre- and post-verbal fluency)

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Condition 1	130.4	9.5	116.5	10.8
Condition 2	131.8	10.7	118.1	8.2
Mid-Hostile Group				
Condition 1	127.5	11.9	117.0	7.1
Condition 2	130.8	11.5	116.9	10.0
High-Hostile Group				
Condition 1	119.5	9.5	115.2	8.1
Condition 2	122.4	8.5	118.0	9.9

Table 8

Means and Standard Deviations of DBP by Sex, Group, and Condition (pre- and post-verbal fluency)

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Condition 1	75.3	9.9	84.0	7.9
Condition 2	75.9	11.0	84.4	9.1
Mid-Hostile Group				
Condition 1	76.1	9.0	82.0	10.0
Condition 2	77.2	8.4	84.9	8.3
High-Hostile Group				
Condition 1	71.3	9.9	84.7	7.9
Condition 2	73.9	9.5	84.9	7.1

Table 9

Means and Standard Deviations of MAP by Sex, Group, and Condition (pre- and post-verbal fluency)

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Condition 1	93.7	8.2	94.8	7.3
Condition 2	94.5	8.5	95.6	7.8
Mid-Hostile Group				
Condition 1	93.2	8.6	93.7	8.1
Condition 2	95.1	8.6	95.6	7.3
High-Hostile Group				
Condition 1	87.4	5.9	94.9	7.0
Condition 2	90.1	6.3	95.9	6.7

Table 10

Means and Standard Deviations of HR by Sex, Group, and Condition (pre- and post-verbal fluency)

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Condition 1	73.2	12.1	78.6	13.4
Condition 2	75.9	11.9	79.5	9.9
Mid-Hostile Group				
Condition 1	76.3	14.0	75.5	14.5
Condition 2	75.7	12.6	77.3	13.2
High-Hostile Group				
Condition 1	74.5	10.4	77.4	10.5
Condition 2	73.9	9.9	75.5	10.5

Table 11

ANOVA Results for SBP, DBP, MAP, and HR Changes to the Verbal Fluency Task

Source	DF	SS	MS	F Value	p
Systolic BP					
Sex	(1,120)	6425.62	6425.62	37.74	<0.0001**
Group	(2,120)	1355.51	677.75	3.98	<0.0212*
Sex*Group	(2,120)	1078.28	539.14	3.17	<0.0457*
Condition	(1,120)	255.01	255.01	13.44	<0.0004**
Sex*Condition	(1,120)	20.29	20.29	1.07	<0.3032
Group*Condition	(2,120)	23.29	11.65	0.61	<0.5430
Sex*Group*Condition	(2,120)	41.61	20.80	1.10	<0.3374
Diastolic BP					
Sex	(1,120)	5302.92	5302.92	36.46	<0.0001**
Group	(2,120)	90.18	45.09	0.31	<0.7340
Sex*Group	(2,120)	314.60	157.30	1.08	<0.3424
Condition	(1,120)	102.86	102.86	5.56	<0.0200*
Sex*Condition	(1,120)	1.15	1.15	0.06	<0.8037
Group*Condition	(2,120)	23.82	11.91	0.64	<0.5268
Sex*Group*Condition	(2,120)	48.84	24.45	1.32	<0.2708
Mean Arterial Pressure					
Sex	(1,120)	476.44	476.43	4.54	<0.0351*
Group	(2,120)	338.85	169.43	1.62	<0.2031
Sex*Group	(2,120)	488.54	244.27	2.33	<0.1018
Condition	(1,120)	146.03	146.03	14.36	<0.0002**
Sex*Condition	(1,120)	4.91	4.91	0.48	<0.4886
Group*Condition	(2,120)	14.96	7.48	0.74	<0.4813
Sex*Group*Condition	(2,120)	10.15	5.07	0.50	<0.6083
Heart Rate					
Sex	(1,120)	355.95	355.95	1.32	<0.2526
Group	(2,120)	95.04	47.52	0.18	<0.8385
Sex*Group	(2,120)	178.66	89.33	0.33	<0.7184
Condition	(1,120)	7.51	7.51	0.39	<0.5324
Sex*Condition	(1,120)	0.72	0.72	0.04	<0.8463
Group*Condition	(2,120)	96.86	48.43	2.53	<0.0840
Sex*Group*Condition	(2,120)	53.97	26.99	1.41	<0.2484

* $p < .05$
** $p < .01$

Table 12

Means and Standard Deviations of SBP for Sex by Group Interaction across verbal fluency task.

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group	131.1	10.0	117.3	9.5
Mid-Hostile Group	129.2	11.7	117.0	8.5
High-Hostile Group	121.0	9.0	116.6	9.1

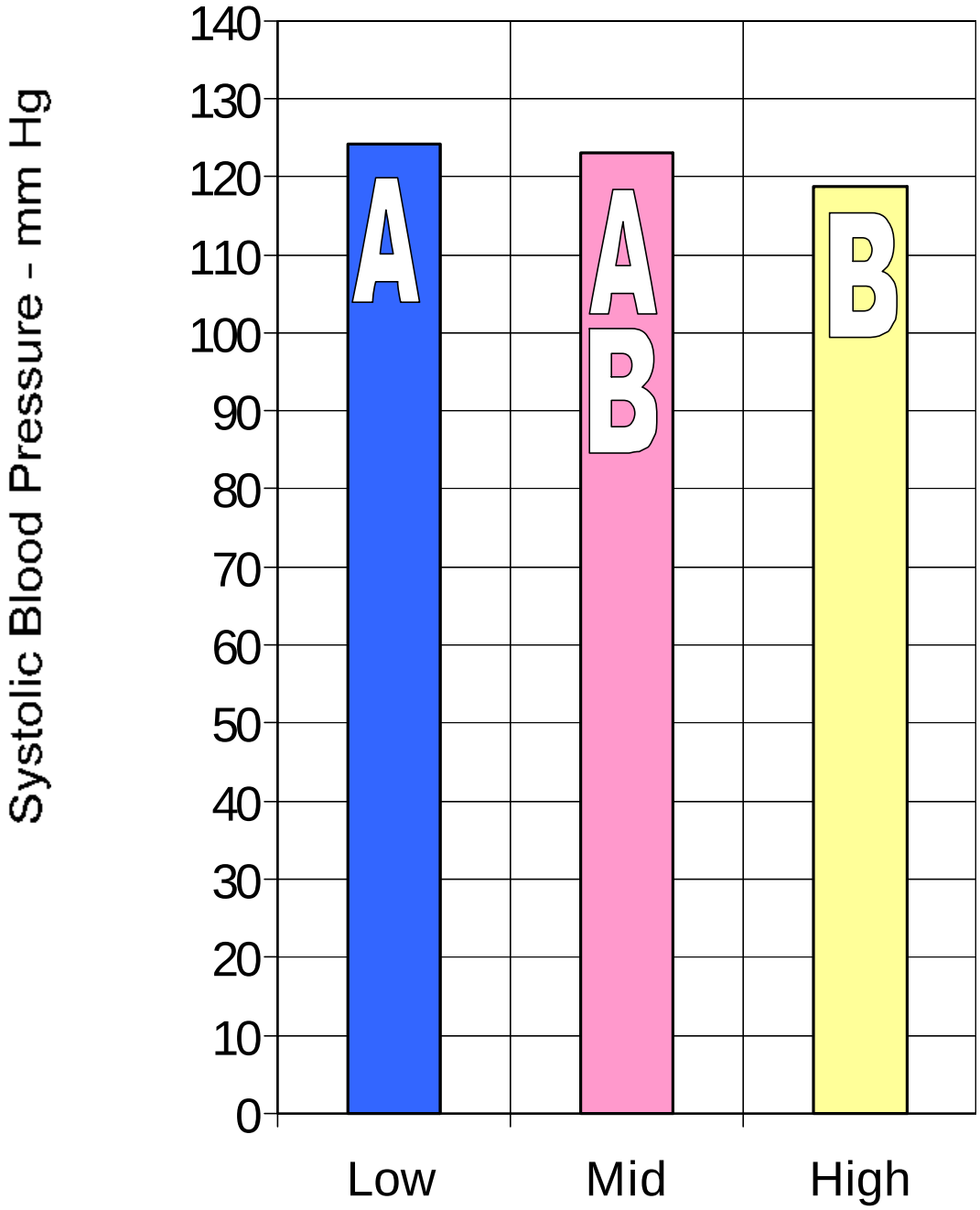


Figure 21. Main Effect of Group on Systolic Blood Pressure Across Verbal Fluency Stressor.

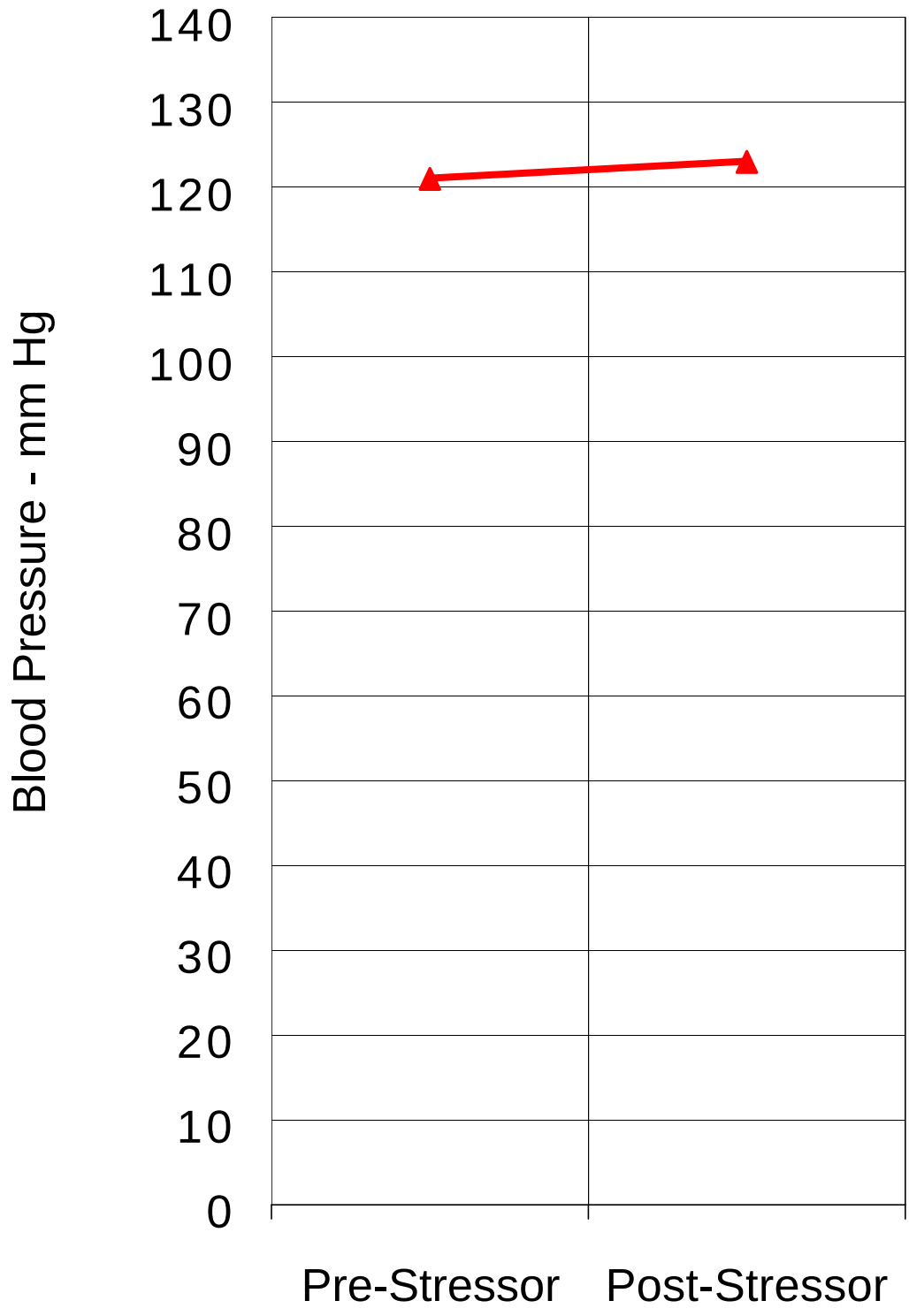


Figure 22. Main Effect of Condition on SBP for Verbal Fluency.

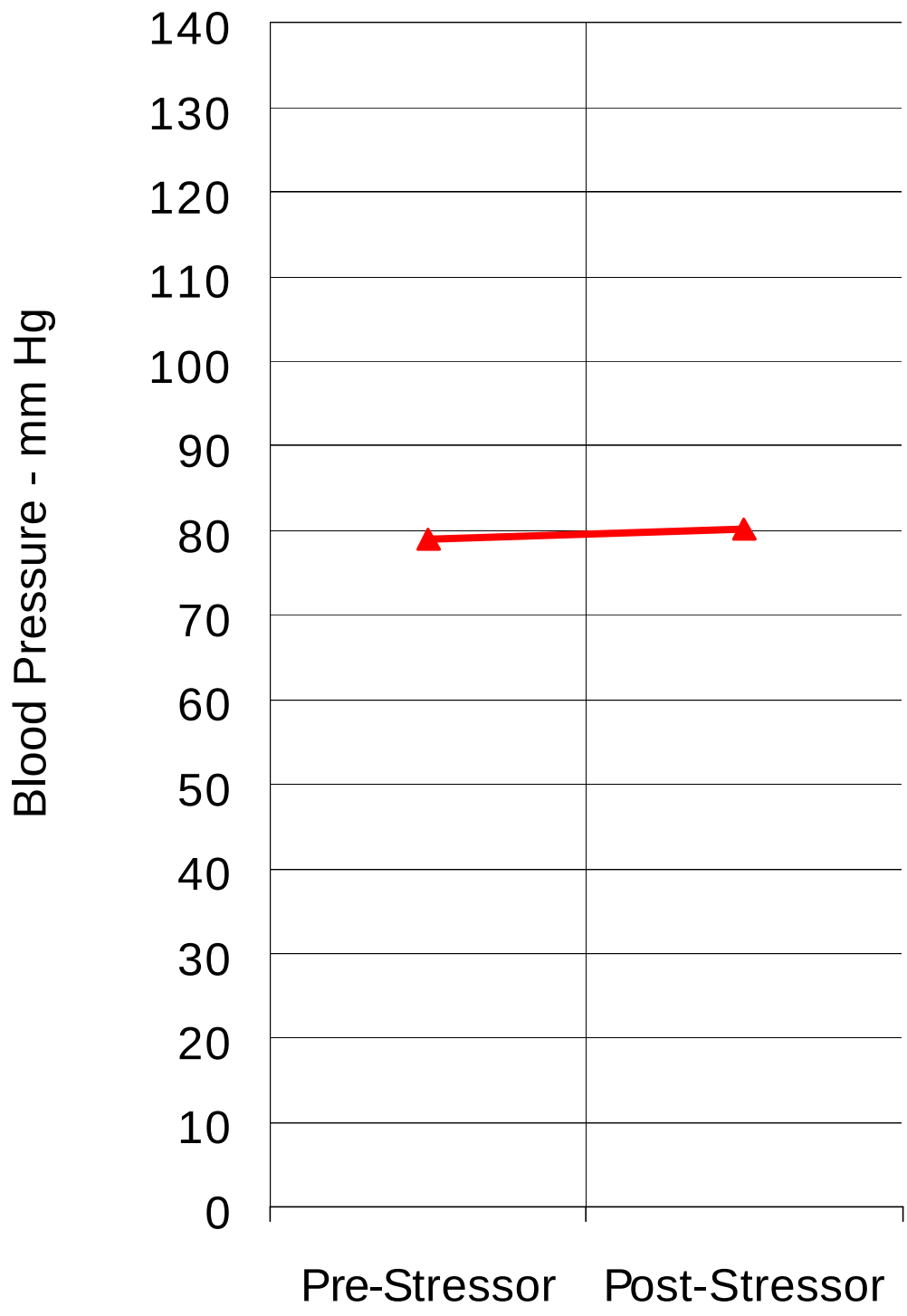


Figure 23. Main Effect of Condition on DBP for Verbal Fluency.

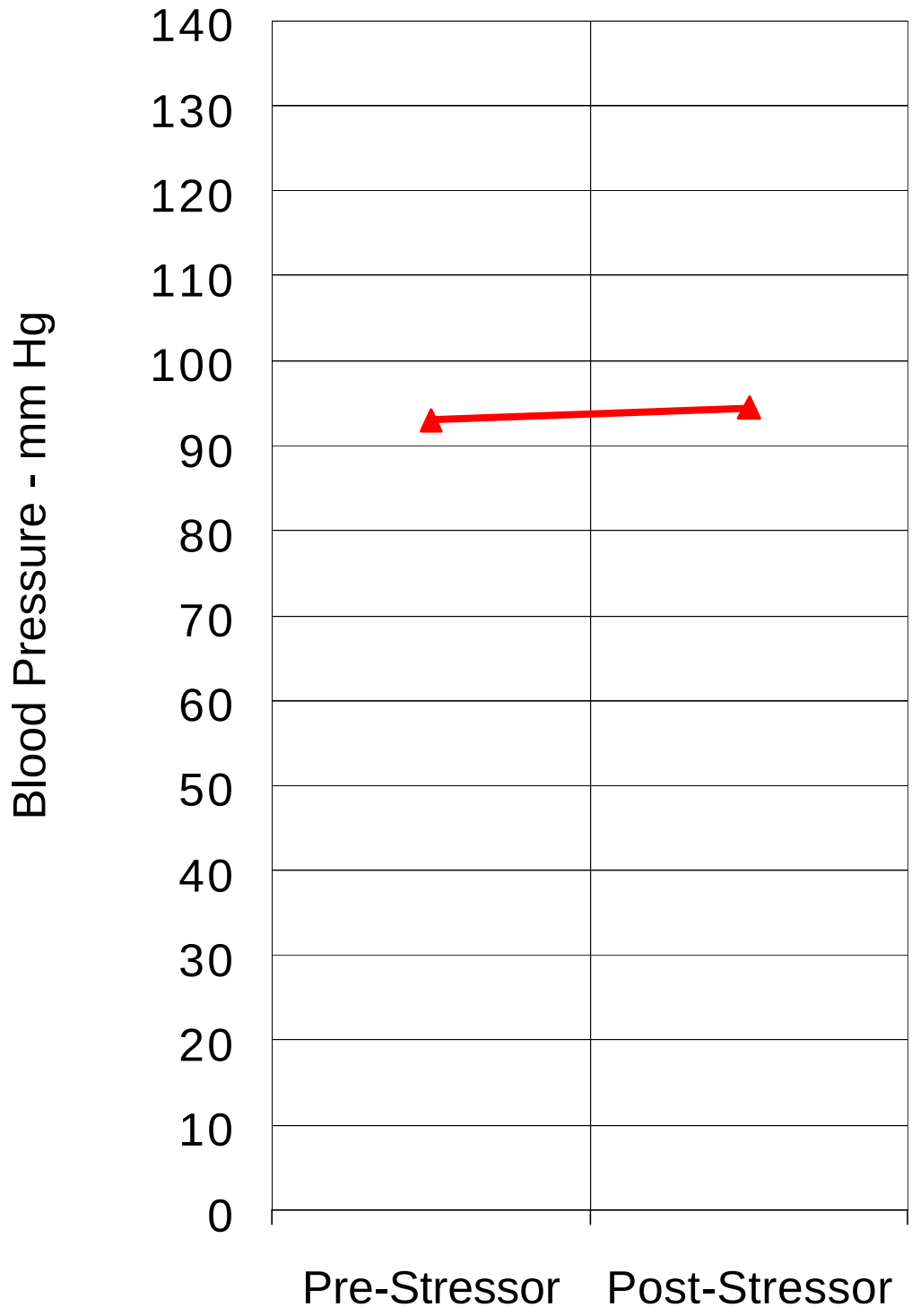


Figure 24. Main Effect of Condition on MAP for Verbal Fluency.

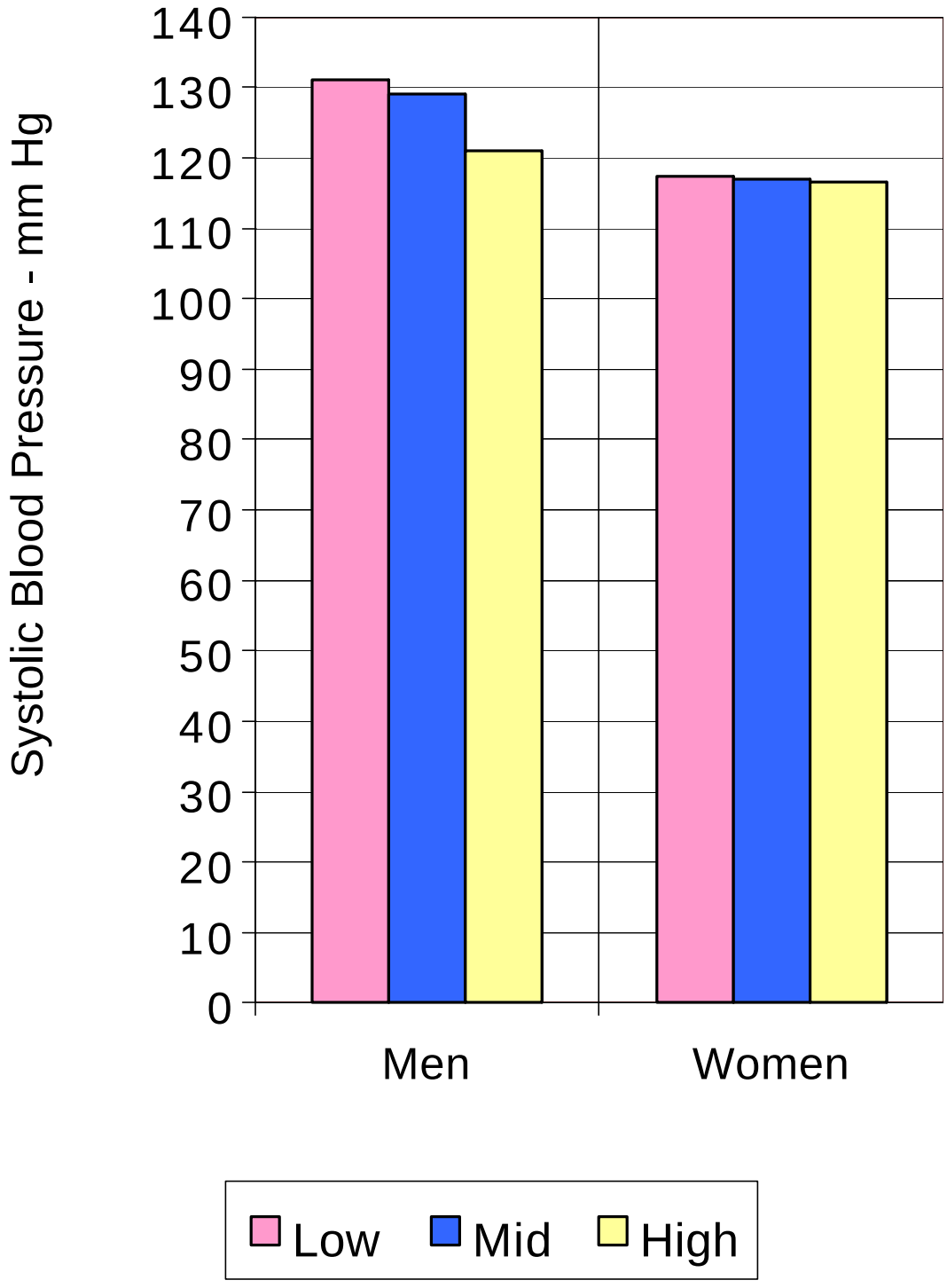


Figure 25. Sex by Group Interaction for SBP during Verbal Fluency Stressor Task.

Cardiovascular Reactivity to Figural Fluency

In order to assess cardiovascular reactivity to the figural fluency task, independent analyses of variance (ANOVA) were performed on the following dependent variables of cardiovascular reactivity: systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR). Data were analyzed for overall sex and group effects/interactions across conditions first, and then refined ANOVAs were performed to test group and condition effects within each sex and condition effects within each group. Group means and standard deviations of SBP, DBP, MAP, and HR measures are presented in Tables 13, 14, 15, and 16 respectively. Independent ANOVA results of these cardiovascular measures are presented in Table 17.

A main effect of sex was found for systolic blood pressure and diastolic blood pressure. Overall, men evidenced significantly higher SBP than women ($M = 125.4$ vs. 117.0, $F(1,120) = 24.01$, $p < 0.0001$). However, women evidenced significantly higher DBP than men ($M = 76.0$ vs. 83.9, $F(1,120) = 26.16$, $p < 0.0001$). Main effects of sex were not found for MAP or HR.

No main effects of group were found.

A main effect of condition was found for DBP ($F(1,120) = 9.40$, $p < 0.0027$) and MAP ($F(1,120) = 6.49$, $p < 0.0121$), with significant *increases* occurring between conditions 1 and 2 (Figures 26 and 27, respectively).

Table 13

Means and Standard Deviations of SBP by Sex, Group, and Condition (pre- and post-figural fluency)

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Condition 1	125.4	8.9	117.3	9.0
Condition 2	126.3	8.9	116.9	9.6
Mid-Hostile Group				
Condition 1	129.5	11.8	117.3	10.1
Condition 2	128.9	11.0	117.8	8.9
High-Hostile Group				
Condition 1	120.4	11.5	116.6	11.0
Condition 2	121.9	10.3	115.8	10.5

Table 14

Means and Standard Deviations of DBP by Sex, Group, and Condition (pre- and post-figural fluency)

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Condition 1	74.2	9.7	82.4	9.8
Condition 2	75.0	8.1	85.3	8.9
Mid-Hostile Group				
Condition 1	77.8	10.9	84.9	9.9
Condition 2	78.5	9.6	84.9	8.3
High-Hostile Group				
Condition 1	74.1	8.5	81.7	7.9
Condition 2	76.1	11.1	84.4	6.2

Table 15

Means and Standard Deviations of MAP by Sex, Group, and Condition (pre- and post-figural fluency)

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Condition 1	91.3	6.9	94.0	8.4
Condition 2	92.1	6.6	95.8	7.9
Mid-Hostile Group				
Condition 1	95.0	10.1	95.7	8.4
Condition 2	95.3	9.3	95.9	6.8
High-Hostile Group				
Condition 1	89.6	6.5	93.3	7.8
Condition 2	91.3	7.1	94.9	6.6

Table 16

Means and Standard Deviations of HR by Sex, Group, and Condition (pre- and post-figural fluency)

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Condition 1	75.3	10.4	77.7	13.1
Condition 2	76.0	11.7	79.5	9.9
Mid-Hostile Group				
Condition 1	75.8	13.0	77.7	13.5
Condition 2	76.0	12.0	79.0	14.5
High-Hostile Group				
Condition 1	74.9	10.8	73.3	10.8
Condition 2	74.7	10.4	75.0	11.4

Table 17

ANOVA Results for SBP, DBP, MAP, and HR Changes to the Figural Fluency Task

Source	DF	SS	MS	F Value	p
Systolic BP					
Sex	(1,120)	4488.22	4488.22	24.01	<0.0001**
Group	(2,120)	932.04	466.02	2.49	<0.0870
Sex*Group	(2, 120)	479.29	239.65	1.28	<0.2813
Condition	(1,120)	2.01	2.01	0.10	<0.7522
Sex*Condition	(1,120)	12.22	12.22	0.61	<0.4366
Group*Condition	(2,120)	1.33	0.66	0.03	<0.9675
Sex*Group*Condition	(2,120)	30.61	15.31	0.76	<0.4686
Diastolic BP					
Sex	(1,120)	3996.08	3996.08	26.16	<0.0001**
Group	(2,120)	316.47	158.24	1.04	<0.3580
Sex*Group	(2,120)	65.01	32.50	0.21	<0.8086
Condition	(1,120)	144.01	144.01	9.40	<0.0027**
Sex*Condition	(1,120)	9.33	9.33	0.61	<0.4367
Group*Condition	(2,120)	45.26	22.62	1.48	<0.2325
Sex*Group*Condition	(2,120)	19.01	9.51	0.62	<0.5395
Mean Arterial Pressure					
Sex	(1,120)	392.50	392.50	3.59	<0.0606
Group	(2,120)	443.81	221.91	2.03	<0.1361
Sex*Group	(2,120)	115.70	57.85	0.53	<0.5907
Condition	(1,120)	71.79	71.79	6.49	<0.0121*
Sex*Condition	(1,120)	0.76	0.76	0.07	<0.7937
Group*Condition	(2,120)	23.70	11.85	1.07	<0.3458
Sex*Group*Condition	(2,120)	4.40	2.20	0.20	<0.8198
Heart Rate					
Sex	(1,120)	133.62	133.62	0.49	<0.4855
Group	(2,120)	370.47	185.24	0.68	<0.5093
Sex*Group	(2,120)	135.86	67.93	0.25	<0.7801
Condition	(1,120)	36.95	36.95	2.29	<0.1332
Sex*Condition	(1,120)	18.08	18.08	1.12	<0.2925
Group*Condition	(2,120)	0.20	0.10	0.01	<0.9937
Sex*Group*Condition	(2,120)	5.91	2.96	0.18	<0.8332

* $p < .05$
** $p < .01$

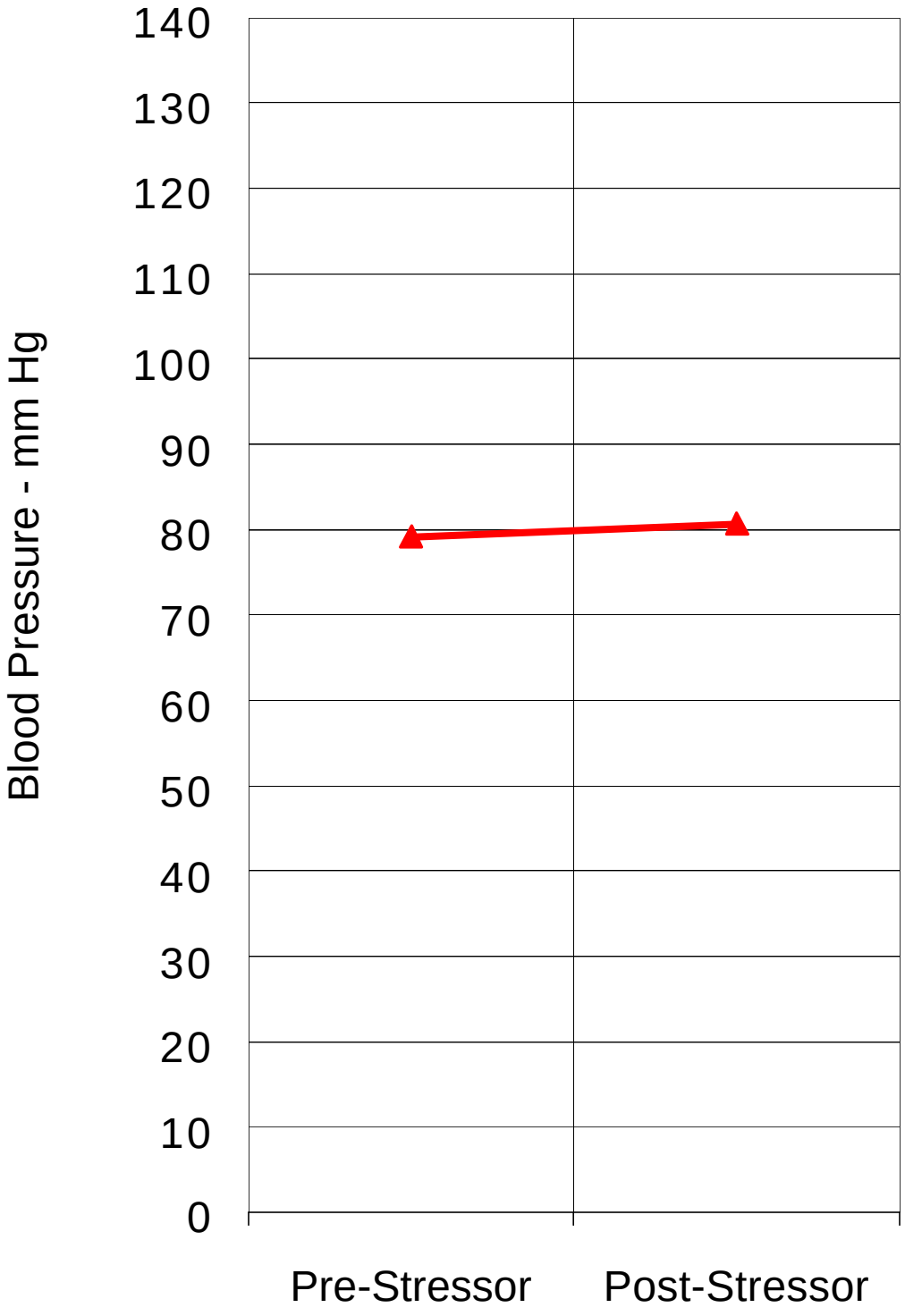


Figure 26. Main Effect of Condition on DBP for Figural Fluency Task.

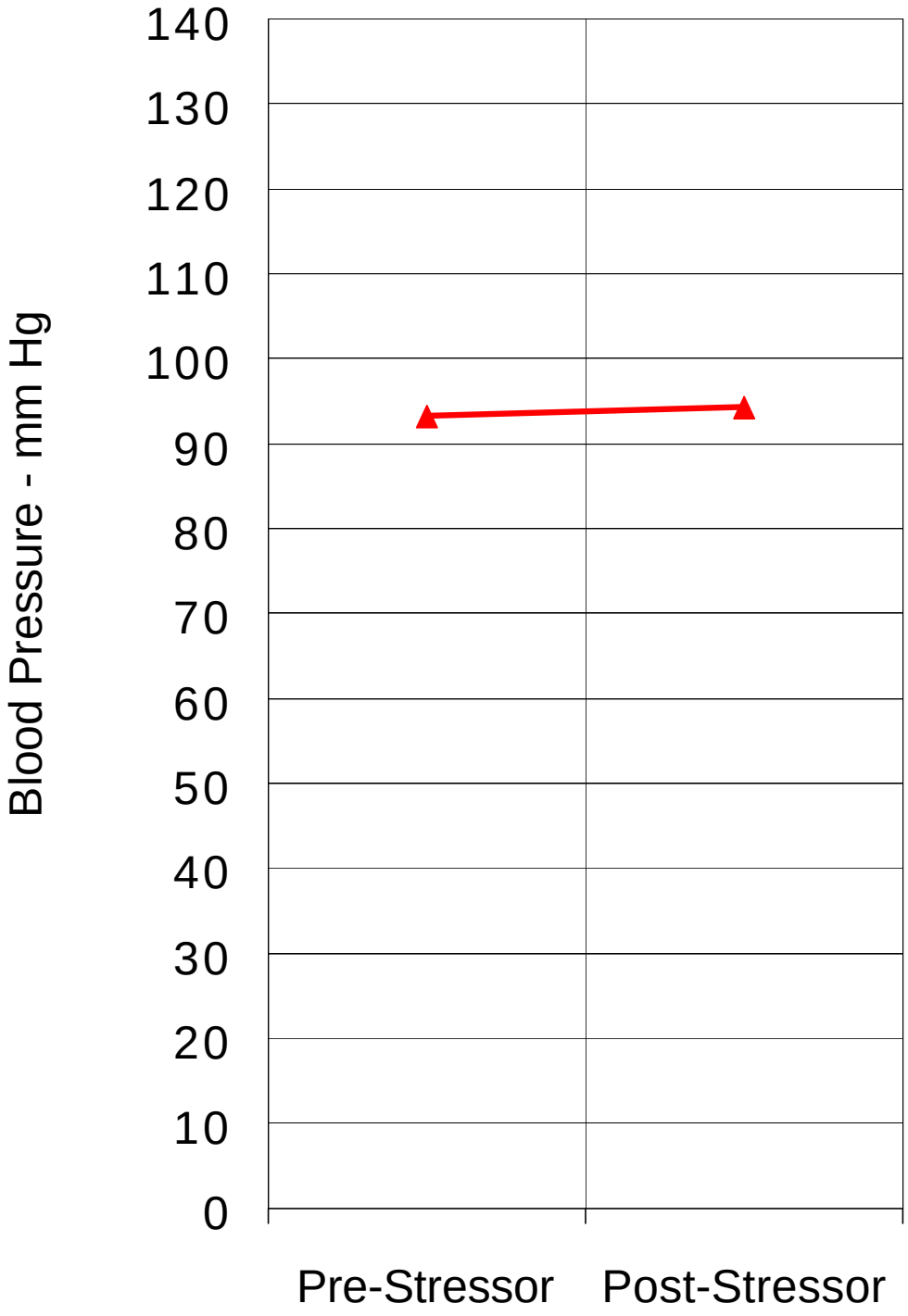


Figure 27. Main Effect of Condition on MAP for Figural Fluency Task.

Verbal Fluency

In order to analyze verbal fluency, an independent analysis of variance (ANOVA) was performed on the verbal fluency total score. Data were analyzed for overall sex and group effects/interactions across trials first, and then refined ANOVAs were performed to test group and trial effects within each sex and trial effects within each group. Group means and standard deviations of verbal fluency are presented in Table 18; independent ANOVA results are presented in Table 19.

No main effects of sex or of group were found for verbal fluency; however, a main effect of trial was found for verbal fluency ($F(2,120) = 23.29$, $p < 0.0001$). Trial 2 ($M = 14.17$) was significantly different from Trials 1 and 3 ($M = 12.60$ and 12.91, respectively), which were not significantly different from each other.

A refined ANOVA revealed a Sex by Group Interaction Effect on Trial 1 of verbal fluency ($F(2,120) = 3.82$, $p < 0.0247$; Table 20; Figure 28).

Table 18

Means and Standard Deviations of Verbal Fluency by Sex, Group, and Trial

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Trial 1	11.9	2.5	14.0	2.9
Trial 2	14.0	2.2	14.5	3.2
Trial 3	12.8	2.8	12.7	3.8
Mid-Hostile Group				
Trial 1	12.5	2.6	13.6	3.9
Trial 2	13.8	3.7	14.2	2.9
Trial 3	13.2	3.0	13.5	3.5
High-Hostile Group				
Trial 1	12.5	2.9	11.0	3.1
Trial 2	14.7	3.4	13.8	3.8
Trial 3	13.5	2.9	11.8	4.0

Table 19

ANOVA Results for Verbal Fluency Scores by Sex, Group, and Trial

Source	DF	SS	MS	F Value	p
Sex	(1,120)	0.02	0.02	0.00	<0.9747
Group	(2,120)	25.15	12.57	0.53	<0.5876
Sex*Group	(2,120)	90.59	45.29	1.92	<0.1505
Trial	(2,240)	173.99	86.99	23.29	<0.0001**
Sex*Trial	(2,240)	17.33	8.67	2.32	<0.1004
Group*Trial	(4,240)	30.77	7.69	2.06	<0.0868
Sex*Group*Trial	(4,240)	16.13	4.03	1.08	<0.3673

* $p < .05$
** $p < .01$

Table 20

Means and Standard Deviations of Verbal Fluency Trial 1 score (Sex by Group Interaction Effect).

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Trial 1	11.9	2.5	14.0	2.9
Mid-Hostile Group				
Trial 1	12.5	2.6	13.6	3.9
High-Hostile Group				
Trial 1	12.5	2.9	11.0	3.1

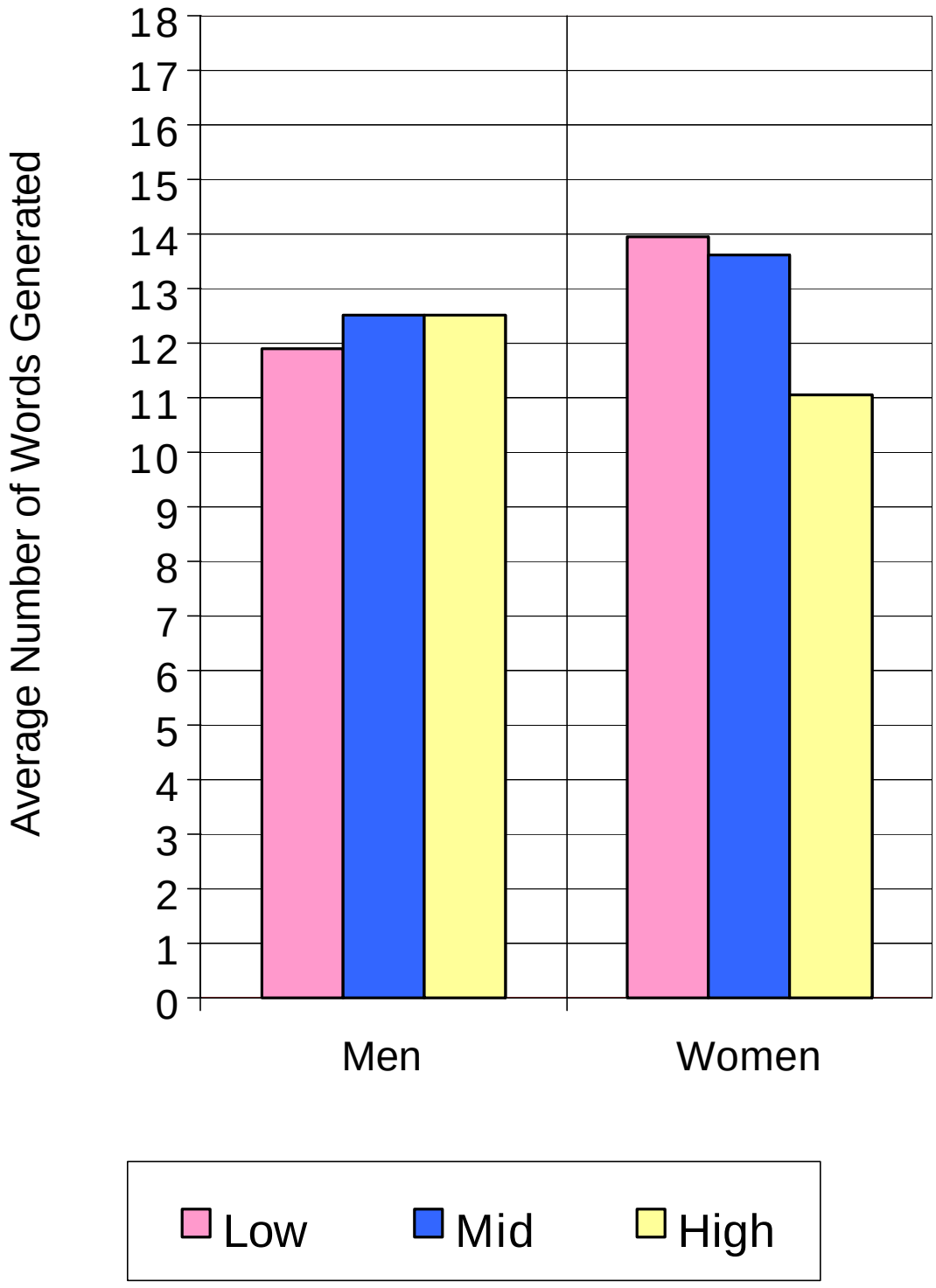


Figure 28. Sex by Group Interaction Effect on Verbal Fluency Trial 1 Score.

Verbal Perseveration

In order to analyze verbal perseveration, an independent analysis of variance (ANOVA) was performed on the verbal perseveration (repeated words : words generated) score. Data were analyzed for overall sex and group effects/interactions across trials first, and then refined ANOVAs were performed to test group and trial effects within each sex and trial effects within each group. Group means and standard deviations of verbal perseveration are presented in Table 21; independent ANOVA results are presented in Table 22.

No main effects were found for verbal perseveration; however, a sex by trial interaction effect was found ($F(2,240) = 3.19$, $p < 0.0429$; Figure 29).

Table 21

Means and Standard Deviations of Verbal Perseveration by Sex, Group, and Trial (expressed as a percentage)

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Trial 1	0.00	0.00	0.00	0.00
Trial 2	0.00	0.00	0.00	0.00
Trial 3	0.43	0.02	0.00	0.00
Mid-Hostile Group				
Trial 1	0.00	0.00	0.92	0.03
Trial 2	0.30	0.01	0.00	0.00
Trial 3	0.68	0.03	0.00	0.00
High-Hostile Group				
Trial 1	0.00	0.00	0.34	0.02
Trial 2	1.19	0.02	0.25	0.01
Trial 3	0.00	0.00	0.00	0.00

Table 22

ANOVA Results for Verbal Perseveration by Sex, Group, and Trial

Source	DF	SS	MS	F Value	p
Sex	(1,120)	0.00013916	0.00013916	0.66	<0.4194
Group	(2,120)	0.00046348	0.00023174	1.09	<0.3384
Sex*Group	(2,120)	0.00005355	0.00002678	0.13	<0.8814
Trial	(2,240)	0.00007502	0.00003751	0.17	<0.8414
Sex*Trial	(2,240)	0.00138485	0.00069243	3.19	<0.0429*
Group*Trial	(4,240)	0.00145372	0.00036343	1.67	<0.1565
Sex*Group*Trial	(4,240)	0.00113265	0.00028316	1.30	<0.2688

* $p < .05$
** $p < .01$

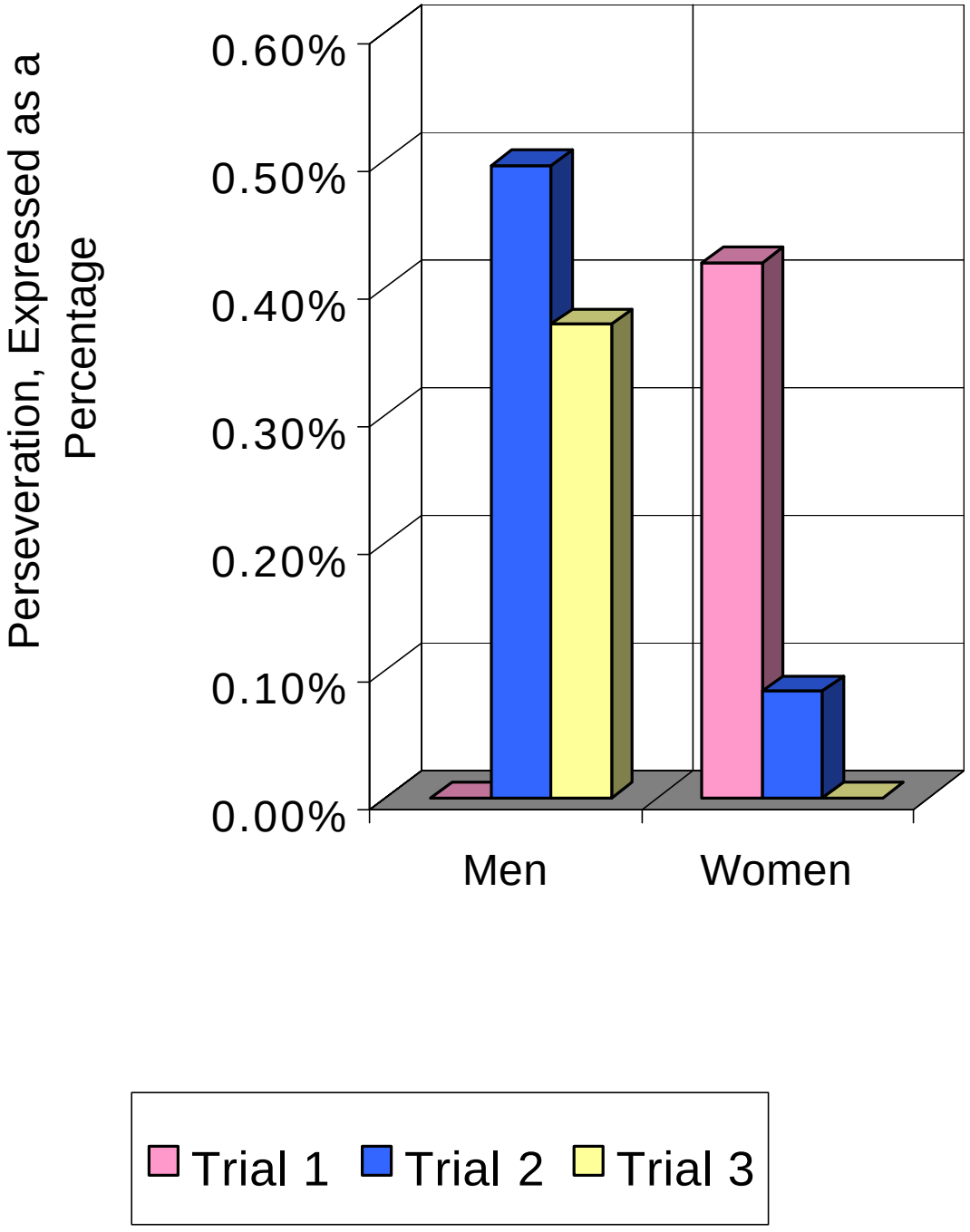


Figure 29. Sex by Trial Interaction Effect on Verbal Perseveration.

Figural Fluency

In order to analyze figural fluency, an independent analysis of variance (ANOVA) was performed on the figural fluency total score. Data were analyzed for overall sex and group effects/interactions across trials first, and then refined ANOVAs were performed to test group and trial effects within each sex and trial effects within each group. Group means and standard deviations of figural fluency are presented in Table 23; independent ANOVA results are presented in Table 24.

No main effects of sex or of group were found for figural fluency; however, a main effect of trial was found for figural fluency ($F(2,240) = 30.62$, $p < 0.0001$). Trial 1 ($M = 13.72$) was significantly different from Trials 2 and 3 ($M = 15.07$ and 15.52, respectively), which were not significantly different from each other.

Table 23

Means and Standard Deviations of Verbal Fluency by Sex, Group, and Trial

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Trial 1	13.5	5.3	12.5	4.0
Trial 2	15.2	5.4	13.4	4.6
Trial 3	14.9	5.2	13.2	4.3
Mid-Hostile Group				
Trial 1	14.6	6.2	14.6	5.6
Trial 2	15.9	6.5	15.9	5.4
Trial 3	17.2	7.1	16.9	4.9
High-Hostile Group				
Trial 1	14.1	4.7	13.0	5.7
Trial 2	15.7	5.5	14.4	6.0
Trial 3	16.5	5.8	14.4	5.8

Table 24

ANOVA Results for Figural Fluency Scores by Sex, Group, and Trial

Source	DF	SS	MS	F Value	p
Sex	(1,120)	104.76	104.76	1.25	<0.2655
Group	(2,120)	263.54	131.77	1.57	<0.2115
Sex*Group	(2,120)	39.15	19.57	0.23	<0.7919
Trial	(2,240)	219.87	109.94	30.62	<0.0001**
Sex*Trial	(2,240)	8.40	4.20	1.17	<0.3121
Group*Trial	(4,240)	29.44	7.36	2.05	<0.0881
Sex*Group*Trial	(4,240)	3.90	0.97	0.27	<0.8962

Figural Perseveration

In order to analyze figural perseveration, an independent analysis of variance (ANOVA) was performed on the figural perseveration (repeated designs : unique designs) score. Data were analyzed for overall sex and group effects/interactions across trials first, and then refined ANOVAs were performed to test group and trial effects within each sex and trial effects within each group. Group means and standard deviations of figural perseveration are presented in Table 25; independent ANOVA results are presented in Table 26.

A main effect of Trial was found for figural perseveration ($F(2,240) = 5.81$, $p < 0.0034$), wherein Trial 3 (M = 6.7%) was significantly different from Trials 1 and 2 (M = 3.8% and 4.0%, respectively), which were not significantly different from each other.

In addition, a main effect of task was found for perseveration (across sex and group), as participants perseverated more on the figural fluency task than the verbal fluency task (Figure 30).

Table 25

Means and Standard Deviations of Figural Perseveration by Sex, Group, and Trial (expressed as a percentage)

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Trial 1	3.62	0.04	2.22	0.06
Trial 2	5.90	0.06	3.50	0.06
Trial 3	7.44	0.11	4.78	0.09
Mid-Hostile Group				
Trial 1	3.17	0.05	5.05	0.07
Trial 2	3.30	0.05	4.89	0.10
Trial 3	6.40	0.18	8.31	0.08
High-Hostile Group				
Trial 1	4.03	0.08	4.91	0.09
Trial 2	2.21	0.05	3.95	0.06
Trial 3	6.13	0.06	7.19	0.09

Table 26

ANOVA Results for Figural Perseveration by Sex, Group, and Trial

Source	DF	SS	MS	F Value	p
Sex	(1,120)	0.00078719	0.00078719	0.09	<0.7682
Group	(2,120)	0.00253097	0.00126549	0.14	<0.8692
Sex*Group	(2,120)	0.02866831	0.01433415	1.59	<0.2083
Trial	(2,240)	0.06649324	0.03324662	5.81	<0.0034**
Sex*Trial	(2,240)	0.00020283	0.00010142	0.02	<0.9824
Group*Trial	(4,240)	0.01190125	0.00297531	0.52	<0.7210
Sex*Group*Trial	(4,240)	0.00123722	0.00030930	0.05	<0.9945

* $p < .05$
** $p < .01$

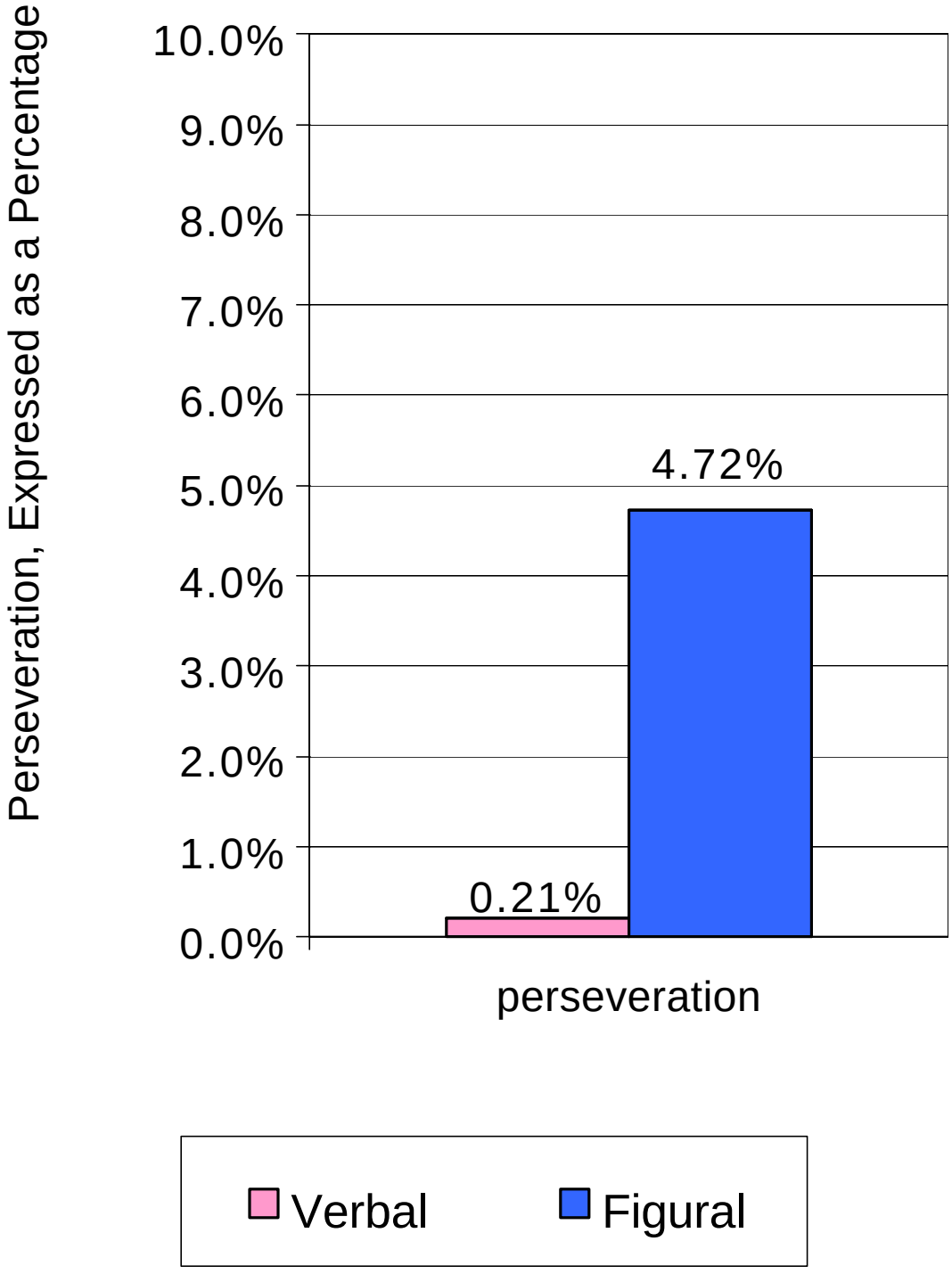


Figure 30. Main effect of task on perseveration (across sex and group).

Time Estimation Task

In order to analyze the participant's ability to accurately judge the passing of time (the participant's *perception* of time), an independent analysis of variance (ANOVA) was performed on the time estimation scores (30 second and 180 second). Data were analyzed for overall sex and group effects/interactions across trials first, and then refined ANOVAs were performed to test group and trial effects within each sex and trial effects within each group. Group means and standard deviations of time estimation scores are presented in Table 27; independent ANOVA results are presented in Table 28.

No main effects of sex or of group were found for time estimation scores. A main effect of trial was found, but this is an artifact of the nature of the task (judging 30 seconds and then 180 seconds of time produced a significant difference between these scores). A sex by group interaction effect was found across trials ($F(2,120) = 3.76$, $p < 0.0260$; Figure 31), and during refined ANOVAs, for each trial: 30 seconds ($F(2,120) = 3.52$, $p < 0.0326$; Figure 32); and 180 seconds ($F(2,120) = 3.30$, $p < 0.0403$; Figure 33).

In addition, a Sex by Group by Trial Interaction Effect was found on the time estimation task ($F(2,120) = 3.30$, $p < 0.0402$; Figure 34). In separate groups of men, as self-reported levels of hostility increased, their perception of time passage changed. The more hostile groups judged time to pass more quickly, compared to the less hostile groups. In separate groups of women, as self-reported levels of hostility increased, their perception of time passage also changed. However, the more hostile groups judged time to pass more slowly, compared to the less hostile groups.

Table 27

Means and Standard Deviations of Time Estimation Task Scores

	Men		**Women**	
	Mean	SD	Mean	SD
Low-Hostile Group				
Trial 1 (30 seconds)	31.3	7.4	25.2	8.7
Trial 2 (180 seconds)	193.0	45.4	169.5	34.7
Mid-Hostile Group				
Trial 1 (30 seconds)	29.9	5.5	26.1	7.4
Trial 2 (180 seconds)	185.6	30.7	165.8	46.1
High-Hostile Group				
Trial 1 (30 seconds)	27.6	5.0	30.0	10.2
Trial 2 (180 seconds)	172.6	33.0	203.0	92.5

Table 28

ANOVA Results for Time Estimation Task

Source	DF	SS	MS	F Value	p
Systolic BP					
Sex	(1,120)	720.14286	720.14286	0.44	<0.5108
Group	(2,120)	1761.05556	880.52778	0.53	<0.5889
Sex*Group	(2, 120)	12455.64286	6227.82143	3.76	<0.0260*
Trial	(1,120)	1479360.571	1479360.571	1391.62	<0.0001**
Sex*Trial	(1,120)	48.016	48.016	0.05	<0.8321
Group*Trial	(2,120)	1333.881	666.940	0.63	<0.5357
Sex*Group*Trial	(2,120)	7016.722	3508.361	3.30	<0.0402*

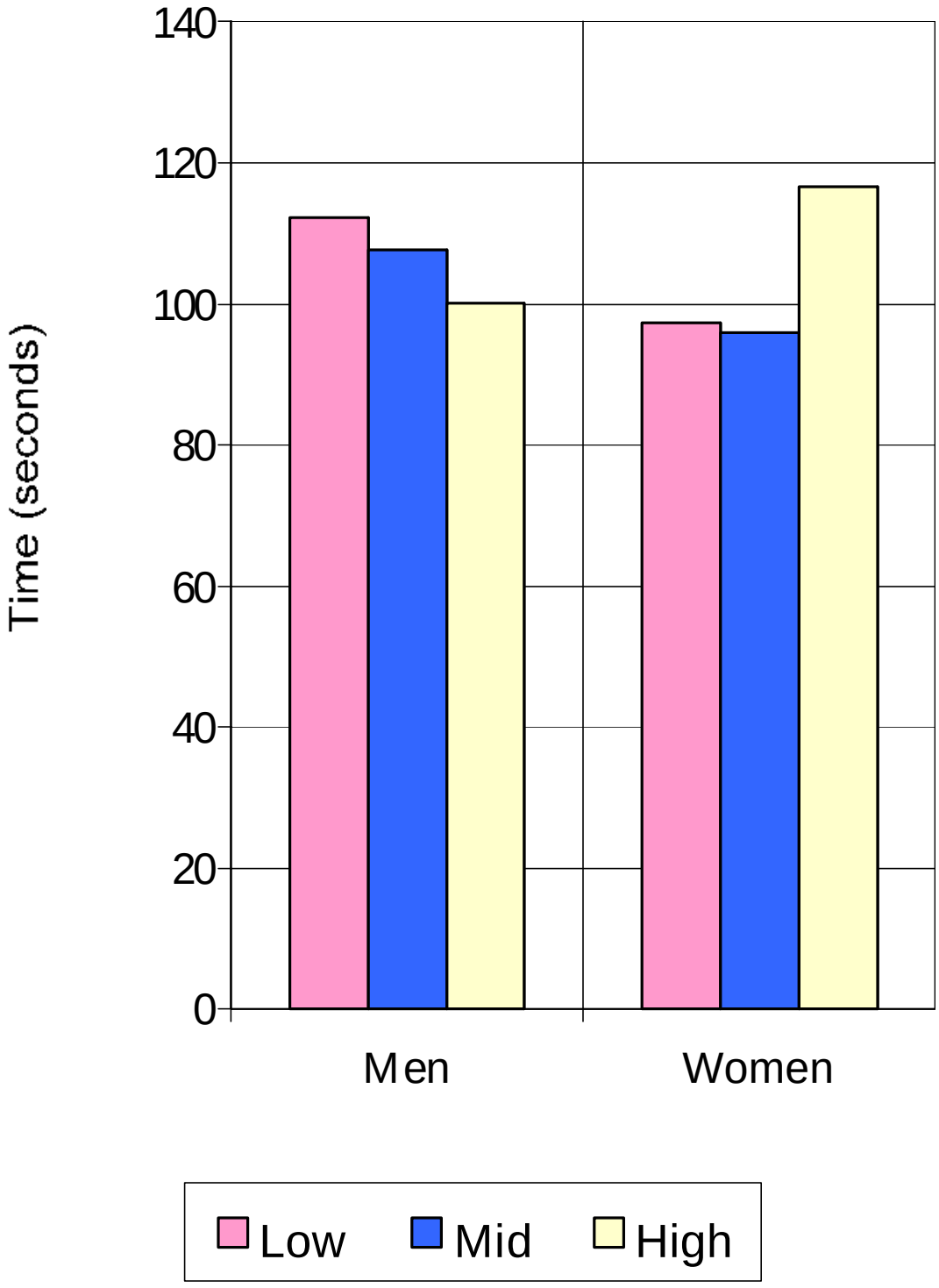


Figure 31. Sex by Group Interaction for Time Estimation Tasks (across trials).

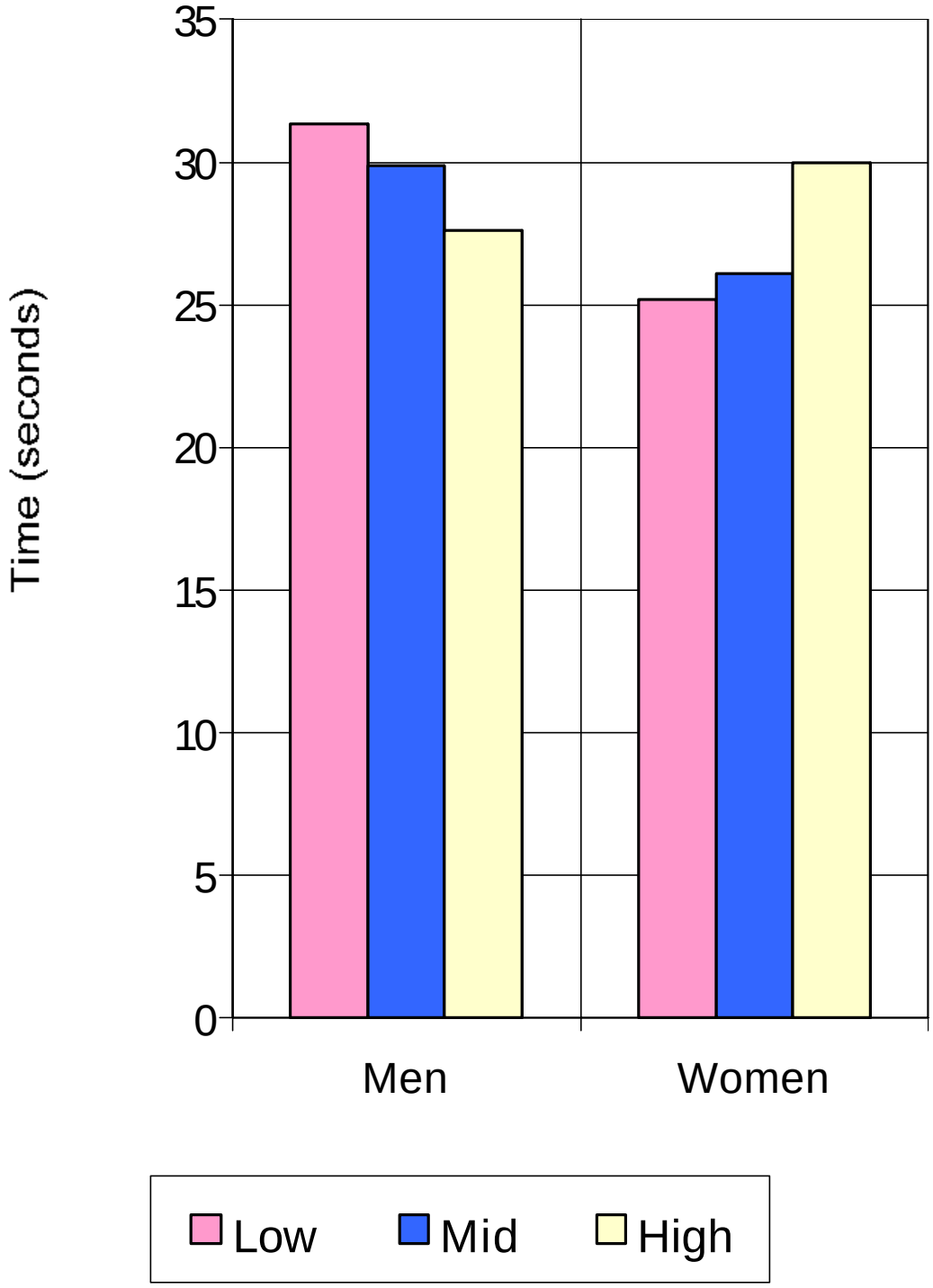


Figure 32. Sex by Group Interaction for Time Estimation Task 1 (30 seconds).

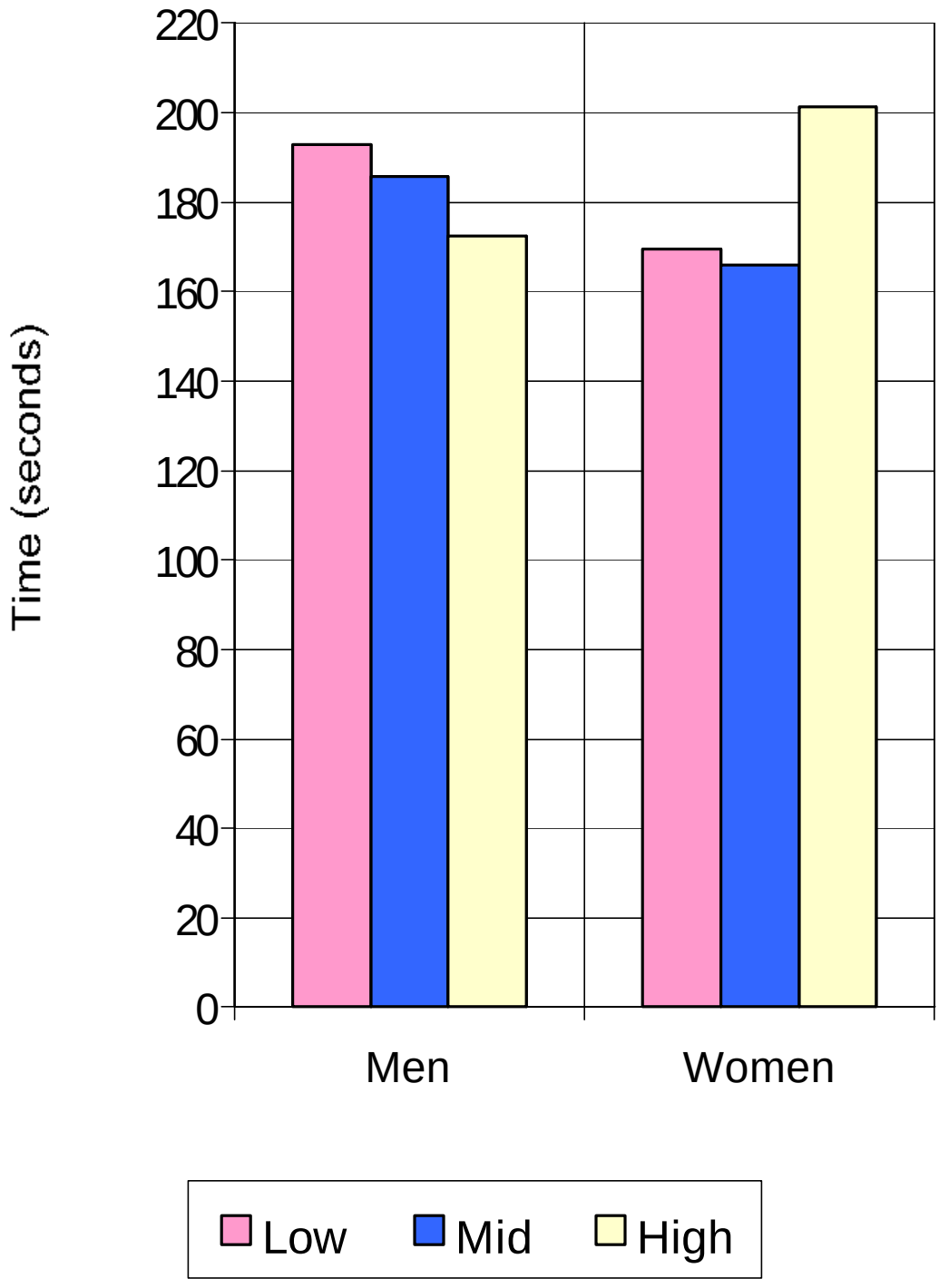


Figure 33. Sex by Group Interaction for Time Estimation Task 2 (180 seconds).

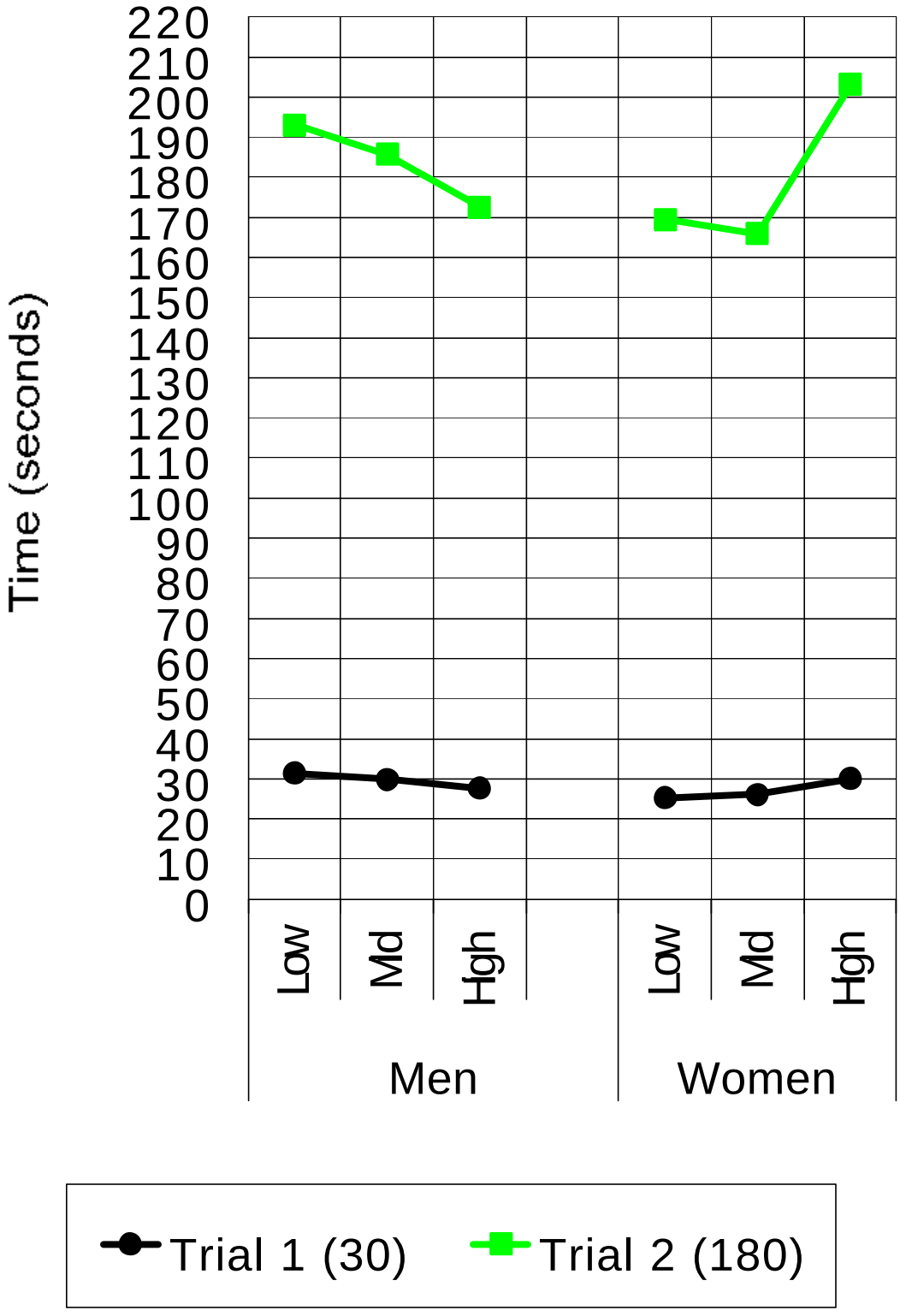


Figure 34. Sex by Group by Trial Interaction for Time Estimation Tasks 1 and 2.

Stress Scores

In order to analyze stress scores (self-reports of stress felt from various components of the experiment), an independent analysis of variance (ANOVA) was performed on the various stress scores (1-4). Data were analyzed for overall sex and group effects/interactions. Refined ANOVAs were performed to test group effects within each sex.

A main effect of sex was found on the third stress scale, when participants were asked, "How stressful was the figural fluency task?" ($F(1,120) = 4.68$, $p < 0.0325$). On this question, men rated perceived stress from this task significantly lower than women ($M = 2.5$ vs. $M = 3.1$, respectively; Figure 35).

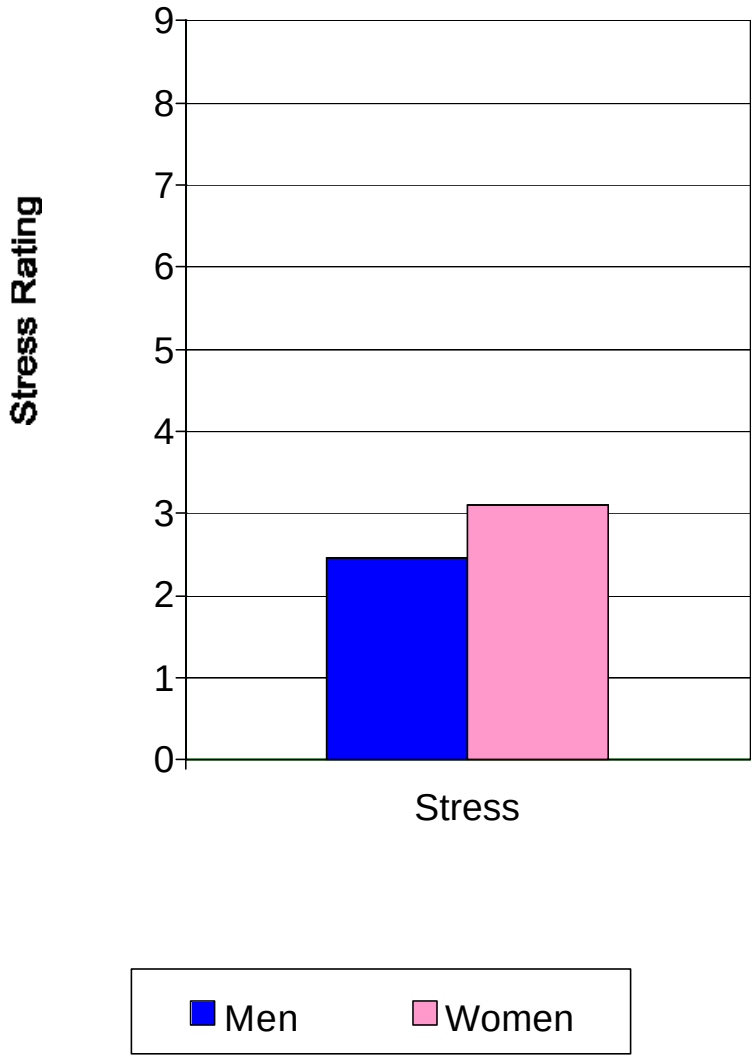


Figure 35. Sex Difference on Stress Rating of Figural Fluency Task.

Menstrual Phase

A General Linear Model, or GLM ANOVA was performed on women's verbal fluency scores, using menstrual phase ("Phase") as a between subjects factor. Data were analyzed for overall group effects and interactions with menstrual phase across trials. A GLM model was used, as cell sizes were unequal. 28 women were identified to be in the Follicular Phase, or the first half of menstruation (identified as equal to or less than day 13); 35 women were identified to be in the Luteal Phase, or second half of menstruation (identified as equal to or greater than day 14). Group means and standard deviations of verbal fluency are presented in Table 29; independent GLM ANOVA results are presented in Table 30. (Spatial Fluency was also analyzed, but menstrual phase had no effect on it).

No main effects of group were found for verbal fluency; however, a main effect of Phase was found for verbal fluency ($F(1,57) = 4.73$, $p < 0.0339$; Figure 36). Also, a Main Effect of Trial was found ($F(2,114) = 12.44$, $p < 0.0001$) on Verbal Fluency, wherein Trial 2 ($M = 14.16$) was significantly different from Trials 1 and 3 ($M = 12.87$ and 12.67, respectively), which were not significantly different from each other.

In addition, a Group by Trial Interaction effect was found on Verbal Fluency ($F(4,114) = 12.44$, $p < 0.0001$). A Phase by Group by Trial Interaction effect was also found on Verbal Fluency ($F(4,114) = 3.01$, $p < 0.0210$; Figure 37), as groups that identified themselves as being low-hostile, mid-hostile, or high-hostile were differentially affected by their current menstrual phase when completing the verbal fluency task.

Table 29

Means and Standard Deviations of Verbal Fluency Scores by Phase

	Follicular		**Luteal**	
	Mean	SD	Mean	SD
Low-Hostile Group	*N=10*		*N=11*	
Trial 1	13.5	2.4	14.4	3.4
Trial 2	13.9	3.7	15.0	2.9
Trial 3	11.1	3.7	14.2	3.3
Mid-Hostile Group	*N=8*		*N=13*	
Trial 1	12.0	3.6	14.6	3.9
Trial 2	13.8	3.3	14.5	2.8
Trial 3	13.8	3.2	13.4	3.8
High-Hostile Group	*N=10*		*N=11*	
Trial 1	10.1	3.6	11.9	2.5
Trial 2	12.6	3.4	14.8	4.0
Trial 3	10.1	3.8	13.3	3.7

Table 30

ANOVA Results for Menstrual Cycle Effects on Verbal Fluency

Source	DF	SS	MS	F Value	p
Phase	(1,57)	146.0447090	146.0447090	5.17	<0.0268*
Group	(2,57)	102.8677249	51.4338624	1.82	<0.1714
Phase*Group	(2,57)	4.1581659	2.0790830	0.07	<0.9292
Trial	(2,114)	82.35978836	41.17989418	12.40	<0.0001**
Phase*Trial	(2,114)	4.13068783	2.06534392	0.62	<0.5386
Group*Trial	(4,114)	41.48148148	10.37037037	3.12	<0.0177*
Phase*Group*Trial	(4,114)	38.92035002	9.73008751	2.93	<0.0238*

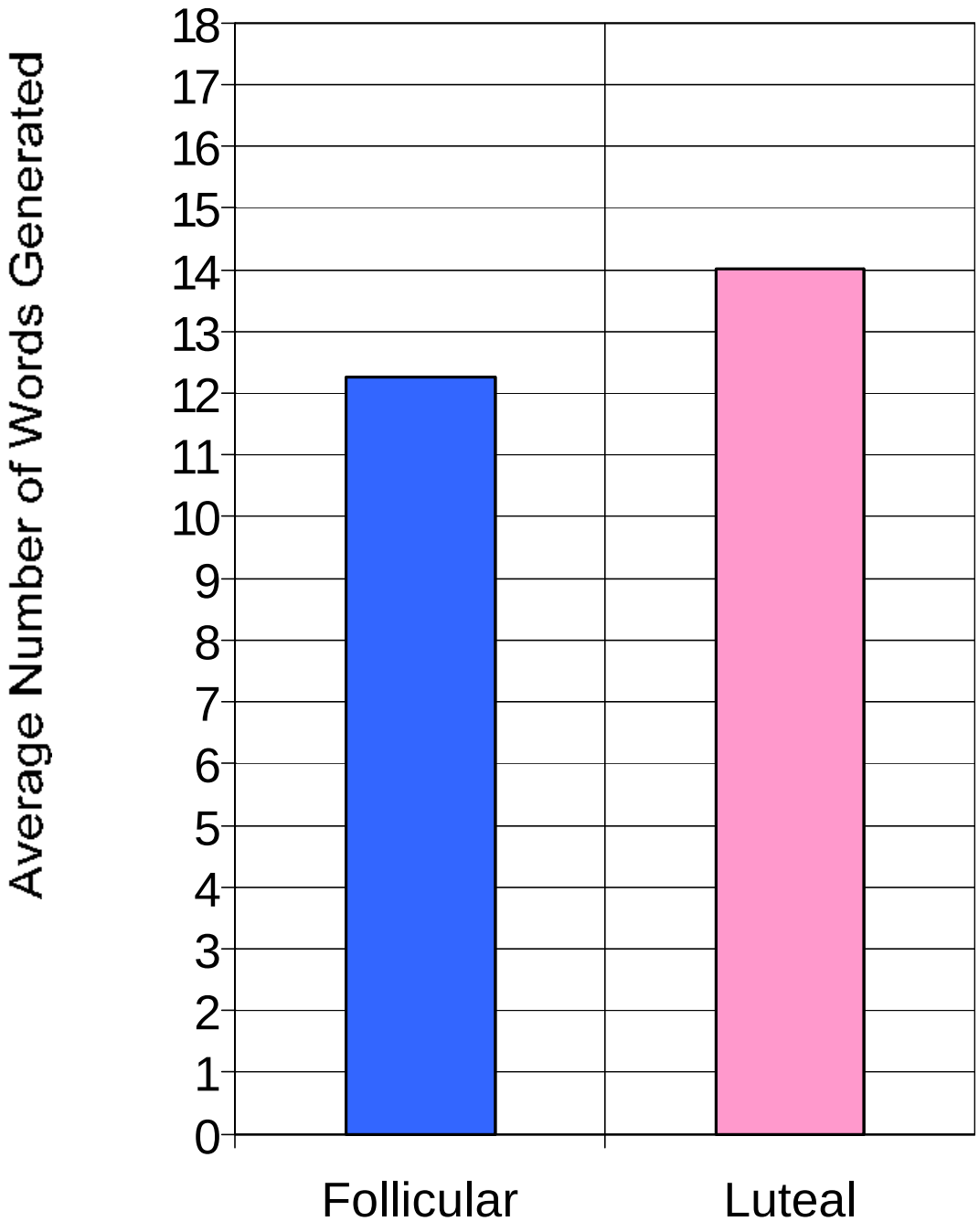


Figure 36. Main Effect of Menstrual Phase on Verbal Fluency.

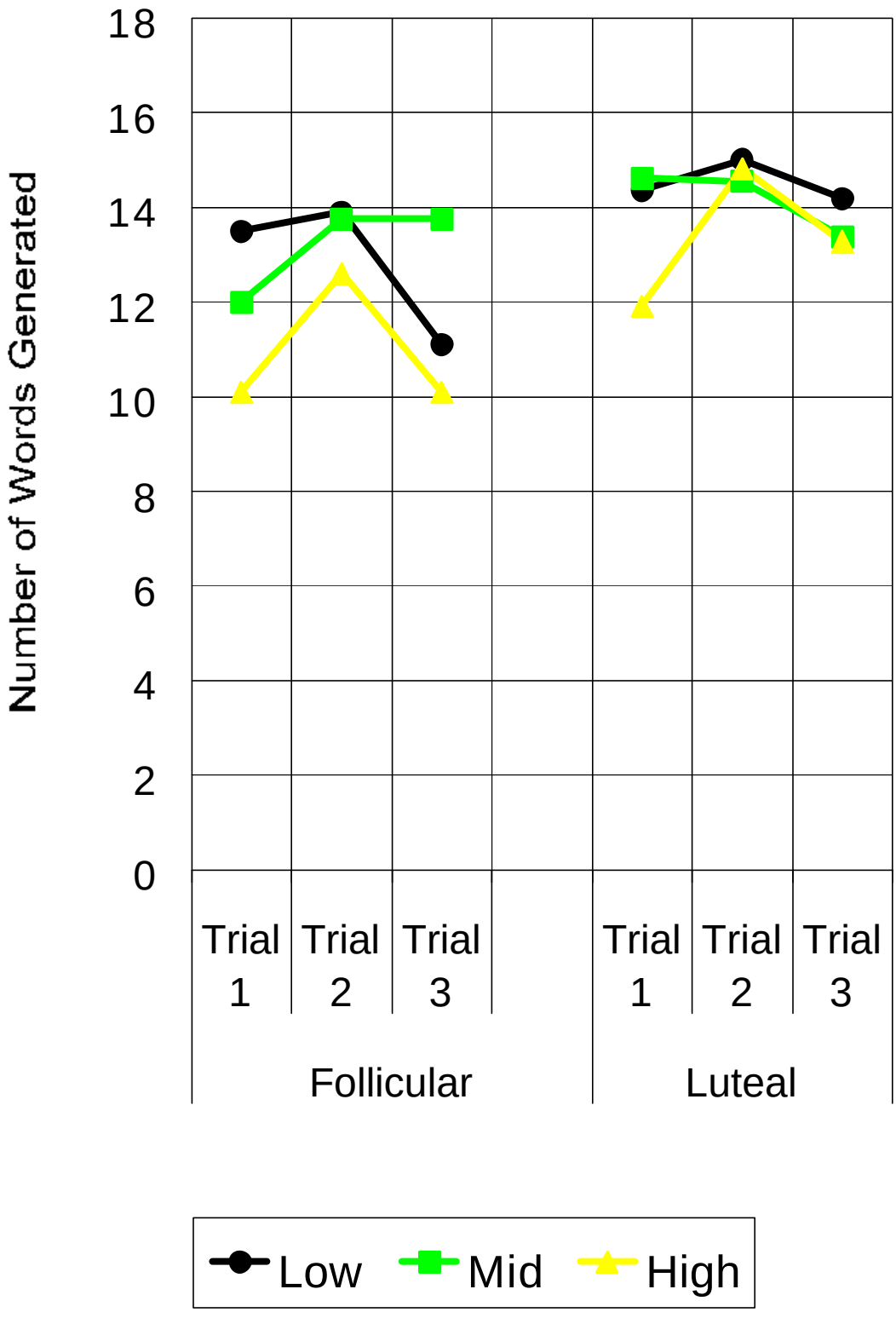


Figure 37. Menstrual Phase by Group by Trial Interaction on Verbal Fluency Performance.

Discussion

The principal findings of the current investigation were that both verbal fluency and figural fluency cognitive stressors raised blood pressure significantly (differently for SBP and DBP), and it interacted with sex and group (low-hostile, mid-hostile, and high-hostile). Some evidence of using cognitive tasks as cardiovascular stressors was therefore gained; however, results did not generally support the proposed neuropsychological model of cardiovascular regulation. Women, in general, were less prone to SBP regulation "disturbances" from cognitive tasks, compared to men; however, women also evidenced higher DBP levels, leading to higher MAP levels, compared to men. It is interesting to note that similar sex differences in DBP have been found previously (Emerson & Harrison, 1990; Higgins, 1999). Systolic blood pressure in men was more responsive to verbal fluency measures, and this affected each group (hostility level) of men differently. Evidence of different cardiovascular regulatory systems in men and women was found, but it was mixed between SBP, DBP, MAP, and HR differences. These systems seem to be more complex than proposed by this model and beyond the design used in this experiment.

Men classifying themselves as more hostile performed better on the first trial of the verbal fluency test compared to men classifying themselves as less hostile, while women classifying themselves as more hostile performed worse on the first trial of the verbal fluency test, compared to women classifying themselves as less hostile. Thus, hostility was found to interact with verbal fluency, and it has also been found to be integral to cardiovascular reactivity and increased health risks (Contrada, 1994; Barefoot, Dahlstrom, & Williams, 1983; Helmers et al., 1993; Miller et al., 1996; Shekelle, Gale, Ostfeld, & Paul, 1983; Smith, 1994; Suarez & Williams, 1989; Williams, 1984; Williams, 1986; Williams, 1987a; Williams, 1987b; Williams, 1996; Williams et al., 1988; Williams, Barefoot, & Shekelle, 1985; Williams et al., 1980; Williams, Suarez, Kuhn, Zimmerman, & Schanberg, 1991). High-hostile men perseverated more on the verbal fluency test, while lower hostile women did the same. In addition, high-hostile men's time perception seems to be more rapid than that of low-hostile men, while for women it was the opposite. Hostility has been viewed as a deficit of self-awareness (Demaree & Harrison, 1997a), which lends support to this finding. However, it was surprising that men and women responded in opposite patterns on this task. This begins to uncover another level of analysis, that of differing levels of perception and awareness, which may lead to differences in feelings of time urgency. Time perception and reproduction has been linked to right, more than left, hemispheric functioning (Kagerer, Wittmann, Szelag, & Steinbüchel, 2002).

Women reported significantly more stress from the figural fluency task than men; nevertheless, this did not lead to significant sex differences on this task. In fact, this may be one of the reasons why sex differences were not found, in that women were more distressed by this task, thereby raising their blood pressure more equal to men's increases and negating the hypothesis that men would evidence more sympathetic response to this task than women.

Women in the luteal phase of menstruation did better on the verbal fluency test than women in the follicular phase of menstruation (who performed at levels similar to men). Hostility and menstrual phase interacted in verbal fluency where some mid-hostile women improved across trials during the follicular phase and while other mid-hostile women performed more poorly across trials during the luteal phase, while low-hostile and high-hostile women did better during the luteal phase on this task, compared to other low-hostile and high-hostile women in their follicular phase. Again, the idea of a dynamic state of neuropsychological functioning, leading to (temporary) changes in laterality, was supported in this finding.

Sex differences in the functional cerebral regulation of basic cardiovascular factors were predicted, mediated by group differences (high-hostile, mid-hostile, and low-hostile men and women). The functional cerebral regulation of cardiovascular reactivity was assessed, specifically, the dynamic nature of cardiovascular regulation within each sex and each group (with hostile groups identified as having increased cardiovascular disease risk). Nevertheless, most of the hypotheses in this experiment were not supported by the results (see Table 31). This may be due, in part, to the healthy cardiovascular

state of many of the participants; to the tasks chosen to stress, or tax the two frontal lobes; to the interaction of hostile vs. denial effects (not assessed); or to other factors. These ideas discussed below.

Heart rate and blood pressure are inversely related through the baroreceptor reflex (Rushmer, 1976). That is to say, as blood pressure increases as a function of the cold pressor (SBP and DBP), heart rate decreases, compensating for some of the extra pressure effects in the vasculature. As many cardiovascular reactions were in the direction opposite of what had been predicted, it is possible that participants were basically "healthy," and thus, did not evidence dysfunctional cardiovascular responding predicted within those with compromised cerebral regulation systems over cardiovascular regulation (found in those with some types of cardiovascular disease, such as hypertension). However, if the model is not useful for healthy individuals, then it is not helpful. The confounds are more complicated such a simple explanation. First, the idea of sex differences in reactivity is discussed.

Sex differences in exaggerated cardiovascular reactivity to stress are a potential pathophysiological mechanism linking behavior with sex differences in cardiovascular disease. As sex differences were found in *types of cardiovascular reactivity* (SBP, DBP, MAP, vs. HR) in this experiment, other studies have also investigated sex differences in *types* of cardiovascular reactivity to laboratory stressors. These different mechanisms may lead to sex differences in the incidence of cardiovascular disease. For example, Allen, Stoney, Owens, and Matthews (1993) studied the effects of types of cognitive challenges and found that men's cardiovascular responses indicated greater total peripheral resistance and systolic and diastolic blood pressure responses (a "vascular" reaction), whereas women exhibited larger increases in heart rate on a subset of tasks (a "cardiac" reaction). In a follow-up study with children and adolescents, sex differences usually seen in adult cardiovascular reactivity patterns (i.e., men showing more reactivity than women) were found in the adolescents more than in the children (Allen & Matthews, 1997). Although men traditionally show more cardiovascular reactivity than women, Emerson and Harrison (1990) found that women with scores placing them in a high-denial category (measured by the Marlowe-Crowne Social Desirability Scale) *and low anger* (measured by the State-Trait Anger Expression Inventory) exhibited the most elevated cardiovascular reactivity in response to a stressor. Thus, it is possible that a group of highly reactive women (who also deny their emotions) has not previously been identified (Emerson & Harrison, 1990). One of the most significant predictors of CHD is proposed by Haynes and Feinleib (1980) to be "suppressed hostility." Thus, combined cardiovascular and different patterns of sympathetic and parasympathetic nervous system reactivity to specific cognitive tasks in hostile persons may suggest some mechanisms underlying cardiovascular disease (Williams & et al., 1982).

Along with differences in responding to stressors, men and women have been found to respond differently to supports. For instance, the *quality* of the network of social support was found to be related in an inverse manner to ambulatory systolic pressure in women, but not in men. Nevertheless, in men, high scores on a measure of self-deception and hostility were independently related to elevated blood pressure and heart rate (Linden, Chambers, Maurice, & Lenz, 1993). These differences underscore an important detail in this type of research (sex differences), that an individual's "perception" of a stressor is just as important as "acceptance" of a stressor. That is to say, hostility is perilous to cardiovascular health because faulty *perceptions* of threat can lead to unhealthy cardiovascular responding/reactivity patterns. The idea that hostility arises from a deficit in right hemisphere systems, which can also lead to deficits in self-awareness (Demaree and Harrison, 1996, 1997a) contributes wholly to the current model of cardiovascular dysregulation and disease, as the idea of a dysfunctional cerebral system (involved in emotional regulation and self-awareness), leading to health threats is the core (ideologically and neurologically) of the current study.

Table 31. *Hypotheses and Results at a glance*

1. A sex by condition interaction was predicted for cardiovascular reactivity to figural fluency tasks, as relative cardiovascular stability was predicted for women as a function of figural fluency tasks, while greater sympathetic cardiovascular changes (increases in SBP and HR) were predicted to figural fluency tasks in men.	Support for this hypothesis was not found, as a sex by condition interaction effect was not found in SBP or HR changes in response to the figural fluency stressor.
2. Another sex by condition interaction was predicted for cardiovascular reactivity to verbal fluency tasks, as relative cardiovascular stability was predicted for women as a function of verbal fluency tasks, while greater parasympathetic cardiovascular effects/changes (relative stability or decreases in SBP and HR) were predicted to verbal fluency tasks in men.	Support for this hypothesis was not found, as a sex by condition interaction was not found for SBP or HR changes in response to the verbal fluency stressor. *However, related analyses revealed a group by task interaction effect where low hostile groups reacted (SBP increases) to verbal more than figural fluency tasks. Also, a task by condition interaction effect on SBP was found, where verbal fluency tasks increased SBP significantly more than figural fluency tasks.*
3. Sex differences were predicted in verbal fluency and figural fluency performance. Women were expected to outperform men on the verbal fluency task, while men were expected to outperform women on the figural fluency task.	Support for this hypothesis was not found, as sex by task by fluency interaction effects were not found. That is to say, no significant differences were found between men's and women's performance on verbal fluency or on figural fluency.
4. Men were predicted to perform better on figural compared to verbal fluency tasks; while women were expected to perform better on verbal compared to figural fluency tasks.	Men did perform significantly better on the figural fluency task compared to the verbal fluency task; however, women performed similarly on each task (no statistically significant differences.
5. Prior to the stressors (fluency tasks), men were predicted to evidence higher levels of systolic blood pressure than women.	Men did evidence higher levels of systolic blood pressure than women prior to stressors. *However, related analyses revealed that women evidenced significantly higher levels of diastolic blood pressure and mean arterial pressure prior to these tasks.*

6. Men were predicted to evidence significantly more perseverative errors on figural fluency than women.	Support for this hypothesis was not found, as men were found to perseverate on the figural fluency task 4.9%, compared to 4.6% in women (not a significant difference). Nor were sex by trial interaction effects found. *However, related analyses revealed a sex by trial interaction effect on the verbal fluency task, wherein men tended to perseverate more as the task continued, opposite the pattern found in women. In addition, a main effect of task was found across sex and groups, as both men and women perseverated more on the figural fluency task.*
7. A sex by group by condition interaction was predicted for cardiovascular reactivity to figural fluency tasks, as greater sympathetic changes (increases in SBP and HR) were predicted for high-hostile men than for high-hostile women as a function of figural fluency tasks.	Support for this hypothesis was not found, as sex by group by condition interaction effects were not found for SBP or HR changes in response to the figural fluency task. *However, related analyses revealed a main effect of condition for DBP and MAP during the figural fluency task (across sex and group), as both increased significantly after the figural fluency task.*
8. A sex by group by condition interaction was predicted for cardiovascular reactivity to verbal fluency tasks, as greater parasympathetic changes (relative stability or decreases in SBP and HR) were predicted for low-hostile men than for low-hostile women as a function of verbal fluency tasks.	Support for this hypothesis was not found, as a sex by group interaction was found across the verbal fluency task, but a 3 way interaction effect with condition was not found. *However, related analyses revealed main effects of condition for SBP, DBP, and MAP in response to the verbal fluency task (across sex and group), as they all increased significantly*.
9. Sex differences in overall cardiovascular stability were predicted for low-hostile women more than low-hostile men.	Support for this hypothesis was not found, as low-hostile men and low-hostile women reacted similarly to fluency stressors, as their cardiovascular measures increased an average of one to two units. That is to say, SBP, DBP, MAP, and HR each increased in similar patterns across condition and trials in low-hostile men and women.
10. High-hostile men will evidence more perseverative errors on figural fluency tasks than low-hostile men.	Support for this hypothesis was not found, as no group effects were found in men on figural perseveration. Low-hostile men perseverated 5.7%, mid-hostile men 4.3%, and high-hostile men 4.1%. There were no significant differences between any of these groups.

It is also important to analyze the neuropsychological tests used in this study, in an attempt to understand the (unexpected) results. Frontal lobe tasks, in particular, can be studied at different levels, as both quantity of response and quality of response can be analyzed separately. Schwartz and Baldo (2001) found that left frontal lobe patients generated unusual word-profiles, diversification thought to be mediated by preserved right frontal lobe functioning; whereas, right frontal lobe patients generated highly typical word-profiles, responses thought to be constrained by preserved left frontal lobe functioning. This dynamic interplay of the cerebral hemispheres, on a task traditionally thought to be a left frontal task, makes it difficult to find a "pure" task, one that affects only one area of the brain, or one part of a neuropsychological system. As most of the hypotheses in this experiment were not supported, it is possible that the tasks used did not test, or tax, specific cerebral areas. It is likely that these tasks (verbal and figural fluency) affected cerebral systems in both hemispheres. It is difficult to find neuropsychological tasks that only affect one or few cerebral areas. For example, a task purported to affect only right frontal areas, for instance, activates left frontal areas also if a patient uses his left hand to complete the task (to draw). Similarly, if the same patient is asked to answer questions or otherwise speak throughout the examination, then left temporal and frontal areas are activated, which can affect right frontal systems. Although, verbal fluency, both letter and category, is produced more exclusively by left frontal areas (Baldo & Shimamura, 1998); recent research suggests that design fluency is managed by both left and right frontal areas (Baldo, Shimamura, Delis, Kramer, & Kaplan, 2001). Elfgren and Risberg (1998) also suggest that design fluency involves processing by both left and right frontal areas. They found that during the verbal fluency task, cerebral blood flow findings indicated mainly left frontal activation; however, during the design fluency task, both frontal lobes were active. Furthermore, changes in cognitive strategy produced different patterns of brain activation. They theorized that left frontal cortical areas are involved in the "generation of internally driven responses." Dynamic functioning of areas and systems was supported. Furthermore, they argue that brain areas activated during tasks also *reflect cognitive strategy*. Thus, areas are involved in basic tasks and in higher-order processes.

Similarly, Vilkki, Levänen, and Servo (2002) studied the dynamic relationship between anterior and posterior cerebral systems, using verbal and figural fluency tasks, administered to patients with anterior or posterior lesions. They found that when patients completed these tasks alone (as single tasks) there were not differences between patients with anterior and posterior lesions. However, when the tasks were administered simultaneously, patients with left anterior lesions evidenced more impairment than other patients in letter fluency, but not figural fluency. As this frontal, or anterior, systems is responsible for dual task execution and also for specific tasks, it is difficult to separate the dysfunctional area from the taxed system.

The proposed model, tested in this experiment, might also need to be revised. It is difficult to deduce whether the task or the model is responsible for the mixed, generally lacking, results. It is likely due to both, as the experiment and the model can both be improved. In the model, the importance of perception, both of "self" and of the "outside world" should be emphasized more. Hostility, for example, can be seen as a test of self-awareness and of an individual's perceived world. Denial, or social desirability, can be viewed in like manner. Many factors historically categorized as *personality constructs* are more useful when seen as *modes of perception*.

Many sex differences in brain anatomy and functionality are found in the temporo-parietal regions of the brain, which are related to linguistic and figural functions (Witelson, 1991). Moreover, these areas have been shown to be involved in emotion processing and perception (as well as cardiovascular regulation, both sympathetic and parasympathetic responding). Consequently, one proposal of this model is the idea that in order to better understand cardiovascular disease one must consider possible, previously unexplored reasons for sex differences in cardiovascular disease risk. One way to understand possible mechanisms leading to this epidemiological disparity is through a consideration of sex differences in perception and emotion processing, leading to sex differences in cardiovascular reactivity and cardiovascular disease.

Sex differences have been found in verbal processing, spatial skills, emotional perception, motor performance, and even a simple line bisection task. When right-handed men and women were asked to bisect a line with each hand, a sex by hand interaction was found wherein women showed a left bias (spatial) with each hand, while men showed the bias only at the left hand (Hausmann, Ergun, Yazgan, Güntürkün, 2002). This difference found in a basic perceptual task, with spatial undertones, underscores an important idea: that sex differences in symmetry can lead to differences in perception. Differences in perception can lead to differences in reactivity and cardiovascular disease states. It is important to note that perception and reactivity have already been linked through a common construct, hostility. This part of the system cannot be isolated without considering how the system functions with and without it. That is to say, hostility has only recently been more appreciated as a faulty mode of perception, analysis, and awareness, a *neuropsychological construct*, leading to differences in behavioral reactions. As hostile persons analyze situations and react, how sensory portions of the brain contribute (in a flawed manner) to this process must be considered.

There is a necessity for researchers to consider sex differences when interpreting cardiovascular research. Often, results of cardiovascular studies are generalized to both sexes. However, as sex differences in the brain and neuropsychological functioning contribute to sex differences in cardiovascular disease, a new concept should be considered, *neuropsychological risk for cardiovascular disease*. Neuropsychological sex differences found in the literature can often be conceptualized within a functional cerebral laterality and/or functionality framework. Nevertheless, findings often ascribe dissimilar levels of importance to sex as a factor. As sex differences in cerebral functioning lead to differences in cardiovascular regulation and corresponding disease risk, it may be important to reconsider some health variables, such as differences in recovery profiles for various forms of cardiovascular disease. Sex differences in functional cerebral cardiovascular regulation can be better appreciated when considering both current disease rate differences and how these differences affect health across the lifespan (epidemiological differences). Furthermore, certain types of cardiovascular disease, like hypertension, coronary heart disease, and stroke might be studied more closely to investigate how this model applies to specific manifestations, or forms, of cardiovascular disease. Recently, research data has led some investigators to theorize that individual differences in cerebral asymmetry may contribute to cardiac failure (Lane & Jennings, 1995). This is a continuation of a previous relationship found between emotional stress, cardiac failure, and sudden death (Hugdahl, 1995; Lane & Schwartz, 1987). Thus, as sex differences have been found in cerebral asymmetry, functionality, and even structure, this may be one of the primary mechanisms by which sex differences in cardiovascular regulation and disease originate. Hemispheric specialization seems to have led to asymmetric sex differences and elaborations of the dorsal and ventral pathways between frontal and limbic areas. Tucker, Luu, and Pribram propose that through an understanding of the asymmetries of this corticolimbic (frontolimbic) architecture, it may be possible to interpret the increasing evidence that the "left and right frontal lobes contribute differently to normal and pathological forms of self- regulation" (Tucker et al., 1995).

In addition, the social impact of this model should be considered, as counseling services may benefit through consideration of possible implications of this model. That is to say, if sex differences exist in the neuropsychological functioning of language (perception of speech; Higgins, 1999) and speech fluency (current study) while under stress (high cardiovascular reactivity), then therapy would likely have different effects on each sex, as basic communication skills would differ for each sex. Furthermore, as women have been found to vocalize more often than men (Joseph, 1996), it is possible that stress affects men and women's available verbal capacities differently (Higgins, 1999). A related concept was discussed by Crews (1995), who presented a neuropsychological model of cognitive therapy. While this mode of therapy is often effective, it may sometimes prove ineffective in treating depression due to neuropsychological factors (Crews & Harrison, 1995). Thus, as sex differences in dynamic functional cerebral laterality have been found (i.e. changes in speech processing among men and women and the impact of speech processing on cardiovascular measures), this model may thus be relevant in emotional processing or therapy research (e.g., cognitive therapies) where verbal or other processing may

differentially impact cardiovascular regulation in men and women (Higgins, 1999) or emotional processing systems (Crews & Harrison, 1995).

An integration of various lines of research suggests that men's right cerebrum is differentially implicated in cardiovascular regulation, the processing of negative emotions, and consequent dysfunctional patterns of cardiovascular reactivity. Thus, being more lateralized, or asymmetrically organized, may contribute to increased cardiovascular disease in men, as proximal neural systems are taxed by multiple demands. That is to say, the right hemisphere processes emotions and regulates cardiovascular sympathetic responses; therefore, neural system dysfunction in cardiovascular regulation becomes more likely as emotional processing demands increase. Future research should continue to evaluate the role that hostility plays in the incidence of cardiovascular disease as a function of the neuropsychology of sex differences. The emerging perspective of dynamic functional cerebral regulation of cardiovascular indices can be considered as a viable explanation for certain sex differences in cardiovascular disease incidence. Given the mixed findings of this preliminary study, versus the potential benefits of continued research in this area, the proposed neuropsychological model of sex differences in cardiovascular regulation and disease warrants further scientific study. Appreciating sex differences in the neuropsychology of cardiovascular regulation leads to a better conceptualization of differing levels cardiovascular disease risk. Treatment implications include viewing some forms of cardiovascular disease as an extension or manifestation of cerebral dysfunction, then treating dysfunctional neuropsychological patterns while pharmacologically treating cardiovascular disease. As sex differences in cardiovascular reactivity are viewed through a functional systems model, potential treatment protocols and strategies may tend to be more innovative than classic, and more investigative than established. Nevertheless, this is how scientific progress is made, as complex ideas are tested, then accepted, as data supports new models.

References

A.H.A. (1998). Heart and Stroke Statistical Update . Dallas, Texas: American Heart Association.

A.H.A. (1999). 2000 Heart and Stroke Statistical Update . Dallas, Texas: American Heart Association.

Alden, J. D., Harrison, D. W., Snyder, K. A., & Everhart, D. E. (1997). Age differences in intention to left and right hemispace using a dichotic listening paradigm. Neuropsychiatry, Neuropsychology, & Behavioral Neurology, 10, 239-242.

Allen, M. T., & Matthews, K. A. (1997). Hemodynamic responses to laboratory stressors in children and adolescents: the influences of age, race, and gender [published erratum appears in Psychophysiology 1997 Nov;34(6):730]. Psychophysiology, 34(3), 329-39.

Allen, M. T., Stoney, C. M., Owens, J. F., & Matthews, K. A. (1993). Hemodynamic adjustments to laboratory stress: The influence of gender and personality. Psychosomatic Medicine, 55(6), 505-17.

Arato, M., Frecska, E., Tekes, K., & MacCrimmon, D. J. (1991). Serotonergic interhemispheric asymmetry: Gender difference in the orbital cortex. Acta Psychiatrica Scandinavica, 84(1), 110-111.

Asenbaum, S., Lang, W., Egkher, A., Lindinger, G., & et al. (1992). Frontal DC potentials in auditory selective attention. Electroencephalography & Clinical Neurophysiology, 82(6), 469-476.

Ayanian, J. Z., & Epstein, A. M. (1991). Differences in the use of procedures between women and men hospitalized for coronary heart disease. New England Journal of Medicine, 325(4), 221-5.

Baldo, J. V., Shimamura, A. P. (1998). Letter and category fluency in patients with frontal lobe lesions. Neuropsychology, 12(2), 259-267.

Baldo, J. V., Shimamura, A. P., Delis, D. C., Kramer, J., & Kaplan, E. (2001). Verbal and design fluency in patients with frontal lobe lesions. Journal of the International Neuropsychological Society, 7, 586-596.

Barefoot, J. C., Dahlstrom, W. G., & Williams, R. B., Jr. (1983). Hostility, CHD incidence, and total mortality: a 25-year follow-up study of 255 physicians. Psychosomatic Medicine, 45(1), 59-63.

Barnett, E. (2000). Evidence of Increasing Coronary Heart Disease Mortality Among Black Men of Lower Social Class. (Abstract). JAMA: The Journal of the American Medical Association, 283(13), 1669.

Basso, A., Capitani, E., & Moraschini, S. (1982). Sex differences in recovery from aphasia. Cortex, 18, 469-475.

Bear, D. M. (1983). Hemispheric specialization and the neurology of emotion. Archives of Neurology, 40(4), 195-202.

Beaumont, J. G. (Ed.). (1982). Divided Visual Field Studies of Cerebral Organization. London: Academic Press.

Beaumont, J. G., Mayes, A. R., & Rugg, M. D. (1978). Asymmetry in EEG alpha coherence and power: Effects of task and sex. Electroencephalography and Clinical Neurophysiology, 45, 393-401.

Benbow, C. P., & Stanley, J. C. (1980). Sex differences in mathematical ability: Fact or artifact? Science, 210, 1262-1264.

Benton, A., & Hamsher, K. S. (1976). Multilingual Aphasia Examination. Iowa City: University of Iowa.

Benton, A. L. (1968). Differential Behavioral Effects in Frontal Lobe Disease. Neuropsychologia, 6(1), 53-60.

Berlin, D. F., & Languis, M. L. (1980). Age and sex differences in measures of brain lateralization. Perceptual and Motor Skills, 50(3 Pt 1), 959-67.

Borokowski, J. G., Benton, A. L., & Spren, O. (1981). Word fluency measure. In F. L. Darley (Ed.), Evaluation of Appraisal Techniques in Speech and Language Pathology . Reading, MA: Addison-Wesley Publishing Company.

Bradshaw, J. L., & Nettleton, N. C. (1981). The nature of hemispheric specialization in man. Behavioral and Brain Sciences, 4, 51-91.

Bradshaw, J. L., & Nettleton, N. C. (1983a). Hemispheric specialization: Return to a house divided. Behavioral and Brain Sciences, 6, 528-533.
Bradshaw, J. L., & Nettleton, N. C. (1983b). Human Cerebral Asymmetry. Englewood Cliffs, N. J.: Prentice-Hall.
Braun, C. M., Baribeau, J. M., Ethier, M., Guerette, R., & Proulx, R. (1988). Emotional facial expressive and discriminative performance and lateralization in normal young adults. Cortex, 24(1), 77-90.
Broverman, D. M., Klaiber, E. L., & Kobayashi, Y. (1968). Roles of Activation and Inhibition in Sex Differences in Cognitive Abilities. Psychological Review, 75(1), 23-50.
Bryden, M. P. (1982). Laterality: Functional Asymmetry in the Intact Brain. New York: Academic Press.
Butler, R. W., Rorsman, I., Hill, J. M., & Tuma, R. (1993). The effects of frontal brain impairment on fluency: Simple and complex paradigms. Neuropsychology, 7(4), 519-529.
Butler, S. (1984). Sex differences in human cerebral function. Progress in Brain Research, 61, 443-55.
Butter, C. M., Snyder, D. R., & McDonald, J. A. (1970). Effects of orbital frontal lesions on aversive and aggressive behaviors in rhesus monkeys. Journal of Comparative and Physiological Psychology, 72(1), 132-44.
Caltagirone, C., Zoccolotti, P., Originale, G., Daniele, A., & Mammucari, A. (1989). Autonomic reactivity and facial expression of emotion in brain-damaged patients. In G. Gainotti & C. Caltagirone (Eds.), Emotions and the Dual Brain (pp. 204-221). Berlin: Springer-Verlag.
Campbell, R. (1978). Asymmetries in interpreting and expressing a posed facial expression. Cortex, 14(3), 327-42.
Cannon, W. B. (1929). Bodily Changes in Pain, Hunger, Fear and Rage. New York: Appleton.
Casey, M. B., Pezaris, E., & Nuttall, R. L. (1992). Spatial ability as a predictor of math achievement: the importance of sex and handedness patterns. Neuropsychologia, 30(1), 35-45.
Castelli, W. P. (1988). Cardiovascular disease in women. American Journal of Obstetrics and Gynaecology, 158(6 Pt 2), 1553-60, 1566-7.
Chrichton-Browne, J. (1878). On the weight of the brain and its component parts in the insane. Brain, 1, 504-518.
Clarke, J. M., & Zaidel, E. (1994). Anatomical-behavioral relationships: corpus callosum morphometry and hemispheric specialization. Behavioural Brain Research, 64(1-2), 185-202.
Collins, D. W., & Kimura, D. (1997). A large sex difference on a two-dimensional mental rotation task. Behavioral Neuroscience, 111(4), 845-849.
Contrada, R. J. (1994). Personality and anger in cardiovascular disease: Toward a psychological model. In A. W. Siegman & T. W. Smith (Eds.), Anger, Hostility, and the Heart . Hillsdale, NJ: Lawrence Erlbaum Associates.
Contrada, R. J., & Jussim, L. (1992). What does the Cook-Medley hostility scale measure? In search of and adequate measurement model. Journal of Applied Social Psychology, 22, 615-627.
Cook, W. W., & Medley, D. M. (1954). Proposed hostility and pharisaic-virtue scales for the MMPI. Journal of Applied Psychology, 238, 414-418.
Coren, S. P., Porac, C., & Duncan, P. (1979). A behaviorally validated self-report inventory to assess four types of lateral preference. Journal of Clinical Neuropsychology, 1(1), 55-64.
Crawford, H. J., Clarke, S. W., & Kitner-Triolo, M. (1996). Self-generated happy and sad emotions in low and highly hypnotizable persons during waking and hypnosis: Laterality and regional EEG activity differences. International Journal of Psychophysiology, 24(3), 239-266.
Crews, J., W. D., & Harrison, D. W. (1994a). Functional asymmetry in the motor performances of women: Neuropsychological effects of depression. Perceptual and Motor Skills, 78, 1315-1322.
Crews, J., W. D., Harrison, D. W., & Rhodes, R. D. (1999). Neuropsychological test performances of young depressed outpatient women: An examination of executive functions. Archives of Clinical Neuropsychology, 14, 517-529.
Crews, J., W. D., Harrison, D. W., Rhodes, R. D., & Demaree, H. A. (1995). Hand fatigue asymmetry in the motor performances of women with depressed mood. Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 8, 277-281.

Crews, W. D., & Harrison, D. W. (1994b). Cerebral asymmetry in facial affect perception by women: Neuropsychological effects of depressed mood. Perceptual and Motor Skills, 79, 1667-1679.
Crews, W. D., Jr., & Harrison, D. W. (1995). The neuropsychology of depression and its implications for cognitive therapy. Neuropsychology Review, 5(2), 81-123.
Crowne, D. P., & Marlowe, D. (1960). A new scale of social desirability independent of psychopathology. Journal of Consulting Psychology, 24, 349-354.
Crowne, D. P., & Marlowe, D. (1964). The Approval Motive: Studies in Evaluative Dependence. New York: John Wiley & Sons, Inc.
Davidson, H., Cave, K. R., & Sellner, D. (2000). Differences in visual attention and task interference between males and females reflect differences in brain laterality. Neuropsychologia, 38(4), 508-19.
Davidson, R. J. (1993). The neuropsychology of emotion and affective style. In M. H. J. M. Lewis (Ed.), Handbook of emotions (pp. 143-154). New York, NY, USA: Guilford Press.
Davidson, R. J., Ekman, P., Saron, C. D., Senulis, J. A., & Friesen, W. V. (1990). Approach-withdrawal and cerebral asymmetry: Emotional expression and brain physiology I. Journal of Personality and Social Psychology, 58(2), 330-341.
Davidson, R. J., & Fox, N. A. (1982). Asymmetrical brain activity discriminates between positive and negative affective stimuli in human infants. Science, 218(4578), 1235-7.
Davidson, R. J., & Schwartz, G. E. (1976). Patterns of cerebral lateralization during cardiac biofeedback versus the self-regulation of emotion: Sex differences. Psychophysiology, 13(1), 62-68.
Davidson, R. J., Schwartz, G. E., Pugash, E., & Bromfield, E. (1976). Sex differences in patterns of EEG asymmetry. Biological Psychology, 4(2), 119-38.
Dawson, J. L., Cheung, Y. M., & Lau, R. T. (1975). Developmental effects of neonatal sex hormones on spatial and activity skills in the white rat. Biological Psychology, 3(3), 213-229.
De Vries, G. J., & Boyle, P. A. (1998). Double duty for sex differences in the brain. Behavioural Brain Research, 92(2), 205-13.
Delgado, J. M. R. (1981). Neuronal constellations in aggressive behavior. In L. Valzelli & L. Morgese (Eds.), Aggression and Violence: A Psychobiological and Clinical Approach (pp. 82-98). Milan, Italy: Edizioni Saint Vincent.
Demakis, G. J., & Harrison, D. W. (1997). Relationships between verbal and nonverbal fluency measures: Implications for assessment of executive functioning. Psychological Reports, 81(2), 443-448.
Demaree, H. A., & Harrison, D. W. (1996). Case study: Topographical brain mapping in hostility following mild closed head injury. International Journal of Neuroscience, 87(1-2), 97-101.
Demaree, H. A., & Harrison, D. W. (1997a). A neuropsychological model relating self awareness to hostility. Neuropsychology Review, 7(4), 171-185.
Demaree, H. A., & Harrison, D. W. (1997b). Physiological and neuropsychological correlates of hostility. Neuropsychologia, 35(10), 1405-1411.
Demaree, H. A., Harrison, D. W., & Rhodes, R. D. (in press). Quantitative electroencephalographic analyses of cardiovascular regulation: Low- and high-hostiles to stress. Psychobiology.
Derryberry, D., & Tucker, D. M. (1992). Neural mechanisms of emotion. Journal of Consulting & Clinical Psychology, 60(3), 329-338.
Dimond, S. J., Farrington, L., & Johnson, P. (1976). Differing emotional response from right and left hemispheres. Nature, 261(5562), 690-2.
Dorion, A. A., Chantome, M., Hasboun, D., Zouaoui, A., Marsault, C., Capron, C., & Duyme, M. (2000). Hemispheric asymmetry and corpus callosum morphometry: a magnetic resonance imaging study. Neuroscience Research, 36(1), 9-13.
Duffy, E. (1962). Activation and Behavior. New York: Wiley.
Easterbrooks, J. A. (1959). The effect of emotion on cue utilization and the organization of behavior. Psychological Review, 66, 183-201.
Ehrhardt, A. A., & Meyer-Bahlburg, H. F. L. (1981). Effects of prenatal sex hormones on gender-related behavior. Science, 211, 1312-1318.

Ekman, P., Levenson, R. W., & Friesen, W. V. (1983). Autonomic nervous system activity distinguishes among emotions. Science, 221(4616), 1208-1210.
Elfgren, C. I., & Risberg, J. (1998). Lateralized frontal blood flow increases during fluency tasks: Influence of cognitive strategy. Neuropsychologia, 36(6), 505-512.
Elliott, D., Weeks, D. J., Lindley, S., & Jones, R. (1986). Sex differences in dual-task interference between speaking and manual force-production task. Perceptual and Motor Skills, 62, 3-8.
Emerson, C. S., & Harrison, D. W. (1990). Anger and denial as predictors of cardiovascular reactivity in women. Journal of Psychopathology and Behavioral Assessment, 12(4), 271-283.
Erdi, P. (1993). Neurodynamic system theory: Scope and limits. Theoretical Medicine, 14(2), 137-52.
Everhart, D. E. (1997). Cerebral Regulation of Cardiovascular Functioning and Fluency Among Anxious and Nonanxious Men. Unpublished Dissertation. Virginia Tech, Blacksburg, VA.
Everhart, D. E., & Harrison, D. W. (1995, Fall). Hostility following right CVA: Support for right orbital frontal deactivation and right temporal activation. Journal of Neurotherapy, 55-59.
Fairweather, H. (1982). Sex differences. In J. G. Beaumont (Ed.), Divided Visual Field Studies of Cerebral Organization . London: Academic Press.
Farace, E., & Turkheimer, E. (1996). Gender differences in brain morphometry and function. In E. D. Bigler (Ed.), Neuroimaging II: Clinical Applications (pp. 127-151). New York: Plenum Press.
Filimonov, I. N. (1940). On the localization of function in the cerebral cortex, according to the data of the neurological clinic and of anatomy. Nevropatologieia, Psiekhiatrieia Ie Psiekhogiegieena, 9(6), 23-24.
Fishbein, D. H. (1992). The psychobiology of female aggression. Criminal Justice and Behavior, 19, 99-126.
Flor-Henry, P. (1983). Mood, the right hemisphere and the implications of spatial information perceiving systems. Research Communications in Psychology, Psychiatry and Behavior, 8(2), 143-170.
Flor-Henry, P., & Koles, Z. J. (1982). EEG characteristics of normal subjects: A comparison of men and women and of dextrals and sinistrals. Research Communications in Psychology, Psychiatry and Behavior, 7, 21-38.
Fox, N. A. (1994). Dynamic cerebral processes underlying emotion regulation. Monographs of the Society for Research in Child Development, 59, 152-166.
Fox, N. A., & Davidson, R. J. (1988). Patterns of brain electrical activity during facial signs of emotion in 10-month-old infants. Developmental Psychology, 24, 230-236.
Frost, J. A., Binder, J. R., Springer, J. A., Hammeke, T. A., Bellgowan, P. S., Rao, S. M., & Cox, R. W. (1999). Language Processing is Strongly Left Lateralized in Both Sexes. Evidence from Functional MRI. Brain, 122(Pt 2), 199-208.
Gaillard, W. D., Hertz-Pannier, L., Mott, S. H., Barnett, A. S., LeBihan, D., & Theodore, W. H. (2000). Functional anatomy of cognitive development: fMRI of verbal fluency in children and adults. Neurology, 54(1), 180-185.
Gainotti, G. (1972). Emotional behavior and hemispheric side of the lesion. Cortex, 8(1), 41-55.
Gainotti, G. (1983). Emotions and hemispheric lateralization. [Review of the literature]. Encephale, 9(4), 345-64.
Gainotti, G. (1984). Some methodological problems in the study of the relationships between emotions and cerebral dominance. Journal of Clinical Neuropsychology, 6(1), 111-21.
Gentile, D. A. (1993). Just what are sex and gender, anyway? A call for a new terminological standard. Psychological Science, 4(2), 120-122.
Geschwind, N., & Galaburda, A. M. (1987). Cerebral lateralization: Biological mechanisms, associations, and pathology. Cambridge, MA: MIT Press.
Glass, A., Butler, S. R., & Carter, J. C. (1984). Hemispheric asymmetry of EEG alpha activation: Effects of gender and familial handedness. Biological Psychology, 19(3-4), 169-187.
Gur, R. C., Turetsky, B. I., Matsui, M., Yan, M., Bilker, W., Hughett, P., & Gur, R. E. (1999). Sex differences in brain gray and white matter in healthy young adults: Correlations with cognitive performance. Journal of Neuroscience, 19(10), 4065-72.

Guyton, A. C., Coleman, T. G., & Granger, H. J. (1972). Circulation: overall regulation. Annual Review of Physiology, 34, 13-46.
Hachinski, V. C., Oppenheimer, S. M., Wilson, J. X., Guiraudon, C., & Cechetto, D. F. (1992). Asymmetry of sympathetic consequences of experimental stroke. Archives of Neurology, 49, 697-702.
Hanske-Petitpierre, V., & Chen, A. C. (1985). Sex differences in brain organization: implications for human communication. International Journal of Neuroscience, 28(3-4), 197-214.
Harbin, T. J. (1989). The relationship between the type A behavior pattern and physiological responsivity: A quantitative review. Psychophysiology, 26(1), 110-9.
Harrington, A. (1995). Unfinished business: Models of laterality in the nineteenth century. In R. J. Davidson & K. Hugdahl (Eds.), Brain Asymmetry (pp. 3-27). Cambridge, Massachusetts: Massachusetts Institute of Technology Press.
Harris, L. J. (1978). Sex differences in spatial ability. In M. Kinsbourne (Ed.), Asymmetrical Function of the Brain . Cambridge: Cambridge University Press.
Harrison, D. W., & Edwards, M. C. (1988). Blood pressure reactivity and bias vary with age in a comparison of traditional and automated methods of measurement. Medical Instrumentation, 22(5), 230-3.
Harrison, D. W., & Gorelczenko, P. M. (1990). Functional asymmetry for facial affect perception in high and low hostile men and women. International Journal of Neuroscience, 55, 89-97.
Harrison, D. W., Gorelczenko, P. M., & Cook, J. C. (1990). Sex differences in the functional asymmetry for facial affect perception. International Journal of Neuroscience, 52, 11-16.
Harrison, D. W., Gorelczenko, P. M., & Kelly, P. L. (1988). Human factors and design evaluation of digital blood pressure/pulse meters. Medical Instrumentation, 22(5), 226-9.
Harrison, D. W., & Pavlik, W. B. (1983). The effects of age, exposure, pre-exposure, and noise conditions on variable interval performance. Behavioral & Neural Biology, 39(2), 268-276.
Hausmann, M., Ergun, G., Yazgan, Y., Güntürkün, O. (2002). Sex differences in line bisection as a function of hand. Neuropsychologia, 40, 235-240.
Haynes, S. G., & Feinleib, M. (1980). Women, work and coronary heart disease: Prospective findings from the Framingham heart study. American Journal of Public Health, 70(2), 133-141.
Hebb, D. O. (1955). Drives and the C.N.S. (conceptual nervous system). Psychological Review, 62, 243-254.
Heilman, K. M., & Bowers, D. (1990). Neuropsychological studies of emotional changes induced by right and left hemispheric lesions. In N. L. Stein, B. Leventhal, & T. Trabasso (Eds.), Psychological and Biological Approaches to Emotion (pp. 97-113). Hillsdale, NJ: Lawrence Erlbaum.
Heilman, K. M., Bowers, D., & Valenstein, E. (1985). Emotional disorders associated with neurological diseases. In K. M. Heilman & E. Valenstein (Eds.), Clinical Neuropsychology (2nd ed.,). New York: Oxford University Press.
Heilman, K. M., Bowers, D., & Valenstein, E. (1993). Emotional disorders associated with neurological diseases. In K. M. Heilman & E. Valenstein (Eds.), Clinical Neuropsychology (3rd ed., pp. 461-497). New York: Oxford University Press.
Heilman, K. M., & Van den Abell, T. (1979). Right hemispheric dominance for mediating cerebral activation. Neuropsychologia, 17(3-sup-4), 315-321.
Heller, W. (1990). The neuropsychology of emotion: Developmental patterns and implications for psychopathology. In N. L. L. B. Stein (Ed.), Psychological and biological approaches to emotion (pp. 167-211). Hillsdale, NJ, USA: Lawrence Erlbaum Associates, Inc.
Hellige, J. B. (1990). Hemispheric asymmetry. Annual Review of Psychology, 41, 55-80.
Helmers, K. F., Krantz, D. S., Howell, R. H., Klein, J., Bairey, C. N., & Rozanski, A. (1993). Hostility and myocardial ischemia in coronary artery disease patients: Evaluation by gender and ischemic index [see comments]. Psychosomatic Medicine, 55(1), 29-36.

Herridge, M. L. (1996). The effects of hostility and arousal on facial affect perception: A test of a neuropsychological model of hostility. Dissertation Abstracts International: Section B: the Sciences & Engineering, 57(3-B), 2208.

Herridge, M. L., Harrison, D. W., & Demaree, H. A. (1997). Hostility, facial configuration, and bilateral asymmetry on galvanic skin response. Psychobiology, 25(1), 71-76.

Higgins, D. A. (1999). Cardiovascular Reactivity to Speech Processing and Cold Pressor Stress: Evidence for Sex Differences in Dynamic Functional Cerebral Laterality. Unpublished master's thesis. Virginia Polytechnic Institute and State University, Blacksburg.

Higgins, D. A., Williamson, J., & Harrison, D. W. (in preparation). Sex differences in motor asymmetry. .

Hines, M. (1990). Gonadal hormones and cognitive development. In Balthazart (Ed.), Hormones, Brain, and Behavior in Vertebrates: 1. Comparative Physiology (pp. 51-63). Basel, Switzerland: Karger.

Hines, M., & Shipley, C. (1984). Prenatal exposure to diethylstilbestrol (DES) and the development of sexually dimorphic cognitive abilities and cerebral lateralization. Developmental Psychology, 20, 81-94.

Hirst, G. (1982). An evaluation of evidence for innate sex differences in linguistic ability. Journal of Psycholinguistic Research, 11(2), 95-113.

Holman, S. D., & Hutchison, J. B. (1991). Lateralized action of androgen on development of behavior and brain sex differences. Brain Research Bulletin, 27, 261-265.

Howard, R., Fenwick, P., Brown, D., & Norton, R. (1992). Relationship between CNV asymmetries and individual differences in cognitive performance, personality and gender. International Journal of Psychophysiology, 13(3), 191-7.

Hugdahl, K. (1995). Psychophysiology: The mind-body perspective. Cambridge, MA, USA: Harvard University Press.

Hugdahl, K. (1996). Brain laterality--beyond the basics. European Psychologist, 1(3), 206-220.

Hugdahl, K., Franzon, M., Andersson, B., & Walldebo, G. (1983). Heart rate responses (HRR) to lateralized visual stimuli. Pavlovian Journal of Biological Science, 18, 186-198.

Inglis, J., & Lawson, J. S. (1981). Sex differences in the effects of unilateral brain damage on intelligence. Science, 212, 693-695.

Irle, E., Peper, M., Wowra, B., & Kunze, S. (1994). Mood changes after surgery for tumors of the cerebral cortex. Archives of Neurology, 51(2), 164-174.

Johnsen, B. H. (1993). Cerebral asymmetry in autonomic conditioning to facial expressions: Sex differences. Scandinavian Journal of Psychology, 34(4), 363-70.

Johnson, E. S., & Meade, A. C. (1987). Developmental patterns of spatial ability: An early sex difference. Child Development, 58(3), 725-740.

Johnson, S. C., Pinkston, J. B., Bigler, E. D., & Blatter, D. D. (1996). Corpus callosum morphology in normal controls and traumatic brain injury: Sex differences, mechanisms of injury, and neuropsychological correlates. Neuropsychology, 10, 408-415.

Jones-Gotman, M., & Milner, B. (1977). Design fluency: The invention of nonsense drawings after focal cortical lesions. Neuropsychologia, 15(4-sup-5), 653-674.

Joseph, R. (1992). The limbic system: Emotion, laterality, and unconscious mind. Psychoanalytic Review, 79(3), 405-456.

Joseph, R. (1996). Neuropsychiatry, Neuropsychology, and Clinical Neuroscience: Emotion, Evolution, Cognition, Language, Memory, Brain Damage, and Abnormal Behavior (2nd ed.). Baltimore, MD, USA: Williams & Wilkins Co.

Joslyn, W. D. (1973). Androgen-induced social dominance in infant female rhesus monkeys. Journal of Child Psychology and Psychiatry, 14, 137-145.

Julius, S., Weder, A. B., & Hinderliter, A. L. (1986). Does behaviorally induced blood pressure variability lead to hypertension? In K. A. Matthews, S. M. Weiss, T. Detre, T. M. Dembroski, B. Falkner, S. B. Manuck, & R. B. Williams (Eds.), Handbook of Stress, Reactivity, and Cardiovascular Disease (pp. 71-82). New York: John Wiley & Sons.

Kagerer, F. A., Wittmann, M., Szelag, E., Steinbüchel, N. V. (2002). Cortical involvement in temporal reproduction: Evidence for differential roles of the hemispheres. Neuropsychologia, 40, 357-366.
Kalat, J. W. (1995). Emotion, Autonomic Nervous System Arousal, and Health Problems, Biological Psychology (5th ed., pp. 415-447). Pacific Grove, CA: Brooks-Cole Publishing Co.
Katkin, E. S., & Reed, S. D. (1988). Cardiovascular asymmetries and cardiac perception. International Journal of Neuroscience, 39(1-2), 45-52.
Kaufer, D. I., & Lewis, D. A. (1999). Frontal lobe anatomy and cortical connectivity. In B. L. Miller & J. L. Cummings (Eds.), The Human Frontal Lobes: Functions and Disorders (pp. 27-44). New York: The Guilford Press.
Kelly, D. D. (1991). Sexual differentiation of the nervous system. In E. R. Kandel, J. H. Schwartz, & T. M. Jessell (Eds.), Principles of Neural Science (3rd ed., pp. 958-973). Norwalk, Connecticut: Appleton & Lange.
Keltikangas-Järvinen, L. (1992). Gender differences in coronary-prone aggression. In K. Björkqvist & P. Niemelä (Eds.), Of Mice and Women: Aspects of Female Aggression (pp. 123-131). San Diego, CA: Academic Press.
Kertesz, A., Polk, M., Black, S. E., & Howell, J. (1992). Anatomical Asymmetries and Functional Laterality. Brain, 115(Pt 2), 589-605.
Khaw, K. T., & Barrett-Connor, E. (1988). Blood pressure and endogenous testosterone in men: An inverse relationship. Journal of Hypertension, 6(4), 329-32.
Kimura, D. (1961). Cerebral dominance and the perception of verbal stimuli. Canadian Journal of Psychology, 15, 166-171.
Kimura, D. (1967). Functional asymmetry of the brain in dichotic listening. Cortex, 3, 163-178.
Kimura, D. (1987). Are men's and women's brains really different? Canadian Psychology, 28(2), 133-147.
Kimura, D. (1993a). Neuromotor mechanisms in human communication. New York, NY, USA: Clarendon Press/Oxford University Press.
Kimura, D. (1993b). Sex differences in the brain .
Kimura, D., & Harshman, R. A. (1984). Sex differences in brain organization for verbal and non-verbal functions. Progress in Brain Research, 61, 423-441.
Kingsberg, S. A., LaBarba, R. C., & Bowers, C. A. (1987). Sex differences in lateralization for spatial abilities. Bulletin of the Psychonomics Society, 25, 247-250.
Kinsbourne, M. (1978). Asymmetrical Function of the Brain. New York, NY: Cambridge University Press.
Kinsbourne, M. (1982). Hemispheric specialization and the growth of human understanding. American Psychologist, 37(4), 411-20.
Kinsbourne, M., & Cook, J. (1971). Generalized and lateralized effects of concurrent verbalization on a unimanual skill. Quarterly Journal of Experimental Psychology, 23, 341-345.
Klüver, H., & Bucy, P. C. (1937). "Psychic blindness" and other symptoms following bilateral temporal lobectomy in rhesus monkeys. American Journal of Psychology, 119, 352-353.
Klüver, H., & Bucy, P. C. (1938). An analysis of certain effects of bilateral temporal lobectomy in the rhesus monkey. Journal of Psychology, 5, 33-54.
Knight, G. P., Fabes, R. A., & Higgins, D. A. (1996). Concerns about drawing causal inferences from meta-analyses: An example in the study of gender differences in aggression. Psychological Bulletin, 119, 410-421.
Knudsen, E. I. (1991). Dynamic space codes in the superior colliculus. Current Opinion in Neurobiology, 1, 628-632.
Knudsen, E. I., & Brainard, M. S. (1991). Visual instruction of the neural map of auditory space in the developing optic tectum. Science, 253, 85-87.
Knudsen, E. I., Esterly, S. D., & Du Lac, S. (1991). Stretched and upside-down maps of auditory space in the optic tectum of blind-reared owls; acoustic basis and behavioral correlates. Journal of Neuroscience, 11, 1727-1747.

Kramer, J. H., Delis, D. C., Kaplan, E., O'Donnell, L., & Prifitera, A. (1997). Developmental sex differences in verbal learning. Neuropsychology, 11(4), 577-584.
Krantz, D. S., & Raisen, S. E. (1988). Environmental stress, reactivity, and ischaemic heart disease. British Journal of Medical Psychology, 61, 3-16.
Krynicki, W. E. (1978). Cerebral dysfunction in repetitively assaultive adolescents. Journal of Nervous and Mental Disease, 166, 59-67.
Kubany, E. S., Gino, A., Denny, N. R., & Torigoe, R. Y. (1994). Relationship of cynical hostility and PTSD among Vietnam veterans. Journal of Traumatic Stress, 7(1), 21-31.
Lane, R. D., & Jennings, J. R. (1995). Hemispheric asymmetry, autonomic asymmetry, and the problem of sudden cardiac death. In R. J. H. K. Davidson (Ed.), Brain asymmetry (pp. 271-304). Cambridge, MA, USA: M.I.T. Press.
Lane, R. D., Novelly, R., Cornell, C., Zeitlin, S., & Schwartz, G. E. (1988). Asymmetrical hemispheric control of heart rate (abstract). Psychophysiology, 25, 464.
Lane, R. D., & Schwartz, G. E. (1987). Induction of lateralized sympathetic input to the heart by the CNS during emotional arousal: A possible neurophysiologic trigger of sudden cardiac death. Psychosomatic Medicine, 49(3), 274-284.
Lane, R. D., & Schwartz, G. E. (1990). The neuropsychophysiology of emotion. Functional Neurology, 5, 263-266.
Lawler, K. A., Wilcox, Z. C., & Anderson, S. F. (1995). Gender differences in patterns of dynamic cardiovascular regulation. Psychosomatic Medicine, 57(4), 357-65.
Lawlor, D. A., Ebrahim, S., Smith, G. D. (2001). Sex matters: Secular and geographical trends in sex differences in coronary heart disease mortaility. British Medical Journal, 323, 541-545.
Lee, M. A., & Cameron, O. G. (1986-87). Anxiety, Type A behavior, and cardiovascular disease. International Journal of Psychiatry in Medicine, 16, 123-129.
Lerner, D. J., & Kannel, W. B. (1986). Patterns of coronary heart disease morbidity and mortality in the sexes: a 26-year follow-up of the Framingham population. American Heart Journal, 111, 383-390.
Levy, J., & Heller, W. (1992). Gender differences in human neuropsychological function. In A. A. M. H. Gerall (Ed.), Sexual differentiation. Handbook of behavioral neurobiology (Vol. 11, pp. 245-274). New York, NY, USA: Plenum Press.
Lewis, D. W., & Diamond, M. C. (1995). The influence of gonadal steroids on the asymmetry of the cerebral cortex. In R. J. Davidson & K. Hugdahl (Eds.), Brain Asymmetry (pp. 31-50). Cambridge, Massachusetts: Massachusetts Institute of Technology Press.
Ley, R., & Bryden, M. (1979). Hemispheric differences in processing emotions and faces. Brain and Language, 7, 127-138.
Lezak, M. D. (1995). Neuropsychological Assessment (3rd ed.). New York: Oxford University Press.
Linden, W., Chambers, L., Maurice, J., & Lenz, J. W. (1993). Sex differences in social support, self-deception, hostility, and ambulatory cardiovascular activity. Health Psychology, 12(5), 376-80.
Lindsley, D. B. (1951). Emotion. In S. S. Stevens (Ed.), Handbook of Experimental Psychology (pp. 473-516). New York: Wiley.
Liotti, M., & Tucker, D. M. (1995). Emotion in asymmetric corticolimbic networks. In R. J. Davidson & K. Hugdahl (Eds.), Brain Asymmetry (pp. 389-423). Cambridge, Massachusetts: Massachusetts Institute of Technology Press.
Lloyd-Jones, D. M., Larson, M. G., Beiser, A., & Levy, D. (1999). Lifetime risk of developing coronary heart disease. Lancet, 353, 89-92.
Luria, A. R. (1962). Higher Cortical Functions In Man. Moscow: Moscow University Press.
Luria, A. R. (1973). The Working Brain: An Introduction to Neuropsychology (B. Haigh, Trans.). London: The Penguin Press.
Malmo, R. B. (1959). Activation: A neuropsychological dimension. Psychological Review, 66, 367-386.
Manuck, S. B., Kaplan, J. R., & Clarkson, T. B. (1983). Behaviorally induced heart rate reactivity and atherosclerosis in cynomolgus monkeys. Psychosomatic Medicine, 45(2), 95-108.

Manuck, S. B., & Krantz, D. S. (1986). Psychophysiologic reactivity in coronary heart disease and essential hypertension. In T. M. Dembroski, T. Detre, B. Falkner, S. B. Manuck, K. A. Matthews, S. M. Weiss, & J. R. B. Williams (Eds.), Handbook of Stress, Reactivity, and Cardiovascular Disease (pp. 11-34). New York: John Wiley & Sons.

Mark, D. B. (2000). Sex bias in cardiovascular care: should women be treated more like men? JAMA: The Journal of the American Medical Association, 283(5), 659-61.

Matthews, K. A., Woodall, K. L., & Allen, M. T. (1993). Cardiovascular reactivity to stress predicts future blood pressure status. Hypertension, 22(4), 479-85.

Matthews, K. A., Woodall, K. L., Engebretson, T. O., McCann, B. S., Stoney, C. M., Manuck, S. B., & Saab, P. G. (1992). Influence of age, sex, and family on Type A and hostile attitudes and behaviors. Health Psychology, 11(5), 317-23.

McCourt, M. E., Mark, V. W., Radonovich, K. J., Willison, S. K., & Freeman, P. (1997). The effects of gender, menstrual phase and practice on the perceived location of the midsagittal plane. Neuropsychologia, 35(5), 717-24.

McGlone, J. (1977). Sex differences in the cerebral organization of verbal functions in patients with unilateral brain lesions. Brain, 100(4), 775-93.

McGlone, J. (1980). Sex differences in human brain asymmetry: A critical survey. Behavioral and Brain Sciences, 3, 215-263.

McGlone, J., & Kertesz, A. (1973). Sex differences in cerebral processing of visuospatial tasks. Cortex, 9(3), 313-20.

McKeever, W. F. (1986). The influences of handedness, sex, familial sinistrality and androgyny on language laterality, verbal ability, and spatial ability. Cortex, 22(4), 521-37.

McKeever, W. F., & Dixon, M. S. (1981). Right-hemisphere superiority for discriminating memorized from nonmemorized faces: affective imagery, sex, and perceived emotionally effects. Brain and Language, 12(2), 246-60.

Mead, L. A., & Hampson, E. (1996). Asymmetric effects of ovarian hormones on hemispheric activity: Evidence from dichotic and tachistoscopic tests. Neuropsychology, 10, 578-587.

Miceli, G., Caltagirone, C., Gainotti, G., Masullo, C., & Silveri, M. (1981). Neuropsychological correlates of localized cerebral lesions in non-aphasic brain-damaged patients. Journal of Clinical Neuropsychology, 3(1), 53-63.

Miller, E. (1984). Verbal fluency as a function of a measure of verbal intelligence and in relation to different types of cerebral pathology. British Journal of Clinical Psychology, 23(1), 53-57.

Miller, T. Q., Smith, T. W., Turner, C. W., Guijarro, M. L., & Hallet, A. J. (1996). A meta-analytic review of research on hostility and physical health. Psychological Bulletin, 119(2), 322-348.

Milner, B. (1964). Some effects of frontal lobectomy in man. In J. Warren & K. Akert (Eds.), The Frontal Granular Cortex and Behavior (pp. 313-334). New York: McGraw Hill.

Moffat, S. D., & Hampson, E. (2000). Salivary testosterone concentrations in left-handers: an association with cerebral language lateralization? Neuropsychology, 14(1), 71-81.

Moffat, S. D., Hampson, E., & Hatzipantelis, M. (1998). Navigation in a "virtual" maze: Sex differences and correlation with psychometric measures of spatial ability in humans. Evolution & Human Behavior, 19(2), 73-87.

Muniz, J., Garcia-Cueto, E., Garcia-Alcaniz, E., & Yela, M. (1985). Analysis of verbal, oral, and written fluency in men and women. Revista de Psicologia General y Aplicada, 40(2), 255-275.

Nauta, W. J. (1971). The problem of the frontal lobe: a reinterpretation. Journal of Psychiatric Research, 8(3), 167-87.

Nolte, J., & Angevine, J., J. B. (1995). The Human Brain: In Photographs and Diagrams. St. Louis: Mosby.

O'Boyle, M. W., & Hellige, J. B. (1989). Cerebral hemisphere asymmetry and individual differences in cognition. Learning & Individual Differences, 1(1), 7-35.

Panksepp, J. (1992). A critical role for "affective neuroscience" in resolving what is basic about basic emotions. Psychological Review, 99(3), 554-60.

Papez, J. W. (1995). A proposed mechanism of emotion. 1937 [classical article]. Journal of Neuropsychiatry and Clinical Neuroscience, 7(1), 103-12.
Pardo, J. V., Pardo, P. J., & Raichle, M. E. (1993). Neural correlates of self-induced dysphoria. American Journal of Psychiatry, 150, 713-719.
Pendleton, M. G., Heaton, R. K., Lehman, R. A., & Hulihan, D. M. (1982). Diagnostic utility of the Thurston Word Fluency Test in neuropsychological evaluations. Journal of Clinical Neuropsychology, 4(4), 307-317.
Perret, E. (1974). The left frontal lobe of man and the suppression of habitual responses in verbal categorical behaviour. Neuropsychologia, 12(3), 323-330.
Pizzamiglio, L., Mammucari, A., & Razzano, C. (1985). Evidence for sex differences in brain organization in recovery from aphasia. Brain and Language, 25, 213-223.
Pope, M. K., Smith, T. W., & Rhodewalt, F. (1990). Cognitive, behavioral, and affective correlates of the Cook and Medley Hostility Scale. Journal of Personality Assessment, 54(3-4), 501-14.
Porges, S. W. (1995). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A polyvagal theory. Psychophysiology, 32, 301-318.
Porges, S. W., Doussard-Roosevelt, J. A., & Maiti, A. K. (1994). Vagal tone and the physiological regulation of emotion. Monographs of the Society for Research in Child Development, 59(2-3), 167-86.
Previc, F. H. (1991). A general theory concerning the prenatal origins of cerebral lateralization in humans. Psychological Review, 98(3), 299-334.
Pribram, K. H. (1964). Perspektivy razvitiia neiropsikhologii. / Toward a science of neuropsychology. Voprosy Psikhologii, 2, 16-26.
Pribram, K. H. (1969a). The frontal lobes of primates, Organization of Physiological Functional Systems . Moscow: Meditsina.
Pribram, K. H. (1969b). The primate frontal cortex. Neuropsychologia, 7, 259-266.
Pribram, K. H. (1975). The primate frontal cortex: Progress report 1975. Acta Neurobiologiae Experimentalis, 35(5-6), 609-25.
Pribram, K. H., & Luria, A. R. (1973). Psychophysiology of the Frontal Lobes. New York: Academic Press.
Ramier, A., & Hecaen, H. (1970). Role respectif des atteintes frontales et de la lateralisation lesionelle dans les deficits de la "fluence verbale". Revue Neurologique, 123, 17-22.
Ray, W. J., Morell, M., Frediani, A. W., & Tucker, D. (1976). Sex differences and lateral specialization of hemispheric functioning. Neuropsychologia, 14, 391-394.
Rich, J. B., & Maki, P. (2000). Estrogen, Testosterone, and the Brain: A Review of Neuropsychological, Aging, and Neuroimaging Studies. Paper presented at the International Neuropsychological Society, Denver, Colorado.
Rinn, W. E. (1984). The neuropsychology of facial expression: A review of the neurological and psychological mechanisms for producing facial expressions. Psychological Bulletin, 95(1), 52-77.
Rippon, G. (1990). Individual differences in electrodermal and electroencephalographic asymmetries. International Journal of Psychophysiology, 8(3), 309-20.
Rosen, A. D., Gur, R. C., Sussman, N., Gur, R. E., & Hurtig, H. (1982). Hemispheric asymmetry in the control of heart rate. Abstracts of Social Neuroscience, 8, 917.
Rosenman, R. H., Brand, R. J., Jenkins, D., Friedman, M., Straus, R., & Wurm, M. (1975). Coronary heart disease in Western Collaborative Group Study. Final follow-up experience of 8 1/2 years. JAMA: The Journal of the American Medical Association, 233(8), 872-7.
Rosenman, R. H., Brand, R. J., Sholtz, R. I., & Friedman, M. (1976). Multivariate prediction of coronary heart disease during 8.5 year follow-up in the Western Collaborative Group Study. American Journal of Cardiology, 37(6), 903-10.
Rosenman, R. H., Friedman, M., Straus, R., Wurm, M., Jenkins, C. D., & Messinger, H. B. (1966). Coronary heart disease in the Western collaborative group study. A follow-up experience of two years. JAMA: The Journal of the American Medical Association, 195(2), 86-92.

Rosvold, H. E., Mirsky, A. F., & Pribram, K. H. (1954). Influence of amygdalectomy on social behavior in monkeys. Journal of Comparative and Physiological Psychology, 47, 173-178.
Rothbart, M. K., Taylor, S. B., & Tucker, D. M. (1989). Right-sided facial asymmetry in infant emotional expression. Neuropsychologia, 27(5), 675-87.
Ruff, R. M., Allen, C. C., Farrow, C. E., Niemann, H., & et al. (1994). Figural fluency: Differential impairment in patients with left versus right frontal lobe lesions. Archives of Clinical Neuropsychology, 9(1), 41-55.
Ruff, R. M., Evans, R., & Marshall, L. F. (1986). Impaired verbal and figural fluency after head injury. Archives of Clinical Neuropsychology, 1(2), 87-101.
Ruff, R. M., Light, R. H., & Evans, R. W. (1987). The Ruff Figural Fluency Test: A normative study with adults. Developmental Neuropsychology, 3(1), 37-51.
Rushmer, R. F. (1976). Structure and Function of the Cardiovascular System (2nd ed.). Philadelphia: Saunders.
Sackeim, H. A., & Gur, R. C. (1978). Lateral asymmetry in intensity of emotional expression. Neuropsychologia, 16(4), 473-81.
Sackeim, H. A., Gur, R. C., & Saucy, M. C. (1978). Emotions are expressed more intensely on the left side of the face. Science, 202(4366), 434-6.
Schloesser, R., Hutchinson, M., Joseffer, S., Rusinek, H., Saarimaki, A., Stevenson, J., Dewey, S. L., & Brodie, J. D. (1998). Functional magnetic resonance imaging of human brain activity in a verbal fluency task. Journal of Neurology, Neurosurgery & Psychiatry, 64(4), 492-498.
Schwartz, G. E., Ahern, G. L., & Brown, S. L. (1979). Lateralized facial muscle response to positive and negative emotional stimuli. Psychophysiology, 16(6), 561-71.
Schwartz, G. E., Davidson, R. J., & Maer, F. (1975). Right hemisphere lateralization for emotion in the human brain: interactions with cognition. Science, 190(4211), 286-8.
Schwartz, S., & Baldo, J. (2001). Distinct patterns of word retrieval in right and left frontal lobe patients: A multidimensional perspective. Neurospsychologia, 39, 1209-1217.
Shaikh, M. B., Schubert, K., & Siegel, A. (1994). Basal amygdaloid facilitation of midbrain periaqueductal gray elicited defensive rage behavior in the cat is mediated through NMDA receptors. Brain Research, 635(1-2), 187-95.
Shaikh, M. B., Steinberg, A., & Siegel, A. (1993). Evidence that substance P is utilized in medial amygdaloid facilitation of defensive rage behavior in the cat. Brain Research, 625(2), 283-94.
Shapiro, D., Jamner, L. D., & Goldstein, I. B. (1997). Daily mood states and ambulatory blood pressure. Psychophysiology, 34(4), 399-405.
Shaywitz, B. A., Shaywitz, S. E., Pugh, K. R., Constable, R. T., Skudlarski, P., Fulbright, R. K., Bronen, R. A., Fletcher, J. M., Shankweller, D. P., Katz, L., & Gore, J. C. (1995). Sex differences in the functional organization of the brain for language. Nature, 373(6515), 607-609.
Shekelle, R. B., Gale, M., Ostfeld, A. M., & Paul, O. (1983). Hostility, risk of coronary heart disease, and mortality. Psychosomatic Medicine, 45(2), 109-14.
Shepard, R. N., & Metzler, J. (1971). Mental rotation of three-dimensional objects. Science, 171(3972), 701-703.
Siegel, A., & Pott, C. B. (1988). Neural substrates of aggression and flight in the cat. Progress in Neurobiology, 31(4), 261-83.
Silberman, E. K., & Weingartner, H. (1986). Hemispheric lateralization of functions related to emotion. Brain & Cognition, 5(3), 322-353.
Silverman, I., & Eals, M. (1992). Sex differences in spatial abilities: Evolutionary theory and data. In J. H. C. L. Barkow (Ed.), The adapted mind: Evolutionary psychology and the generation of culture (pp. 533-549). New York, NY, USA: Oxford University Press.
Smith, T. W. (1994). Concepts and methods in the study of anger, hostility and health. In A. W. Siegman & T. W. Smith (Eds.), Anger, Hostility and the Heart (pp. 23-42). Hillsdale, NJ: Erlbaum.
Snyder, K. A., Harrison, D. W., & Shenal, B. V. (1998). The affective auditory verbal learning test: Peripheral arousal correlates. Archives of Clinical Neuropsychology, 13, 251-258.

Speilberger, C. D. (1968). Self-Evaluation Questionnaire. STAI Form X-2 . Palo Alto, CA: Consulting Psychologists Press.
Spence, S., Shapiro, D., & Zaidel, E. (1996). The role of the right hemisphere in the physiological and cognitive components of emotional processing. Psychophysiology, 33(2), 112-122.
Springer, S. P., & Deutsch, G. (1989). Sex and Asymmetry, Left Brain, Right Brain (3rd ed., pp. 213-225). New York: W. H. Freeman & Co.
Stalans, L., & Wedding, D. (1985). Superiority of the left hemisphere in the recognition of emotional faces. International Journal of Neurosciences, 25, 219-223.
Stamler, J. (1992). Established major coronary risk factors. In M. Marmot & P. Elliott (Eds.), Coronary Heart Disease Epidemiology: From Aetiology to Public Health (pp. 35-66). New York: Oxford University Press.
Steingart, R. M., Packer, M., Hamm, P., Coglianese, M. E., Gersh, B., Geltman, E. M., Sollano, J., Katz, S., Moye, L., Basta, L. L., & et al. (1991). Sex differences in the management of coronary artery disease. Survival and Ventricular Enlargement Investigators. New England Journal of Medicine, 325(4), 226-30.
Steptoe, A. (1980). Blood pressure. In I. Martin & P. H. Venables (Eds.), Techniques in Psychophysiology (pp. 247-273). New York: John Wiley and Sons.
Strub, R. L., & Black, F. W. (1985). The Mental Status Examination in Neurology. Philadelphia: F. A. Davis Company.
Strub, R. L., & Black, F. W. (1988a). The bedside mental status examination. In F. G. J. Boller (Ed.), Handbook of neuropsychology (Vol. 1, pp. 29-46). Amsterdam, Netherlands: Elsevier Science Publishing Co, Inc.
Strub, R. L., & Black, F. W. (1988b). Neurobehavioral disorders: A clinical approach. Philadelphia, PA, USA: F.
Suarez, E. C., & Williams, R. B., Jr. (1989). Situational determinants of cardiovascular and emotional reactivity in high and low hostile men. Psychosomatic Medicine, 51(4), 404-18.
Sundet, K. (1986). Sex differences in cognitive impairment following unilateral brain damage. Journal of Clinical and Experimental Neuropsychology, 8(1), 51-61.
Sundet, K. (1988). Sex differences in the severity and type of aphasia. Scandinavian Journal of Psychology, 29, 168-179.
Swartz, C. M., Abrams, R., Lane, R. D., Dubois, M. A., & Srinivasaraghavan, J. (1994). Heart rate differences between right and left unilateral electroconvulsive therapy. Journal of Neurology, Neurosurgery, and Psychiatry, 57, 97-99.
Taylor, E. S. (1976). Beck's Obstetrical Practice and Fetal Medicine (10th ed.). Baltimore: Williams & Wilkins.
Thayer, R. E. (1989). The Biopsychology of Mood and Arousal. New York: Oxford University Press.
Theorell, T., & Haerenstam, A. (2000). Influence of gender on cardiovascular disease. In R. M. Eisler & M. Hersen (Eds.), Handbook of gender, culture, and health (pp. 161-177). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.
Tobin, J. N., Wassertheil-Smoller, S., Wexler, J. P., Steingart, R. M., Budner, N., Lense, L., & Wachspress, J. (1987). Sex bias in considering coronary bypass surgery. Annals of Internal Medicine, 107(1), 19-25.
Tortora, G. J., & Anagnostakos, N. P. (1990). Principles of Anatomy and Physiology (6th ed.). New York: Harper & Row.
Treiber, F. A., Musante, L., Riley, W., Mabe, P. A., Carr, T., Levy, M., & Strong, W. B. (1989). The relationship between hostility and blood pressure in children. Behavioral Medicine, 15(4), 173-8.
Trevarthen, C., (Ed.). (1990). Brain circuits and functions of the mind. Essays in honor of Roger W. Sperry. Cambridge, England: Cambridge University Press.
Trotman, S. C., & Hammond, G. R. (1979). Sex differences in task-dependent EEG asymmetries. Psychophysiology, 16(5), 429-31.

Tucha, O., Smely, C., & Lange, K. W. (1999). Verbal and figural fluency in patients with mass lesions of the left or right frontal lobes. Journal of Clinical & Experimental Neuropsychology, 21(2), 229-236.

Tucker, D. M. (1981). Lateral brain function, emotion, and conceptualization. Psychological Bulletin, 89(1), 19-46.

Tucker, D. M. (1984). Lateral brain function in normal and disordered emotion: Interpreting electroencephalographic evidence. Biological Psychology, 19(3-4), 219-235.

Tucker, D. M., Luu, P., & Pribram, K. H. (1995). Social and emotional self-regulation. Annals of the New York Academy of Sciences, 769, 213-39.

Tucker, D. M., Roth, R. S., Arneson, B. A., & Buckingham, V. (1977). Right hemisphere activation during stress. Neuropsychologia, 15(4-5), 697-700.

Tucker, D. M., Stenslie, C. E., Roth, R. S., & Shearer, S. L. (1981). Right frontal lobe activation and right hemisphere performance. Decrement during a depressed mood. Archives of General Psychiatry, 38(2), 169-74.

Tucker, D. M., Vannatta, K., & Rothlind, J. (1990). Arousal and activation systems and primitive adaptive controls on cognitive priming. In N. L. L. B. Stein (Ed.), Psychological and biological approaches to emotion (pp. 145-166). Hillsdale, NJ, USA: Lawrence Erlbaum Associates, Inc.

Tucker, D. M., & Williamson, P. A. (1984). Asymmetric neural control systems in human self-regulation. Psychological Review, 91(2), 185-215.

Turkewitz, G. (1993). The Influence of timing on the nature of cognition. In G. Turkewitz & D. A. Devenny (Eds.), Developmental Time and Timing (pp. 125-142). Hillsdale: Lawrence Erlbaum Associates.

Turkheimer, E., & Farace, E. (1992). A reanalysis of gender differences in IQ scores following unilateral brain lesions. Psychological Assessment, 4, 498-501.

U.S.D.H.H.S. (1988). Table 6. Mortality by education. A mortality study of one million persons by demographic, social, and economic factors: 1979-1981 follow-up, U.S. National Longitudinal Mortality Study (NIH Publication No. 88-2986; pp. 154-182) . Washington, DC: United States Department of Health and Human Services.

Valzelli, L. (1981). Psychobiology of Aggression and Violence. New York: Raven.

Van Strien, J. W., & Heijt, R. (1995). Altered visual field asymmetries for letter naming and letter matching as a result of concurrent presentation of threatening and nonthreatening words. Brain and Cognition, 29(2), 187-203.

Vandenberg, S. G., & Kuse, A. R. (1978). Mental rotations, a group test of three-dimensional spatial visualization. Perceptual & Motor Skills, 47(2), 599-604.

Vasan, R. S., Beiser, A., Seshadri, S., Larson, M. G., Kannel, W. B., D'Agostino, R. B., Levy, D. (2002). Residual Lifetime Risk for Developing Hypertension in Middle-aged Women and Men. The Framingham Heart Study. JAMA: The Journal of the American Medical Association, 287(8), 1003-1010.

Vikingstad, E. M., George, K. P., Johnson, A. F., & Cao, Y. (2000). Cortical language lateralization in right handed normal subjects using functional magnetic resonance imaging [In Process Citation]. Journal of the Neurological Sciences, 175(1), 17-27.

Vilkki, J., Levänen, S., & Servo, A. (2002). Interference in dual-fluency tasks after anterior and posterior cerebral lesions. Neuropsychologia, 40, 340-348.

Wada, Y., Nanbu, Y., Kadoshima, R., Jiang, Z. Y., Koshino, Y., & Hashimoto, T. (1996). Interhemispheric EEG coherence during photic stimulation: Sex differences in normal young adults. International Journal of Psychophysiology, 22(1-2), 45-51.

Walker, B. B., & Sandman, C. A. (1979). Human visual evoked responses are related to heart rate. Journal of Comparative and Physiological Psychology, 93, 717-729.

Walker, B. B., & Sandman, C. A. (1982). Visual evoked potentials change as heart rate and carotid pressure change. Psychophysiology, 19, 520-527.

Walker, S. F. (1994). The possible role of asymmetric laryngeal innervation in language lateralization: Points for and against. Brain and Language, 46(3), 482-9.
Weber, S. L., & Sackeim, H. A. (1984). The development of functional brain asymmetry in the regulation of emotion. In N. A. Fox & R. J. Davidson (Eds.), The Psychobiology of Affective Development (pp. 325-352). Hillsdale, NJ: Lawrence Erlbaum.
Wechsler, A. F. (1973). The effect of organic brain disease on recall of emotionally charged versus neutral narrative texts. Neurology, 23(2), 130-5.
Wedding, D., & Cyrus, P. (1986). Recognition of emotion in hemifaces presented to the left and right visual fields. International Journal of Neuroscience, 30, 161-164.
Wendt, P. E., & Risberg, J. (1994). Cortical activation during visual spatial processing: Relation between hemispheric asymmetry of blood flow and performance. Brain and Cognition, 24(1), 87-103.
Williams, R. B. (1984). Help your heart: hostility and heart disease. Nebraska Nurse, 17(1), 8.
Williams, R. B. (1986). Patterns of reactivity and stress. In K. A. Matthews, S. M. Weiss, T. Detre, T. M. Dembroski, B. Falkner, S. B. Manuck, & R. B. Williams (Eds.), Handbook of Stress, Reactivity, and Cardiovascular Disease (pp. 109-125). New York: John Wiley & Sons.
Williams, R. B., Jr. (1987a). Psychological factors in coronary artery disease: Epidemiologic evidence. Circulation, 76(1 Pt 2), I117-23.
Williams, R. B., Jr. (1987b). Refining the type A hypothesis: Emergence of the hostility complex. American Journal of Cardiology, 60(18), 27J-32J.
Williams, R. B. (1996). Coronary-prone behaviors, hostility, and cardiovascular health: Implications for behavioral and pharmacological interventions. In K. S. N. Orth-Gomer (Ed.), Behavioral medicine approaches to cardiovascular disease prevention (pp. 161-168). Mahwah, NJ, USA: Lawrence Erlbaum Associates, Inc.
Williams, R. B., & Anderson, N. B. (1987). Hostility and coronary heart disease. In J. W. Elias & P. H. Marshall (Eds.), Cardiovascular Disease and Behavior (pp. 17-37). New York: Hemisphere Publishing Corporation.
Williams, R. B., Jr., Barefoot, J. C., Haney, T. L., Harrell, F. E., Jr., Blumenthal, J. A., Pryor, D. B., & Peterson, B. (1988). Type A behavior and angiographically documented coronary atherosclerosis in a sample of 2,289 patients. Psychosomatic Medicine, 50(2), 139-52.
Williams, R. B., Barefoot, J. C., & Shekelle, R. B. (1985). The health consequences of hostility. In M. A. Chesney, S. E. Goldston, & R. H. Rosenman (Eds.), Anger and Hostility in Cardiovascular and Behavioral Disorders (pp. 173-186). New York: Hemisphere / McGraw Hill.
Williams, R. B., & et al. (1982). Type A behavior and elevated physiological and neuroendocrine responses to cognitive tasks. Science, 218(4571), 483-485.
Williams, R. B., Jr., Haney, T. L., Lee, K. L., Kong, Y. H., Blumenthal, J. A., & Whalen, R. E. (1980). Type A behavior, hostility, and coronary atherosclerosis. Psychosomatic Medicine, 42(6), 539-49.
Williams, R. B., Jr., Suarez, E. C., Kuhn, C. M., Zimmerman, E. A., & Schanberg, S. M. (1991). Biobehavioral basis of coronary-prone behavior in middle-aged men. Part I: Evidence for chronic SNS activation in Type As. Psychosomatic Medicine, 53(5), 517-27.
Williamson, J. (1999). Differential Effects of Hostility on Frontal Lobe Performance: A Dual Task Approach with Fluency and Cardiovascular Regulation. Unpublished master's thesis. Virginia Polytechnic Institute and State University, Blacksburg.
Witelson, S. F. (1991). Neural sexual mosaicism: Sexual differentiation of the human temporo-parietal region for functional symmetry. Psychoneuroendocrinology, 16, 131-153.
Wittling, W. (1990). Psychophysiological correlates of human brain asymmetry: Blood pressure changes during lateralized presentation of an emotionally laden film. Neuropsychologia, 28, 457-470.
Wittling, W. (1995). Brain asymmetry in the control of autonomic-physiologic activity. In R. J. Davidson & K. Hugdahl (Eds.), Brain Asymmetry (pp. 305-357). Cambridge, Massachusetts: Massachusetts Institute of Technology Press.
Wittling, W. (1997). Brain asymmetry and autonomic control of the heart. European Psychologist, 2, 313-327.

Wittling, W., Block, A., Genzel, S., & Schweiger, E. (1998a). Hemisphere asymmetry in parasympathetic control of the heart. Neuropsychologia, 36(5), 461-468.

Wittling, W., Block, A., Schweiger, E., & Genzel, S. (1998b). Hemisphere asymmetry in sympathetic control of the human myocardium. Brain and Cognition, 38, 17-35.

Wolf, O. T., Preut, R., Hellhammer, D. H., Kudielka, B. M., Schuermeyer, T. H., & Kirschbaum, C. (2000). Testosterone and cognition in elderly men: A single testosterone injection blocks the practice effect in verbal fluency, but has no effect on spatial or verbal memory. Biological Psychiatry, 47(7), 650-654.

Wolff, P. H., & Cohen, C. (1980). Dual task performance during bimanual coordination. Cortex, 16, 119-133.

Wood, F. B., Flowers, D. L., & Naylor, C. E. (1991). Cerebral laterality in functional neuroimaging. In F. L. Kitterle (Ed.), Cerebral Laterality: Theory and Research: The Toledo Symposium (pp. 103-115). Hillsdale, New Jersey: Lawrence Erlbaum Associates.

Yokoyama, K., Jennings, R., Ackles, P., Hood, P., & Boller, F. (1987). Lack of heart rate changes during an attention demanding task after right hemisphere lesions. Neurology, 37, 624-630.

Yoon, B. W., Morillo, C. A., Cechetto, D. F., & Hachinski, V. (1997). Cerebral hemispheric lateralization in cardiac autonomic control. Archives of Neurology, 54, 741-744.

Zamrini, E. Y., Meador, K. J., Loring, D. W., Nichols, F. T., Lee, G. P., Figueroa, R. E., & Thompson, W. O. (1990). Unilateral cerebral inactivation produces differential left/right heart rate responses. Neurology, 40, 1408-1411.

Zoccolotti, P., Caltagirone, C., Benedetti, N., & Gainotti, G. (1986). Perturbation des réponses végétatives aux stimuli émotionnels au cours des lésions hémisphériques unilatérales. L'Encéphale, 12, 263-268.

Appendix A

Informed Consent For Participants Of Investigative Projects

Title of the Project: A Neuropsychological Investigation of Sex Differences in Cardiovascular Reactivity to Verbal and Spatial Fluency Tasks: Testing a New Model of Sex Differences in Cardiovascular Regulation and Disease.
Investigator: Dane A. Higgins. Experiment Number: 00-432

I. THE PURPOSE OF EXPERIMENT, PHASE ONE

You are invited to participate in a study to obtain data on measures purportedly assessing different psychological mechanisms. This is a group-screening phase in order to discover populations who qualify for further participation in this study This research ultimately attempts to determine the effects of sex differences on physiological and brain-related activity.

II. PROCEDURES TO BE FOLLOWED IN THE STUDY

To accomplish the goals of this study, you will be asked to complete five questionnaires.

III. DISCOMFORTS AND RISKS THROUGH PARTICIPATION IN THE STUDY

You may feel some embarrassment when answering the questionnaires. You may omit any questions that you feel are embarrassing. If you continue to have any problems associated with this study after you have left the experiment, please call Dr. David W. Harrison at 231-4422 so that he may assist you directly or direct you to other appropriate, available services.

IV. BENEFITS OF THIS PROJECT

Your participation in the project may ultimately help to identify the functions of certain neuropsychological sex differences and the potential cardiovascular benefits of different sex-based neuropsychological mechanisms. However, no specific guarantee of benefits has been made to encourage you to participate.

V. ANONYMITY OF PARTICIPANTS AND CONFIDENTIALITY OF RESULTS

Identifying information will be kept strictly confidential. At no time will the researchers release your personal information from the study to anyone other than individuals associated with the project without your written consent. The information you provide will have your name removed and only a participant number will identify you during subsequent analyses and written reports of this research.

VI. EXTRA CREDIT COMPENSATION

For participation in this study, you will receive one point extra credit for Psychology 2004 or the psychology class for which you are attempting to earn one point of extra credit.

VII. FREEDOM TO WITHDRAW

You are free to withdraw from this study at any time without penalty. If you choose to withdraw, you will not be penalized by reduction in points or grade for Psychology 2004 or the psychology class for

which you are attempting to earn extra credit. There are alternative choices for receiving extra credit for the course evaluation.

VIII. USE OF RESEARCH DATA

The information from this research may be used for scientific or educational purposes. It may be presented at scientific meetings and/or published and reproduced in professional journals, books, or used for any other purpose that the Psychology Department at Virginia Tech considers in the proper interest of education, knowledge, or research.

IX. APPROVAL OF RESEARCH

This project has been approved by the Human Subjects Committee of the Department of Psychology and by the Institutional Review Board of Virginia Tech, and assigned the number 00-432.

X. PARTICIPANT'S RESPONSIBILITIES

I voluntarily agree to participate in this study. I agree to disclose if I have a history of any of the following medical conditions: history of a severe head injury, stroke, epilepsy, seizures, paralysis, neurological surgery, other neurological or nervous system problems, or other conditions listed on the medical history questionnaire.

XI. PARTICIPANT'S PERMISSION

I have read and understand the above description of the study. I have had an opportunity to ask questions and have had them all answered to my satisfaction. I hereby acknowledge the above and give my voluntary consent for participation in this study. I also understand that I may withdraw from participation at any time without penalty.

Participant's Signature **Student Number** **Date** **Telephone Number**

Should I have any questions regarding this research and its conduct, I should contact any of the persons named below:

Dane A. Higgins Primary Researcher	552-6065 dane@vt.edu
David W. Harrison, Ph.D. Faculty Advisor Chair, HSC	231-4422 dwh@vt.edu
David Moore, Ph.D. Chair, IRB	231-4991 moored@vt.edu

Appendix B

Head Injury / Medical History Inventory (HIMHI)

Name:_____ Age:_____

Have you ever experienced or been diagnosed with any of the following, or are you experiencing any of the following at present? Please circle the appropriate response and explain any "Yes" responses below.

1.	Severe head trauma or injury	Yes	No
2.	Stroke or Aneurysm	Yes	No
3.	Learning disabilities (problems with reading, writing, or comprehension)	Yes	No
4.	Epilepsy or seizures	Yes	No
5.	Paralysis	Yes	No
6.	Neurological surgery	Yes	No
7.	Other neurological or nervous system problems	Yes	No
8.	Alcohol or drug problems	Yes	No
9.	Are you using alcohol or drugs (other than for prescribed purposes) at present?	Yes	No
10.	Past psychological / psychiatric problems	Yes	No
11.	Are you currently taking any prescription medications?	Yes	No
12.	Are you currently suffering from any medical conditions or illnesses?	Yes	No
13.	Do you have any uncorrected vision problems?	Yes	No
14.	Do you have an uncorrected hearing impairment?	Yes	No
15.	Do you have any problems or pain with movement (e.g., severe hand, arm, or shoulder pain with movement)?	Yes	No

16. **(Women)** Please estimate where you are in your menstrual cycle: Day _____ (1 - 28)
Day 1 = start of menses (bleeding).

*Please explain any "Yes" responses above:*_____

_____

Appendix C

Coren, Porac, and Duncan (CPD) Laterality Questionnaire

Circle the appropriate number after each item:	Right	Left	Both
With which hand would you throw a ball to hit a target?	1	-1	0
With which hand do you draw?	1	-1	0
With which hand do you use an eraser on paper?	1	-1	0
With which hand do you remove the top card when dealing?	1	-1	0
With which foot do you kick a ball?	1	-1	0
If you wanted to pick up a pebble with your toes, which foot would you use?	1	-1	0
If you had to step up onto a chair, which foot would you place on the chair first?	1	-1	0
Which eye would you use to peep through a keyhole?	1	-1	0
If you had to look into a dark bottle to see how full it was, which eye would you use?	1	-1	0
Which eye would you use to sight down a rifle?	1	-1	0
If you wanted to listen to a conversation going on behind a closed door, which ear would you place against the door?	1	-1	0
If you wanted to listen to someone's heartbeat, which ear would you place against their chest?	1	-1	0
Into which ear would you place the earphone of a transistor radio?	1	-1	0

# of Right + # of Left = Total Score

_____ + _____ = _____

Is mother left or right hand dominant? _____

Is father left or right hand dominant? _____

Appendix D

MCSDS

Listed below are a number of statements concerning personal attitudes and traits. Read each item and decide whether the statement is true or false as it pertains to you personally.

1.	Before voting I thoroughly investigate the qualifications of all the candidates.	T	F
2.	I never hesitate to go out of my way to help someone in trouble.	T	F
3.	It is sometimes hard for me to go on with my work if I am not encouraged.	T	F
4.	I have never intensely disliked anyone.	T	F
5.	On occasion I have had doubts about my ability to succeed in life.	T	F
6.	I sometimes feel resentful when I don't get my way.	T	F
7.	I am always careful about my manner of dress.	T	F
8.	My table manners at home are as good as when I eat out in a restaurant.	T	F
9.	If I could get into a movie without paying and be sure I was not seen, I would probably do it.	T	F
10.	On a few occasions, I have given up doing something because I thought too little of my ability.	T	F
11.	I like to gossip at times.	T	F
12.	There have been times when I felt like rebelling against people in authority even though I knew they were right.	T	F
13.	No matter who I'm talking to, I'm always a good listener.	T	F
14.	I can remember "playing sick" to get out of something.	T	F
15.	There have been occasions when I took advantage of someone.	T	F
16.	I'm always willing to admit it when I make a mistake.	T	F
17.	I always try to practice what I preach.	T	F

18.	I don't find it particularly difficult to get along with loud-mouthed, obnoxious people.	T	F
19.	I sometimes try to get even, rather than forgive and forget.	T	F
20.	When I don't know something I don't at all mind admitting it.	T	F
21.	I am always courteous, even to people who are disagreeable.	T	F
22.	At times, I have really insisted on having things my own way.	T	F
23.	There have been occasions when I felt like smashing things.	T	F
24.	I would never think of letting someone else be punished for my wrongdoings.	T	F
25.	I never resent being asked to return a favor.	T	F
26.	I have never been irked when people expressed ideas very different from my own.	T	F
27.	I never make a long trip without checking the safety of my car.	T	F
28.	There have been times when I was quite jealous of the good fortune of others.	T	F
29.	I have almost never felt the urge to tell someone off.	T	F
30.	I am sometimes irritated by people who ask favors of me.	T	F
31.	I have never felt that I was punished without cause.	T	F
32.	I sometimes think when people have a misfortune they only got what they deserved.	T	F
33.	I have never deliberately said something that hurt someone's feelings.	T	F

Appendix E

Self Evaluation Questionnaire

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of each statement to indicate how you feel right now, that is, at this moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

		Not At All	Somewhat	Moderately So	Very Much So
1.	I feel calm.	1	2	3	4
2.	I feel secure.	1	2	3	4
3.	I am tense.	1	2	3	4
4.	I feel strained.	1	2	3	4
5.	I feel at ease.	1	2	3	4
6.	I feel upset.	1	2	3	4
7.	I am presently worrying over possible misfortune.	1	2	3	4
8.	I feel satisfied.	1	2	3	4
9.	I feel frightened.	1	2	3	4
10.	I feel comfortable.	1	2	3	4
11.	I feel self-confident.	1	2	3	4
12.	I feel nervous.	1	2	3	4
13.	I am jittery.	1	2	3	4
14.	I feel indecisive.	1	2	3	4
15.	I am relaxed.	1	2	3	4
16.	I feel content.	1	2	3	4
17.	I am worried.	1	2	3	4
18.	I feel confused.	1	2	3	4
19.	I feel steady.	1	2	3	4
20.	I feel pleasant.	1	2	3	4

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then circle the number to the right of each statement to indicate how you generally feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

		Almost Never	Sometimes	Often	Almost Always
21.	I feel pleasant.	1	2	3	4
22.	I feel nervous and restless.	1	2	3	4
23.	I feel satisfied with myself.	1	2	3	4
24.	I wish I could be as happy as others seem to be.	1	2	3	4
25.	I feel like a failure.	1	2	3	4
26.	I feel rested.	1	2	3	4
27.	I am "calm, cool, and collected."	1	2	3	4
28.	I feel that difficulties are piling up so that I cannot overcome them.	1	2	3	4
29.	I worry too much over something that really doesn't matter.	1	2	3	4
30.	I am happy.	1	2	3	4
31.	I have disturbing thoughts.	1	2	3	4
32.	I lack self-confidence.	1	2	3	4
33.	I feel secure.	1	2	3	4
34.	I make decisions easily.	1	2	3	4
35.	I feel inadequate.	1	2	3	4
36.	I am content.	1	2	3	4
37.	Some unimportant thought runs through my mind and bothers me.	1	2	3	4
38.	I take disappointments so keenly that I can't put them out of my mind.	1	2	3	4
39.	I am a steady person.	1	2	3	4
40.	I get in a state of tension and turmoil as I think over recent concerns and interests.	1	2	3	4

Appendix F

CMHO

Directions: **If a statement is true or mostly true, as pertaining to you, circle the letter T. If a statement is false or usually not true about you, circle the letter F. Try to give a response to every statement.**

1.	When I take a new job, I like to be tipped off on who should be gotten next to.	T	F
2.	When someone does me wrong I feel I should pay him back if I can, just for the principle of the thing.	T	F
3.	I prefer to pass by school friends, or people I know but have not seen for a long time, unless they speak to me first.	T	F
4.	I have often had to take orders from someone who did not know as much as I did.	T	F
5.	I think a great many people exaggerate their misfortunes in order to gain the sympathy and help of others.	T	F
6.	It takes a lot of argument to convince most people of the truth.	T	F
7.	I think most people would lie to get ahead.	T	F
8.	Someone has it in for me.	T	F
9.	Most people are honest chiefly through the fear of getting caught.	T	F
10.	Most people will use somewhat unfair means to gain profit or an advantage, rather than to lose it.	T	F
11.	I commonly wonder what hidden reason another person may have for doing something nice for me.	T	F
12.	It makes me impatient to have people ask my advice or otherwise interrupt me when I am working on something important.	T	F
13.	I feel that I have often been punished without cause.	T	F
14.	I am against giving money to beggars.	T	F
15.	Some of my family have habits that bother and annoy me very much.	T	F
16.	My relatives are nearly all in sympathy with me.	T	F
17.	My way of doing things is apt to be misunderstood by others.	T	F
18.	I don’t blame anyone for trying to grab everything he can get in this world.	T	F
19.	No one cares much what happens to you.	T	F
20.	I can be friendly with people who do things which I consider wrong.	T	F
21.	It is safer to trust nobody.	T	F
22.	I do not blame a person for taking advantage of someone who lays himself open to it.	T	F
23.	I have often felt that strangers were looking at me critically.	T	F

24.	Most people make friends because friends are likely to be useful to them.	T	F
25.	I am sure I am being talked about.	T	F
26.	I am likely not to speak to people until they speak to me.	T	F
27.	Most people inwardly dislike putting themselves out to help other people.	T	F
28.	I tend to be on guard with people who are somewhat more friendly than I had expected.	T	F
29.	I have sometimes stayed away from another person because I feared saying or doing something that I might regret afterwards.	T	F
30.	People often disappoint me.	T	F
31.	I like to keep people guessing what I'm going to do next.	T	F
32.	I frequently ask people for advice.	T	F
33.	I am not easily angered.	T	F
34.	I have often met people who were supposed to be experts who were no better than I.	T	F
35.	I would certainly enjoy beating a crook at his own game.	T	F
36.	It makes me think of failure when I hear of the success of someone I know well.	T	F
37.	I have at times had to be rough with people who were rude or annoying.	T	F
38.	People generally demand more respect for their own rights than they are willing to allow for others.	T	F
39.	There are certain people whom I dislike so much that I am inwardly pleased when they are catching it for something they have done.	T	F
40.	I am often inclined to go out of my way to win a point with someone who has opposed me.	T	F
41.	I am quite often not in on the gossip and talk of the group I belong to.	T	F
42.	The man who had most to do with me when I was a child (such as my father, step-father, etc.) was very strict with me.	T	F
43.	I have often found people jealous of my good ideas just because they had not thought of them first.	T	F
44.	When a man is with a woman he is usually thinking about things related to her sex.	T	F
45.	I do not try to cover up my poor opinion or pity of a person so that he won't know how I feel.	T	F
46.	I have frequently worked under people who seem to have things so that they get credit for good work but are able to pass off mistakes onto those under them.	T	F
47.	I strongly defend my own opinions as a rule.	T	F
48.	People can pretty easily change me even though I thought that my mind was already made up on a subject.	T	F
49.	Sometimes I am sure that other people can tell what I am thinking.	T	F
50.	A large number of people are guilty of bad sexual conduct.	T	F

Appendix G

Informed Consent For Participants Of Investigative Projects

Title of the Project: A Neuropsychological Investigation of Sex Differences in Cardiovascular Reactivity to Verbal and Spatial Fluency Tasks: Testing a New Model of Sex Differences in Cardiovascular Regulation and Disease.
Investigator: Dane A. Higgins. Experiment Number: 00-432

I. THE PURPOSE OF EXPERIMENT, PHASE TWO

You are invited to participate in a study to obtain data on measures purportedly associated with verbal and spatial fluency, as well as cardiovascular measurements. This research attempts to determine the effects of sex differences on physiological and brain-related activity.

II. PROCEDURES TO BE FOLLOWED IN THE STUDY

To accomplish the goals of this study, you will be asked to complete one additional questionnaire. Following this, you will be asked to complete some verbal fluency tasks and spatial fluency tasks and to have cardiovascular measures taken before and after doing so.

III. DISCOMFORTS AND RISKS THROUGH PARTICIPATION IN THE STUDY

You may feel some embarrassment when answering the questionnaire. You may omit any questions that you feel are embarrassing. If you continue to have any problems associated with this study after you have left the experiment, please call Dr. David W. Harrison at 231-4422 so that he may assist you directly or direct you to other appropriate, available services.

IV. BENEFITS OF THIS PROJECT

Your participation in the project may help identify the functions of certain neuropsychological sex differences and the potential cardiovascular benefits of different sex-based neuropsychological mechanisms. However, no specific guarantee of benefits has been made to encourage you to participate.

V. ANONYMITY OF PARTICIPANTS AND CONFIDENTIALITY OF RESULTS

Identifying information will be kept strictly confidential. At no time will the researchers release your personal information from the study to anyone other than individuals associated with the project without your written consent. The information you provide will have your name removed and only a participant number will identify you during subsequent analyses and written reports of this research.

VI. EXTRA CREDIT COMPENSATION

For participation in this study, you will receive one point extra credit for Psychology 2004 or the psychology class for which you are attempting to earn one point of extra credit.

VII. FREEDOM TO WITHDRAW

You are free to withdraw from this study at any time without penalty. If you choose to withdraw, you will not be penalized by reduction in points or grade for Psychology 2004 or the psychology class for

which you are attempting to earn extra credit. There are alternative choices for receiving extra credit for the course evaluation.

VIII. USE OF RESEARCH DATA

The information from this research may be used for scientific or educational purposes. It may be presented at scientific meetings and/or published and reproduced in professional journals, books, or used for any other purpose that the Psychology Department at Virginia Tech considers in the proper interest of education, knowledge, or research.

IX. APPROVAL OF RESEARCH

This project has been approved by the Human Subjects Committee of the Department of Psychology and by the Institutional Review Board of Virginia Tech, and assigned the number 00-432.

X. PARTICIPANT'S RESPONSIBILITIES

I voluntarily agree to participate in this study. I agree to disclose if I have a history of any of the following medical conditions: history of a severe head injury, stroke, epilepsy, seizures, paralysis, neurological surgery, other neurological or nervous system problems, or other conditions previously listed on the medical history questionnaire.

XI. PARTICIPANT'S PERMISSION

I have read and understand the above description of the study. I have had an opportunity to ask questions and have had them all answered to my satisfaction. I hereby acknowledge the above and give my voluntary consent for participation in this study. I also understand that I may withdraw from participation at any time without penalty.

Participant's Signature **Student Number** **Date** **Telephone Number**

Should I have any questions regarding this research and its conduct, I should contact any of the persons named below:

Dane A. Higgins Primary Researcher	552-6065 dane@vt.edu
David W. Harrison, Ph.D. Faculty Advisor Chair, HSC	231-4422 dwh@vt.edu
David Moore, Ph.D. Chair, IRB	231-4991 moored@vt.edu

Appendix H

Controlled Oral Word Association Test (COWAT)

Administration

State:

"I am going to say a letter of the alphabet and I want you to write as quickly as you can all the words you can think of that begin with that letter. For instance, if I say the letter B, you might write best, bark, bank, battalion or words like that. Do not use words which are proper names such as Boston, Betty, Buick, or numbers, like billion. Also, do not use the same word with a different prefix or suffix, such as beat then beating, or the same word in a different tense, such as buy then bought. You will have one minute for each letter and I will notify you when one minute has passed. Following completion of the first letter I will ask you to write words with other letters. Any questions? Begin when I say the first letter. The first letter is F… go ahead."

Timing begins immediately and one minute is allowed for each letter. When one minute is up, say, "**Stop**." Wait about 5 seconds before beginning the next trial. Next, say, **"Begin when I say the next letter. The next letter is S… go ahead."**

Timing begins immediately and one minute is allowed for each letter. When one minute is up, say, "**Stop**." Wait about 5 seconds before beginning the next trial. Next, say, **"Begin when I say the next letter. The next letter is T… go ahead."**

Timing begins immediately and one minute is allowed for each letter. When one minute is up, say, "**Stop**." The score is the total number of acceptable words produced over the three minutes.

Appendix I

Ruff Figural Fluency Test (RFFT)

Administration

Begin with sample items of Part 1, saying:

> **"In front of you are three squares, each containing five dots. Note that the arrangement of the five dots is always the same. I want you to connect two or more dots by always using straight line. You do not need to connect all five dots. The purpose of the test is for you to make as many designs or figures as possible, but each design has to be different in some way from all the others. Any questions?"**

Following completion of the sample, give feedback about errors, i.e., if there are two identical designs, point out the duplicated designs and repeat the instruction set. If the sample designs are elaborate (e.g. all five dots are consistently connected) re-emphasize the instructions that a design can be drawn by connecting two or more dots. Following completion of the sample, state:

> **"Turn the page. Do not begin until instructed. On this page, draw as many different designs or figures as possible. Connect at least two dots with a straight line. You do not need to use all five dots. Work as quickly as possible and make every design different. Get ready, go."**

Allow one minute for the test. When this time is up, say, **"Stop."** Allow about 5 seconds and then instruct the subject:

> **"Turn the page and complete the practice trials."** (Allow about 20 seconds for this.) Then say, **"Turn the page, start in the upper left square, and work from left to right. Work as quickly as possible and make every design different. Get ready. Go."**

Allow one minute for Part 2. When this time is up, say, **"Stop."** Allow about 5 seconds and then instruct the subject:

> **"Turn the page and complete the practice trials."** (Allow about 20 seconds for this.) Then say, **"Turn the page, start in the upper left square, and work from left to right. Work as quickly as possible and make every design different. Get ready. Go."**

Allow one minute for Part 3. When this time is up, say, **"Stop."** The RFFT is finished.

Appendix J

Recording Form

Time Estimation Task: Estimation of 30 seconds: _____ seconds

Estimation of 180 seconds: _____ seconds

	SBP	**DBP**	**HR**
Baseline 1			
Baseline 2			
_____ Fluency 1			
_____ Fluency 2			
Baseline 3			
Baseline 4			
_____ Fluency 1			
_____ Fluency 2			

Appendix K

Please circle the most appropriate answer to each of the following questions:

1) How stressful was your participation in this experiment?

(1 = not stressful at all, 5 = moderately stressful, 9 = extremely stressful)

1 2 3 4 5 6 7 8 9

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2) How stressful was the verbal fluency task?

(1 = not stressful at all, 5 = moderately stressful, 9 = extremely stressful)

1 2 3 4 5 6 7 8 9

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3) How stressful was the figural fluency task?

(1 = not stressful at all, 5 = moderately stressful, 9 = extremely stressful)

1 2 3 4 5 6 7 8 9

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4) Overall, how anxious did you feel during this experiment?

(1 = not anxious at all, 5 = moderately anxious, 9 = extremely anxious)

1 2 3 4 5 6 7 8 9

Thank you for your time and participation in this study.

CURRICULUM VITA

Dane A. Higgins, Ph.D.

302 Koel Court, Slidell, LA 70461; (985) 649-1754; drdanehiggins@yahoo.com; webpage: www.neuro-psych.biz

Education

Degree:	**Doctor of Philosophy, Spring 2002**
University:	Virginia Polytechnic Institute and State University, Blacksburg, Virginia APA-Accredited Program
Major:	Psychology (Clinical Adult Specialization)
Emphasis:	Neuropsychological research, assessment, and treatment
Major Advisor:	David W. Harrison, Ph.D.
Dissertation:	"A Neuropsychological Investigation of Sex Differences in Cardiovascular Reactivity to Verbal and Spatial Fluency Tasks: Testing a New Model of Sex Differences in Cardiovascular Regulation and Disease"
Coursework:	(see Appendix A: List of Courses/Program of Study)

Degree:	**Master of Science, Fall 1999**
University:	Virginia Polytechnic Institute and State University, Blacksburg, Virginia
Major:	Psychology (Clinical Adult Specialization)
Emphasis:	Neuropsychological research, assessment, and treatment
Major Advisor:	David W. Harrison, Ph.D.
Master's Thesis:	"Cardiovascular Reactivity to Speech Processing and Cold Pressor Stress: Evidence for Sex Differences in Dynamic Functional Cerebral Laterality"

Degree:	**Bachelor of Science, Spring 1994**
University:	Arizona State University, Tempe, Arizona
Major:	Psychology (with distinction: cum laude)
G.P.A.:	3.78 (psychology); 3.65 (4 year cumulative)
Honors:	Arizona State University Honors College Graduate
Major Advisor:	George P. Knight, Ph.D.
Honors Thesis:	"Changing Gender Differences in Aggression?: Clarification Through Meta-Analysis"
Awards:	Regent's Scholarship Recipient: 1993-1994 Dean's Honor List: Spring 1994, Fall 1993, Spring 1993

Clinical Training

Clinical Psychology Intern

2001-present New Orleans Veterans Affairs Medical Center
1601 Perdido Street, COS-6, New Orleans, LA 70461
Neuropsychology Specialty
Supervisors: Dr. Jennifer Vasterling & Dr. John Mendoza (VAMC)
Dr. F. William Black (Tulane)
Rotations: Neuropsychology, Behavioral Medicine, General, and Research.
Duties include the completion of a general training program and a neuropsychology specialty experience through supervised inpatient and

outpatient assessment/consultation and therapy/intervention. Duties also include the use of a variety of assessment instruments and techniques (depending upon the needs of the case), comprehensive didactic training, in-service classes, and off-site seminars at Louisiana State University, Tulane University Health Sciences Center, Charity Hospital, and DePaul-Tulane Behavioral Health Center.

Graduate Clinician/Supervisor

1995-2001 Neuropsychological Assessment and Treatment Practicum Team
Psychological Services Center and Child Study Center
Virginia Polytechnic Institute and State University
3110 Price's Fork Road, Blacksburg, Virginia 24060
Supervisor: Dr. David W. Harrison

Graduate level practicum team specializing in the assessment and treatment of a variety of neuropsychological disorders resulting from cerebrovascular, congenital, convulsive, neoplastic, and traumatic disorders of the brain. Assessment procedures included syndrome analysis, standardized testing, and quantitative electroencephalography (QEEG) techniques. Provided individual and group supervision of graduate clinicians from Fall, 1999, to Spring, 2001.

Assessment Clinician/Supervisor

1997-1998, 1999-2000 Assessment Clinic Practicum Team
Psychological Services Center and Child Study Center
Virginia Polytechnic Institute and State University
3110 Price's Fork Road, Blacksburg, Virginia 24060
Supervisors: Dr. Thomas H. Ollendick; Dr. Lee Cooper (1999-2000 only)

Graduate level assessment practicum team specializing in the assessment and treatment of a variety of learning and psychological disorders in children and adults including: ADHD; adjustment disorders; anxiety; depression; math, reading, and language disorders; and others. Provided individual and group training in assessment techniques and supervision of graduate clinicians during second term of service (1999-2000).

Psychology Trainee

1997-1998 Clinical Externship
Carilion St. Albans Psychiatric Hospital
7516 Lee Highway, Radford, Virginia 24143
Supervisor: Dr. Rick Seidel, Licensed Clinical Psychologist

Completed rotations (480 hours) in three main psychiatric units of the hospital, the third being a locked (committed) unit. Each unit specialized in the treatment of patients at differing levels of need. Assessed and treated children, adolescents, and adults with anger management problems, chemical dependence, depression (with or without anxiety, mania, and suicidal ideation), PTSD, anxiety, and various other disorders with and without psychotic features. Assisted with and/or led the cognitive, community support, coping strategies, planning, and life skills group therapy sessions. Conducted family/couples sessions with patients and their primary support outside of the hospital. Organized follow-up care and at time of discharge.

Graduate Clinician/Supervisor

1997-1998, 1999-2000 Psychological Services Center and Child Study Center
Virginia Polytechnic Institute and State University

3110 Price's Fork Road, Blacksburg, Virginia 24060
Supervisors: Dr. Thomas H. Ollendick; Dr. Lee Cooper (1999-2000 only)

Responsible for the following: therapeutic duties, including the supervision of graduate clinician records; aiding in the conceptualization and organization of the PSC's library of Empirically-Supported Treatment Protocols; maintenance of the PSC's computer systems, Physiology and QEEG Laboratory, and other clinic equipment; design and administration of training sessions offered on the computer testing and scoring programs available at the PSC; and initiated design through implementation of a clinic web page.

Graduate Clinician

1995-1997 Clinical Practicum Team
Psychological Services Center and Child Study Center
Virginia Polytechnic Institute and State University
3110 Price's Fork Road, Blacksburg, Virginia 24060
Supervisors: Dr. Robert S. Stephens & Dr. Jack W. Finney (1995-1996)
Dr. George A. Clum (1996-1997)

Graduate level practicum team specializing in the assessment and treatment of a variety of psychological disorders including anxiety, assertiveness, depression, learning disorders, personality disorders, marital difficulties, and adjustment disorders. Individual, family, and couples therapy techniques used.

Teaching Experience

Graduate Teaching Assistant, Physiological Psychology

Spring, 1997 Department of Psychology
Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436
Supervisor: Bruce H. Friedman, Ph.D.

Teacher's Assistant for Physiological Psychology course with approximately 30 students. Duties involved assisting in the writing and administering of quizzes, grading essays, and providing individual assistance to students.

Graduate Teaching Assistant, Social Psychology

Spring, 1997 Department of Psychology
Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436
Supervisor: Danny K. Axsom, Ph.D.

Teacher's Assistant for Social Psychology course with approximately 30 students. Duties involved assisting in the writing and administering of quizzes, grading essays, and providing individual assistance to students.

Graduate Teaching Assistant, Laboratory Instructor for Introductory Psychology

1995-1996 Department of Psychology
Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436
Supervisor: David W. Harrison, Ph.D.

Taught two Introduction to Psychology laboratories, with 35 students per course, associated with an undergraduate introductory psychology class.

Duties involved teaching a laboratory section, writing and administering quizzes, grading essays, and providing individual assistance to students. Also, in-class demonstrations of psychological concepts were designed and implemented.

Relevant Work Experience

Neuropsychological Rehabilitation Specialist

1994-1995 Residential and Day Treatment Programs (training: 1,500 hours)
NeuroCare, Inc., Phoenix, Arizona
Supervisor: Dr. Jon Van Doren, Licensed Clinical Neuropsychologist

Responsible for patient rehabilitation at the Residential Treatment Center (inpatient) and the Day Treatment Center (outpatient). The Residential Program involved assisting patients with severe brain injuries to learn basic life skills, coping skills, stress management, relaxation skills, anger control, and behavior-management techniques. The Day Treatment Program focused on improving the orientation, language skills, attention, memory, processing speed, and other areas of cognitive difficulty in patients with mild brain injuries. Worked extensively with physical, occupational, speech, and vocational therapists in designing and implementing rehabilitation programs for child, adolescent, and adult patients.

Research

Dissertation Research

2000-2002 Title: A Neuropsychological Investigation of Sex Differences in Cardiovascular Reactivity to Verbal and Spatial Fluency Tasks: Testing a New Model of Sex Differences in Cardiovascular Regulation and Disease
Department of Psychology, Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436; Chairperson: David W. Harrison, Ph.D.

One hundred twenty-six right-handed undergraduate men and women underwent physiological measurements of SBP, DBP, and HR before and after verbal and figural fluency tasks, used as stressors. Dynamic and functional cerebral regulation of cardiovascular reactivity was assessed, specifically, the role that the frontal lobes have in regulating SBP, DBP, and HR in men and women. Sex differences in the functional cerebral regulation of these cardiovascular factors were predicted. Hostility was assessed in these participants, using the Cook-Medley Hostility Inventory (6 total groups of 21 participants each: high-, mid-, and low-hostile participants were identified). Sex and group (hostility) differences were predicted, as well as task (fluency type) differences. Comparisons were also made from a time estimation task (30 and 180 seconds), and the effect that women's menstrual cycle had on fluency. The MCSDS and the STAI were administered.

The principal findings of the current investigation were that the verbal fluency task raised SBP across sex and group, that both stressors raised SBP or DBP in different patterns (no sex differences were found), while stressors interacted with both sex and group. High-hostile men performed better on the first trial of the verbal fluency test compared to low-hostile men, while high-hostile women performed worse on the first trial of the verbal fluency test, compared to low-hostile women. Men perseverated more on each trial of the

verbal fluency test, while women perseverated less across trials. High-hostile men's time perception seems to be more rapid than low-hostile men, while for women it is the opposite. Women reported significantly more stress from the figural fluency task than men. Women in the luteal phase of menstruation did better on the verbal fluency test than women in the follicular phase of menstruation, and hostility and menstrual phase interact with verbal fluency.

This study encourages the consideration of neuropsychological sex differences in order to better understand cardiovascular regulation mechanisms and disease, leading to the development of improved prevention and behavioral management programs. Findings supporting this idea may bring about a new research focus, as some forms of cardiovascular disease may be more appropriately investigated as arising from neuropsychological problems.

Preliminary Exam Research

1999-2000 Title: Sex Differences in Cardiovascular Regulation and Disease: A Neuropsychological Model

Department of Psychology, Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436; Chairperson: David W. Harrison, Ph.D.

Conducted a comprehensive review and merged two bodies of literature that had not been previously integrated (the neuropsychology of sex differences and neuropsychological theories of cardiovascular regulation). Through this merging, a new, testable neuropsychological model of sex differences in cardiovascular regulation was presented to augment the current understanding of cardiovascular regulation, to improve prevention strategies, and to refine the treatment of some forms of cardiovascular disease. Furthermore, the proposed model represents a new, neuropsychological conceptualization of sex differences in cardiovascular regulation and disease, and is meant to aid in the reassessment or reinterpretation of previous research data, where applicable or useful, as possible implications of the model are considered.

Master's Thesis Research

1997-1999 Title: Cardiovascular Reactivity To Speech Processing and Cold Pressor Stress: Evidence For Sex Differences In Dynamic Functional Cerebral Laterality

Department of Psychology, Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436; Chairperson: David W. Harrison, Ph.D.

Investigated sex differences in dynamic functional cerebral laterality effects on cardiovascular reactivity and dichotic listening in response to a stressor (a cold pressor). Findings indicated a sex by focus interaction effect where men's, but not women's, systolic blood pressure increased significantly when focusing on sounds presented at the left ear during the dichotic listening task. Also, a compartmentalized, dynamic response in dichotic listening test performance was evidenced in both men and women (as both experienced increased accuracy at the right, but not left, ear), brought about as a function of the stressor. Men and women both evidenced increased cardiovascular reactivity, with men experiencing significantly more cardiovascular reactivity (SBP) than women in response to cold pressor pain. Women were also able to identify significantly more speech sounds presented to the left ear than men, and they were able to dynamically increase accuracy at the targeted ear identified within each focus group (left or right). Speech sounds processing (dichotic listening task) significantly decreased men's, but not women's, systolic blood pressure.

These results contribute to the literature on sex differences in functional cerebral laterality.

Professional Affiliations

International Neuropsychological Society (INS)- Graduate Student Associate Member
National Academy of Neuropsychology (NAN)- Graduate Student Affiliate
New Orleans Neuropsychological Society (NONS)- Graduate Student Member
American Psychological Association (APA)- Graduate Student Affiliate
Division 12, APA: Clinical Psychology- Graduate Student Affiliate
Division 40, APA: Clinical Neuropsychology- Graduate Student Affiliate
American Psychological Association of Graduate Students (APAGS) Member
American Psychological Society (APS)- Graduate Student Affiliate
Southeastern Psychological Association (SEPA)- Graduate Student Member
Undergraduate: Member of PSI CHI, National Honor Society in Psychology: 1993-1995

Current Research and Scholarly Interests

Cortical, subcortical, and autonomic correlates of emotion, behavior, cognition, and aging.
Sex differences in cardiovascular health as mediated by neuropsychological factors.
Methods in neuropsychology, psychophysiology, and psychopharmacology.
Hostility and other risk factors for cardiovascular disease, with associated functional cerebral system activation patterns and corresponding cardiovascular reactivity.

PUBLICATIONS

Refereed Articles

Harrison, D. W., Demaree, H. A., Higgins, D. A., & Williamson, J. (in press). Asymmetry in hand grip strength and fatigue in low- and high-hostile men. International Journal of Neuroscience.

Shenal, B., Moore, T., Rhodes, R., Higgins, D., & Harrison, D. W. (2001). Quantitative electroencephalography (QEEG) and neuropsychological syndrome analysis. Neuropsychology Review, 11(1), 31-44.

Knight, G. P., Fabes, R. A., & Higgins, D. A. (1996). Concerns about drawing causal inferences from meta-analyses: An example in the study of gender differences in aggression. Psychological Bulletin, 119(3), 410-421.

Manuscripts in Submission

Higgins, D. A., & Harrison, D. W. (under review). Sex differences in cardiovascular regulation and disease: A neuropsychological model. Manuscript under review with Psychological Bulletin.

Manuscripts in Preparation

Higgins, D. A., & Harrison, D. W. (in preparation). Cardiovascular reactivity to speech processing and cold pressor stress: Evidence for sex differences in dynamic functional cerebral laterality. Manuscript in preparation for publication.

Higgins, D. A., Everhart, D. E., & Harrison, D. W. (in preparation). Quantitative electroencephalography (QEEG) as an assessment tool for pseudoseizure diagnosis. Manuscript in preparation for publication.

Higgins, D. A., Williamson, J., Harrison, D. W. (in preparation). Sex differences in motor asymmetry. Manuscript in preparation for publication.

Higgins, D. A., Rhodes, R., Shenal, B., & Harrison, D. W. (in preparation). Quantitative electroencephalography (QEEG) techniques used to increase ADHD diagnostic accuracy. Manuscript in preparation for publication.

Higgins, D. A., & Harrison, D. W. (in preparation). QEEG imaging brain laterality of visualized positive and negative emotions. Manuscript in preparation for publication.

Higgins, D. A., & Harrison, D. W. (in preparation). Sex differences in diastolic blood pressure reactivity as a function of denial. Manuscript in preparation for publication.

Harrison, D. W., & Higgins, D. A. (in preparation). Sex Differences in Aging Effects on Frontal Lobe Decline. Manuscript in preparation for publication.

Higgins, D. A., & Harrison, D. W. (in preparation). QEEG as assessment tool for multi-sensory paresthesia diagnosis (Schizophrenia). Manuscript in preparation for publication.

Refereed Abstracts

Higgins, D. A., & Harrison, D.W. (in press). Sex Differences in Motor Asymmetry. Journal of the International Neuropsychological Society.

Higgins, D. A., Williamson, J. B., & Harrison, D. W. (in press). A Cross-Sectional Comparison of Frontal Lobe Development and Aging. Journal of the International Neuropsychological Society.

Higgins, D. A., Beck, A. L., & Harrison, D. W. (2001). Sex Differences in Aging Effects on Frontal Lobe Decline. Archives of Clinical Neuropsychology, 16(8), 728.

Higgins, D. A., & Harrison, D. W. (2001). Sex differences in diastolic blood pressure reactivity as a function of denial. Journal of the International Neuropsychological Society, 7(2), 256.

Higgins, D. A., Rhodes, R., Shenal, B., & Harrison, D. W. (2001). Quantitative electroencephalography as an assessment tool for ADHD diagnosis. Journal of the International Neuropsychological Society, 7(2), 160-161.

Higgins, D. A., & Harrison, D. W. (2001). QEEG imaging brain laterality of visualized positive and negative emotions. Journal of the International Neuropsychological Society, 7(2), 243.

Higgins, D. A., & Harrison, D. W. (2001). QEEG as assessment tool for multi-sensory paresthesia diagnosis (Schizophrenia). Journal of the International Neuropsychological Society, 7(2), 133.

Higgins, D. A., Demaree, H. A., Williamson, J., & Harrison, D. W. (2000). Hand grip strength and functional cerebral asymmetry differences in low- and high-hostile men. Journal of the International Neuropsychological Society, 6(2), 118.

Higgins, D. A., Everhart, D. E., & Harrison, D. W. (2000). QEEG as an assessment tool for pseudoseizure diagnosis. Journal of the International Neuropsychological Society, 6(2), 158.

Higgins, D. A., & Harrison, D. W. (1999). Sex differences in the functional cerebral laterality of cardiovascular reactivity to speech and a cold pressor. Archives of Clinical Neuropsychology, 14(8), 771.

Everhart, D. E., Higgins, D. A., & Harrison, D. W. (1997). Neuropsychological effects of anxiety without depression on facial affect perception. The Clinical Neuropsychologist, 11(3), 319-320.

Higgins, D. A. (1994). Changing gender differences in aggression?: Clarification through meta-analysis. Proceedings: Eighth National Conference on Undergraduate Research, 1, 441-445.

Conference Presentations and Posters

Higgins, D. A., & Harrison, D.W. (2002, February). Sex Differences in Motor Asymmetry. Poster session presented at the annual meeting of the International Neuropsychological Society, Toronto, Ontario, Canada.

Higgins, D. A., Williamson, J. B., & Harrison, D. W. (2002, February). A Cross-Sectional Comparison of Frontal Lobe Development and Aging. Poster session presented at the annual meeting of the International Neuropsychological Society, Toronto, Ontario, Canada.

Higgins, D. A., Beck, A. L., & Harrison, D. W. (2001, November). Sex Differences in Aging Effects on Frontal Lobe Decline. Poster session presented at the annual meeting of the National Academy of Neuropsychology, San Francisco, California.

Beck, A. L., Higgins, D. A., Williamson, J. B., Foster, P., & Harrison, D. W. (2001, March). Frontal Lobe Deterioration: Evidence from Sex Differences in Aging Effects. Poster session presented at the annual meeting of the Southeastern Psychological Association, Atlanta, Georgia.

Williamson, J., Higgins, D. A., Beck, A. L., & Harrison, D. W. (2001, March). A Cross-Sectional Comparison of Frontal Lobe Development and Aging. Poster session presented at the annual meeting of the Southeastern Psychological Association, Atlanta, Georgia.

Higgins, D. A., & Harrison, D. W. (2001, February). Sex Differences in Diastolic Blood Pressure Reactivity as a Function of Denial. Poster session presented at the annual meeting of the International Neuropsychological Society, Chicago, Illinois.

Higgins, D. A., Rhodes, R., Shenal, B., & Harrison, D. W. (2001, February). Quantitative Electroencephalography as an Assessment Tool for ADHD Diagnosis. Poster session presented at the annual meeting of the International Neuropsychological Society, Chicago, Illinois.

Higgins, D. A., & Harrison, D. W. (2001, February). QEEG Imaging Brain Laterality of Visualized Positive and Negative Emotions. Poster session presented at the annual meeting of the International Neuropsychological Society, Chicago, Illinois.

Higgins, D. A., & Harrison, D. W. (2001, February). QEEG as Assessment Tool for Multi-Sensory Paresthesia Diagnosis (Schizophrenia). Poster session presented at the annual meeting of the International Neuropsychological Society, Chicago, Illinois.

Higgins, D. A., Everhart, D. E., & Harrison, D. W. (2000, February). QEEG as an assessment tool for pseudoseizure diagnosis. Poster session presented at the annual meeting of the International Neuropsychological Society, Denver, Colorado.

Higgins, D. A., Demaree, H. A., Williamson, J. B., & Harrison, D. W. (2000, February). Hand grip strength and functional cerebral asymmetry differences in low- and high-hostile men. Poster session presented at the annual meeting of the International Neuropsychological Society, Denver, Colorado.

Higgins, D. A., & Harrison, D. W. (1999, November). Sex differences in the functional cerebral laterality of cardiovascular reactivity to speech and a cold pressor. Poster session presented at the annual meeting of the National Academy of Neuropsychology, San Antonio, TX.

Higgins, D. A., Everhart, D. E., & Harrison, D. W. (1999, March). Neurological disorders misdiagnosed as conversion disorders: QEEG utility for increased diagnostic accuracy. Poster session presented at the annual meeting of the Southeastern Psychological Association, Savannah, Georgia.

Williamson, J. B., Higgins, D. A., Demaree, H. A., & Harrison, D. W. (1999, March). Diminished asymmetry in hostile men: Motor performance. Poster session presented at the annual meeting of the Southeastern Psychological Association, Savannah, Georgia.

Higgins, D., Shenal, B., Moore, T., Rhodes, R., & Harrison, D. W. (1998, April). Quantitative electroencephalography (QEEG) facilitates neuropsychological syndrome analysis: An alternative to the nomothetic approach. Paper presented at the Spring meeting of the Virginia Psychological Association, Charlottesville, Virginia.

Everhart, D. E., Higgins, D. A., & Harrison, D. W. (1997, August). Neuropsychological effects of anxiety without depression on facial affect perception. Poster session presented at the annual meeting of the American Psychological Association, Chicago, Illinois.

Higgins, D. A. (1994, April). Changing gender differences in aggression?: Clarification through meta-analysis. Paper presented at the annual meeting of the National Conference on Undergraduate Research, Kalamazoo, Michigan.

Professional Conference Participation (I have attended and/or presented at the following)

Feb., 2002 International Neuropsychology Society Conference. Toronto, Ontario, Canada.
Nov., 2001. National Academy of Neuropsychology Conference. San Francisco, California.
Mar., 2001. Southeastern Psychological Association Conference. Atlanta, Georgia.
Feb., 2001. International Neuropsychology Society Conference. Chicago, Illinois.
Nov., 2000. National Academy of Neuropsychology Conference. Orlando, Florida.
Feb., 2000. International Neuropsychology Society Conference. Denver, Colorado.
Nov., 1999. National Academy of Neuropsychology Conference. San Antonio, Texas.
Mar., 1999. Southeastern Psychological Association Conference. Savannah, Georgia.
Nov., 1998. National Academy of Neuropsychology Conference. Washington, D.C.
Apr., 1998. Virginia Psychological Association Spring Convention. Charlottesville, Virginia.
Nov., 1997. National Academy of Neuropsychology Conference. Las Vegas, Nevada.
Aug., 1997. American Psychological Association Conference. Chicago, Illinois.
Feb., 1997. International Neuropsychological Society Conference. Orlando, Florida.
Oct., 1996. National Academy of Neuropsychology Conference. New Orleans, Louisiana.
Aug., 1996. American Psychological Association Conference. Toronto, Ontario, Canada.

Computer Skills

Proficient in: SAS; Microsoft Access, Excel, FrontPage, PowerPoint, Publisher, and Word; computer system design, troubleshooting, and repair; local area network (LAN) administration; web page authoring, graphic design, and maintenance; hardware/software configuration and analysis.

Personal

Born April 3, 1969, in Mesa, Arizona. Husband, and father of 6. Served two-year mission for the Church of Jesus Christ of Latter-Day Saints. Fluent in French (reading, writing, and speaking). Industrious, honest, and earnest. Eagle Scout.

References

F. William Black, Ph.D., Professor of Psychiatry and Neurology and Director of Neuropsychology Laboratory, Department of Psychiatry and Neurology at Tulane University Health Sciences Center, 1440 Canal Street, New Orleans, Louisiana 70112. black@tulane.edu

Helen J. Crawford, Ph.D., Professor of Psychology and Director of Laboratory of Psychological Sciences, Virginia Polytechnic Institute and State University, Department of Psychology, Blacksburg, Virginia 24061-0436. hjc@vt.edu

Jack W. Finney, Ph.D., Professor of Psychology and Chair of Department of Psychology, Virginia Polytechnic Institute and State University, Department of Psychology, Blacksburg, Virginia 24061-0436. finney@vt.edu

David W. Harrison, Ph.D., Associate Professor of Psychology and Director of Laboratory of Neuropsychology, Advisor and Mentor, Virginia Polytechnic Institute and State University, Department of Psychology, Blacksburg, Virginia 24061-0436. dwh@vt.edu

Thomas H. Ollendick, Ph.D., Professor of Psychology and Director of Child Study Center and Psychological Services Center, Virginia Polytechnic Institute and State University, Department of Psychology, Blacksburg, Virginia 24061-0436. tho@vt.edu

Jennifer Vasterling, Ph.D., Clinical Neuropsychologist, Mental Health Service Line, New Orleans Veterans Affairs Medical Center, 1601 Perdido Street, COS-6, New Orleans, LA 70461. jennifer.vasterling@med.va.gov

Richard A. Winett, Ph.D., Professor of Psychology and Director of Clinical Psychology, Director of Clinical Training, Virginia Polytechnic Institute and State University, Department of Psychology, Blacksburg, Virginia 24061-0436. rswinett@vt.edu

Appendix A

List of Graduate Courses (Program of Study):

Semester/Year	Course Title	Course #	Hours
Fall 1995	Assessing Human Intelligence	PSYC 5224	3
	Research Methods	PSYC 5315	3
	Statistics for Social Science Research I	STAT 5665	3
	Clinical Practicum	PSYC 5965	1
	Research & Thesis	PSYC 5994	2
Spring 1996	Behavioral Assessment & Treatment	PSYC 5244	3
	Personality Processes	PSYC 5274	3
	Statistics for Social Science Research II	STAT 5666	3
	Clinical Practicum	PSYC 5965	1
	Research & Thesis	PSYC 5994	2
Summer 1996	Emotional & Social Development- Children	FCD 5224	3
	Research & Thesis	PSYC 5994	3
Fall 1996	Developmental Psychology	PSYC 5534	3
	Clinical Neuropsychology	PSYC 6254	3
	Clinical Practicum	PSYC 5965	2
	Research & Thesis	PSYC 5994	1
Spring 1997	Intervention in Psychological Systems	PSYC 5254	3
	Biological Bases of Behavior	PSYC 5404	3
	Clinical Practicum	PSYC 5966	2
	Research & Thesis	PSYC 5994	4
Summer 1997	Research & Dissertation	PSYC 7994	5
Fall 1997	Psychopathology	PSYC 5284	3
	Psychophysiology	PSYC 5294	3
	Clinical Practicum	PSYC 6965	2
	Research & Dissertation	PSYC 7994	1
Spring 1998	Clinical Practicum	PSYC 6966	2
	Research & Dissertation	PSYC 7994	7
Fall 1998	General Neurochemistry	VMS 5324	Audit
	Clinical Practicum	PSYC 6965	2
	Research & Dissertation	PSYC 7994	7
Spring 1999	Neurochemical Regulation	BIOL 4554	3
	Clinical Practicum	PSYC 6966	2
	Research & Dissertation	PSYC 7994	5
Fall 1999	Clinical Practicum	PSYC 6965	2
	Research & Dissertation	PSYC 7994	7
Spring 2000	Child Psychopathology	PSYC 6264	3
	Research & Dissertation	PSYC 7994	6
Fall 2000	Clinical Practicum	PSYC 6965	1
	Indep. Study (Neuroanatomy and Imaging)	PSYC 5974	3
	Research & Dissertation	PSYC 7994	5
Spring 2001	Clinical Practicum	PSYC 6966	1
	Research & Dissertation	PSYC 7994	4
Fall 2001	Clinical Psychology Internship		
Spring 2002	Clinical Psychology Internship		