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Blood Pressure Increases during a Simulated Night Shift in Persons at Risk for  
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Running Head: Blood Pressure during a Simulated Night Shift

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## **Abstract**

**Background:** Shift work with sleep disruption is a systemic stressor that may possibly be associated with blood pressure dysregulation and hypertension. **Purpose:** We hypothesize that rotation to a simulated night shift with sleep deprivation will produce blood pressure elevations in persons at risk for development of hypertension. **Method:** We examined the effects of a simulated night shift on resting blood pressure in 51 diurnal young adults without current hypertension. Resting blood pressure was monitored throughout a 24 hour period of total sleep deprivation with sustained cognitive work. Twelve participants (23.5%) reported one or more parents with a diagnosis of hypertension. Ten participants were classified as prehypertensive by JNC-7 criteria. Only two prehypertensive subjects reported parental hypertension. **Results:** Results indicate that as the night shift progressed, participants with a positive family history of hypertension showed significantly higher resting diastolic blood pressure than those with a negative family history of hypertension ( $p=.007$ ). Prehypertensive participants showed elevated blood pressure throughout the study. **Conclusions:** These data suggest that rotation to a simulated night shift with sleep deprivation may contribute to blood pressure dysregulation in persons with a positive family history of hypertension.

**Key Words** Cardiovascular disease, Sympathetic nervous system, Family history of hypertension, Prehypertension, Shift work, Sleep deprivation

## **Introduction**

Shift rotations can disrupt sleep patterns and physiological circadian rhythms. These disruptions in normal daily biobehavioral oscillations may produce potentially negative effects on worker health, especially in individuals at risk for functional diseases such as essential hypertension. For example, sleep loss alone, whether from chronic stress, sleep disorder or work-related sleep restriction, is a provocative psychological, neuroendocrine and circulatory stressor. Reduced sleep duration is rapidly increasing in prevalence, especially in industrialized nations (1), in part because of economic and business globalization (2). Sleep loss has been implicated in a number of functional chronic disorders, including essential hypertension (3-5), obesity (6), diabetes (4), metabolic syndrome (2) and coronary heart disease (7). For example, in a longitudinal analysis of the U.S. National Health and Nutrition Examination Study (NHANES; n=4810) Gangwisch, et al. (4) found that persons between 32 and 59 years of age with 5 or fewer hours of sleep per night were twice as likely to have hypertension over an 8 to 10 year follow up period (hazard ratio, 2.10, 95% CI, 1.58-2.79). Statistical adjustment for obesity and diabetes reduced, but did not eliminate the increased risk. The authors concluded that the links between reduced sleep and hypertension were not completely mediated by obesity, diabetes or apnea, suggesting direct effects of sleep loss on blood pressure control mechanisms.

The potentially pathogenic effect of shift work with concomitant sleep disruption has been well documented. For example, multiple investigations have shown increased incidence of cardiovascular disease in shift workers (8,9). The relationship between shift work and cardiovascular disease is likely mediated via several pathways, including health damaging

behavioral and social factors. However, the neuroendocrine and circulatory consequences of disturbances in circadian rhythm and sleep quality may be especially pathogenic. Shift work adversely affects the circadian variation in blood pressure (10,11), and alternating shift work has been shown to be a significant, independent risk factor for elevated blood pressure (12). The precise role of shift work, circadian rhythm disturbance and sleep loss in blood pressure dysregulation and cardiovascular disease development requires further clarification.

The developmental etiology of hypertension is not fully understood, but the pathophysiological profile in the early stages of hypertension includes exaggerated responsivity of the sympathetic nervous system (13,14) and the hypothalamic pituitary adrenocortical (HPA) axis (15,16). Interestingly, this profile closely resembles the systemic effects of sleep deprivation, yet the precise relationship between sleep loss and hypertension development remains to be fully explored. Experimental studies of sleep deprivation and blood pressure control mechanisms have begun to clarify this relationship. Sleep deprivation can contribute directly to blood pressure dysregulation via increased sympathetic nervous system and adrenocortical excitation independent of sleep apnea. For example, experimentally-induced sleep deprivation increases blood pressure and inhibits neuronal nitric oxide synthase in nodose neurons in rats (17). The authors suggest that reduced expression of nitric oxide synthase in primary cardiovascular neurons could increase sympathetic nervous system tone and potentially increase risk for essential hypertension. REM sleep deprivation in rats produces blood pressure dysregulation similar to that of sino-aortic denervation (18). In humans, nights of insufficient sleep produce a rise in blood pressure, heart rate and excretion of norepinephrine on the following day (19).

A microneurographic study (20) indicates that 24 hours of total sleep deprivation elevates blood pressure through resetting of arterial baroreflexes. Moreover, persons with insomnia show reduced heart period and heart period variability, with increased low frequency and decreased high frequency power spectra, suggesting that sleep deprivation may increase sympathetic tone on the heart (21). Therefore sleep deprivation can produce untoward sympathetic nervous system changes that may explain, at least in part, the epidemiological association between shift work, sleep loss and essential hypertension.

We hypothesize that rotation to a simulated night shift will produce exaggerated blood pressure elevations in persons at risk for development of hypertension. Risk for hypertension is increased in persons with mildly elevated resting blood pressure (22) as well as in persons with a positive family history of hypertension. For example, offspring of hypertensive parents are two to four times more likely to develop high blood pressure than those without a parental history (23). Moreover, persons with a positive family history of hypertension show exaggerated neuroendocrine and circulatory responses during and in recovery from stress (24, 25). The theorized effect of chronic sleep loss on blood pressure can be modeled as shown in Figure 1. In this model, chronic sleep loss, whether a result of recurrent shift rotation, sleep disorder, or voluntary sleep restriction, can elevate sympathetic nervous system outflow (19,20), producing blood pressure increases. Persons with low risk for hypertension show modest sympathetic nervous system increases and benign blood pressure changes. In contrast, persons at enhanced risk for hypertension show increased sympathetic nervous system tone with exaggerated blood pressure elevations (14). Their pattern of chronic, elevated blood pressure could possibly play a role, ultimately, in development or progression of hypertension (13). If this causal pathway

is supported by studies of experimentally-induced sleep deprivation, then strategies to reduce chronic sleep loss may reduce development or progression of hypertension in individuals at enhanced risk.

The present study was designed to examine the effects of rotation to a prolonged, simulated night shift on resting blood pressure in normally diurnal young adults at enhanced risk for development of hypertension. We hypothesize that a simulated night shift with sleep deprivation will differentially increase blood pressure in persons at enhanced risk for development of hypertension.

## **Methods**

### Participants

Fifty one healthy volunteers (33 males and 18 females) completed this study. Average age of participants was 23 ( $\pm$  2.9) years. Volunteers completed screening questionnaires to determine their health status and absence of sleep disorders, their alcohol and tobacco use and their sleep habits. Subjects reporting a regular diurnal sleep/wake cycle were selected for participation. Actigraphic studies confirmed that all participants maintained their regular diurnal sleep/wake cycle for the three days immediately preceding the simulated night shift. All procedures were approved by the Clemson University institutional review board. Informed consent was provided by all participants prior to the study.

### Procedures

The sleep deprivation and sustained operations procedures have been described in detail by Pilcher and co-workers (26). Participants were recruited by flyers describing a two-day

sleep deprivation study. Screened participants met with research assistants three days prior to the study to review the consent form, the study design, and the instructions for the study. Participants were asked to sleep approximately eight hours each night for the three days immediately prior to the beginning of the study. They were requested to not drink alcohol the day before the beginning of the study and to not consume any caffeine or eat substances high in sugar (e.g., a candy bar) the morning before arriving for the study.

After screening, participants were given an actiwatch (Mini Mitter Company Inc., Bend, OR) and a sleep log to provide objective and subjective baseline data on sleep habits prior to the onset of the study. The participants were instructed to wear the actiwatch for three days leading up to the night shift simulation, except when showering or swimming.

Actiwatches were worn on the non-dominant arm and recorded wrist accelerations (movement) for assessment of sleep/wake activity cycles. The participants were instructed to complete the sleep log each morning upon awakening for each of the three days prior to the study. The sleep logs contained information about time going to bed, time getting out of bed, napping during the day, and sleep quality.

Participants were assigned in groups of four to five to complete the study. Participants were called between 8:30 and 9:00 am on Day 1 of the laboratory study to ensure they were awake before reporting to the laboratory at 9:30 am on Day 1 of the study. All food, non-caffeinated drinks and water, were provided for the participants throughout the study.

Participants completed a series of surveys and cognitive tasks with scheduled breaks and meals throughout the 28 hour study period. Cognitive tasks included Graduate Record Exams, the logical reasoning component of the Law School Admissions Test, an audio

vigilance task, and memory tasks (26). The study concluded at approximately 1:30 pm on Day 2 after which the participants were transported back to their residences and reminded to sleep before driving or operating heavy equipment.

### Blood Pressure Measures

Resting blood pressure (BP) was measured upon arrival and departure, and at approximately 8:30 PM, 1:00 AM, 5:30 AM and 10:00 AM over the study period. The arrival and departure data was not included in the formal analyses to avoid effects of the novel environment (arrival) and the anticipation of departure. Blood pressure was obtained using GE Dinamap Pro100 machines (Medical Solutions, Minneapolis, MN.). Dinamap performance was verified on a regular basis for zero offset, integral offset and gain using a mercury manometer. All devices performed within manufacturer tolerances. Research assistants were trained on theory and application of blood pressure determination using both auscultatory and oscillometric techniques, including use of appropriate cuff sizes, and other American Heart Associate guidelines for blood pressure determination (27). At each blood pressure determination, participants sat quietly in a comfortable armchair for five minutes prior to taking five BP readings at one-minute intervals. The last three readings were averaged to create a single, stable resting BP index at each time period.

### Classifications of Subjects by Risk for Hypertension

Risk for subsequent development of hypertension was determined in two different ways, by reported parental history of hypertension and by resting blood pressure levels. At the beginning of the study, participants completed a comprehensive personal and family medical history questionnaire. Participants were classified as positive family history of

hypertension if one or both biological parents were identified as having been diagnosed with essential hypertension by a physician. Participants were classified as negative family history if there was no parental hypertension known to the participant. Validity of self reported parental hypertension has been consistently demonstrated through direct contact with parents and parents' physicians (28-30).

An alternative classification of risk by resting blood pressure level was based on criteria outlined in the Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-7, 22). Briefly, subjects rested in a seated position while two blood pressure determinations were made on two different days at approximately the same time each day. The blood pressure measurement procedure was consistent with American Heart Association guidelines. Classifications were based on the average of two readings each day. Participants were classified as normal if they had no blood pressure above 120 mmHg systolic and 80 mmHg diastolic on both days of measurement. Participants were classified as prehypertensive if they had blood pressure between 120-139 systolic or 80-89 diastolic on both days of measurement. No participants were classified above the prehypertensive range.

#### Data Analyses

All BP data were entered into Microsoft Excel and imported into SPSS (SPSS, Inc., Chicago, IL) for statistical analyses. Systolic and diastolic blood pressures were initially analyzed in a 2 X 2 X 4 (Risk X Sex X Time) design using the SPSS General Linear Model with Time as the within subjects variable and multivariate F tests for main effects and interactions with Time. When no main effects or interactions were observed with Sex,

follow up analyses were conducted using similar 2 X 4 (Risk X Time) analyses. One set of analyses were conducted using family history of hypertension (positive versus negative family history) for the Risk variable. Additional analyses were conducted using JNC-7 grouping of blood pressure (prehypertensive versus normal) as an alternate Risk variable.

## **Results**

Using family history of hypertension as an index of risk, 23.5% of participants (12 of 51) reported a positive family history of essential hypertension in at least one biological parent. Average age was 23.0 years for the positive family history group and 22.7 years for the negative family history group. There were no significant group differences in age or body mass index ( $p > .05$ ). Using the JNC-7 criteria for classification of blood pressure, 19.6% of participants (10 of 51) were classified as prehypertensive, with the remaining participants classified as normal. Average age was 21.9 years for the prehypertensive group and 23.0 years for the normal group. There were no significant differences between groups in age or body mass index ( $p > .05$ ). Only 3.9% of participants (2 of 51) had both a prehypertensive classification and a positive family history of hypertension.

### Family History of Hypertension

The results using family history of hypertension for risk classification pressure are shown for resting systolic (see Figure 2a) and diastolic (see Figure 2b) pressure. There were no significant baseline daytime differences in blood pressure between family history groups. Moreover, there were no significant main effects or interactions for Sex and no significant main effects for Family History on either systolic or diastolic blood pressure. However, repeated measures results showed a significant Family History X Time interaction for

diastolic blood pressure [multivariate  $F(3,46)=4.574$ ,  $p=.007$ ,  $\eta^2= .230$ ]. Diastolic blood pressure for the two family history groups significantly diverged across the night of sleep deprivation, with slight decreases across the night in subjects with negative family history, and concomitant increases in subjects with positive family history of hypertension. For example, from the 1:00am test period to 10:00am the following morning, persons with negative family history showed a decrease in diastolic pressure of approximately 2 mmHG, but persons with positive family history of hypertension showed a corresponding increase of approximately 3.5 mmHg (see Table I).

#### JNC-7 Classification of Prehypertension

The prehypertensive group had significantly higher systolic pressure than the normal group throughout the simulated night shift [ $F(1,46) = 20.839$ ,  $p<.001$ ,  $\eta^2= .312$ ]. There was no significant effect for Time, or Prehypertension X Time interaction. A significant Prehypertension X Sex interaction [ $F(1,46)=5.369$ ,  $p=.025$ ,  $\eta^2 = .105$ ] indicated that females had lower systolic blood pressure than males in the normal blood pressure group (see Table II.). The prehypertensive group also had significantly higher diastolic pressure than the normal group throughout the simulated night shift [ $F(1,48) = 4.638$ ,  $p=.036$ ,  $\eta^2 = .088$ ]. Neither main effects for Time nor the Prehypertension X Time interaction were significant for diastolic pressure. There were no main effects for Sex on diastolic pressure, nor were there any significant interactions of Sex with either Time or Prehypertension.

#### **Discussion**

The present findings suggest that rotation to a simulated night shift with sleep deprivation can produce blood pressure elevations that are especially pronounced in some persons with

increased risk for hypertension (see Figure 2). Work-related chronic sleep loss is a growing international problem, but the potential negative health consequences have not been fully characterized. Globalization of business often requires continuous operations around the clock. Individuals working under 24-hour work conditions are more likely to experience a wide range of health-related effects including cardiovascular disorders (31-33, 10), psychosocial stress (34-37), and disturbed sleep (38-41).

### Interactions with Family History of Hypertension

The present results show that the effect of family history of hypertension on resting diastolic blood pressure significantly increased across a simulated night shift rotation with sleep deprivation (see Figure 2b). Post hoc cell-wise comparisons were not significant, but the significant Family History X Time interaction indicates increasingly higher blood pressures in the positive family history group, relative to the negative family history group across time. The present findings differed slightly between participants with parental hypertension and those classified as prehypertensive. For example, the prehypertensive group showed significant main effects on blood pressure with no significant interactions over time. In contrast, diastolic pressure of the positive family history group was initially similar to that of the negative family history group, but their pressures gradually separated as the simulated night shift progressed. It is not surprising that groups selected on JNC-7 blood pressure criteria would show consistently elevated pressures throughout the simulated nightshift. The interaction of JNC-7 classification with sex merits further investigation in future studies. Interestingly, the family history classification did not affect resting pressure except in the latter stages of the shift where sleep deprivation was most pronounced.

## Disruption of the Circadian Variation in Blood Pressure

Although the current study did not formally analyze circadian rhythms of blood pressure, results provide some possible insight into the potentially pathogenic effects of shift work, especially in persons at enhanced risk for development of hypertension. Nighttime blood pressure dipping is an important part of the circadian rhythm in diurnal humans.

Acclimated nocturnal shift workers show blood pressure dipping during their daytime sleep periods (42,10) indicating the likely relationship of blood pressure dipping to cycles of sleep, activity, and perhaps also posture. In contrast, the present study simulated a swing from regular day shifts to a night shift. All participants in the current study reported regular diurnal sleep/wake cycles prior to the study, and this was confirmed by three day actigraphic monitoring. Kitamura, et al. (42) observed disruption of the normal circadian blood pressure pattern on the first night of rotation to a night shift. Consistent with Kitamura (42), the present study found no evidence for reduced nocturnal blood pressure. We suggest that future studies more systematically assess disruption of circadian rhythms in shift workers at risk for hypertension. For example, attenuation of nocturnal blood pressure dipping is more common in several groups at enhanced risk for hypertension and coronary heart disease, including individuals with lower socioeconomic status (43), African Americans (10) and persons with a family history of hypertension (44). This is consistent with the possibility that reduced nocturnal blood pressure dipping in groups at risk for cardiovascular disease may result, at least in part, from sleep loss, sleep disruption, or reduced sleep quality, and may be especially pronounced within a shift rotation context.

## Methodological Limitations

The present study has several limitations that should be considered in interpretation of results. We did not study changes with normal sleep patterns during a dayshift in our sustained operations laboratory. This was beyond the scope of the present study, and furthermore would have required at least two additional overnight studies, one for sleep acclimation in the laboratory, and the second for a day shift with in-laboratory nighttime sleep patterns. The resulting cost and subject burden would be prohibitive. Thus our data are meaningful in comparison with existing normative data on blood pressure during normal work shifts and sleep patterns.

All of our participants performed cognitive work with periodic breaks throughout the simulated shift, so the effect of cognitive activity between resting blood pressure measurement periods may have had some effect on nighttime blood pressures. Using the current design, it is difficult to separate the effects of sleep deprivation alone, prolonged work alone, or the combination of work and sleep loss common in shift rotations.

Notwithstanding these limitations, we believe that reduced sleep during rotation to a night shift is associated with disturbance of normal blood pressure control mechanisms with potential health consequences, especially in persons at enhanced familial risk for development of hypertension.

Diastolic blood pressure differences between positive and negative family history groups across a night shift rotation with sleep deprivation are modest, yet statistically significant.

We cannot establish the effects of recurrent shift rotations on blood pressure with the current methodology. However, if our observed acute blood pressure effects reliably mimic chronic circulatory effects of recurrent shift rotations, then these findings would

further suggest potentially pathogenic effects of shift rotations with sleep loss in persons with familial hypertension. Regardless of limitations, the current findings are consistent with the cardiovascular epidemiology of shift work (8-12, 42), and suggest that shift rotation with sleep loss may negatively affect blood pressure control mechanisms in persons at risk for later hypertension development. Moreover, these data show, in an experimental context independent of the normal psychosocial and socioeconomic factors associated with real world shift work assignments, that rotation to a single night shift can significantly elevate blood pressure in persons with a positive family history of hypertension.

#### Summary and Conclusions

Based on the current findings, we suggest that rotation to a night shift with concomitant sleep loss may induce or contribute to blood pressure dysregulation in persons with a positive family history of hypertension. If repeated on a regular basis, this pattern of sleep loss, circadian rhythm disruption and blood pressure elevation could contribute to development and/or progression of hypertension, especially in persons with familial hypertension.

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Table I. Means and standard errors for systolic and diastolic blood pressure (mmHg) for positive and negative family history groups by time of testing. The Family History X Time interaction multivariate  $F(3,46) = 4.574$ ,  $p=.007$ ,  $\eta^2 = .230$ .

	Time	SBP	SBP	SBP	SBP
		8:30pm	1:00am	5:30am	10:00am
<b>Negative Family History</b>		113.8 (1.92) n=39	113.7 (1.97) n=38	111.2 (1.98) n=39	111.9 (1.82) n=39
	<b>Positive Family History</b>	110.4 (3.04) n=12	109.2 (3.97) n=12	112.3 (3.66) n=12	112.2 (3.28) n=12

	Time	DBP	DBP	DBP	DBP
		8:30pm	1:00am	5:30am	10:00am
<b>Negative Family History</b>		66.1 (1.36) n=39	67.5 (1.16) n=38	65.9 (1.29) n=39	65.5 (1.04) n=39
	<b>Positive Family History</b>	67.1 (1.99) n=12	67.5 (2.25) n=12	68.7 (3.10) n=12	69.8 (2.37) n=12

Table II. Systolic blood pressures (+/- standard error) for normal and prehypertensive (JNC-7 classification) males and females [interaction  $F(1,46)=5.369$ ,  $p=.025$ ,  $\eta^2 = .105$ ].

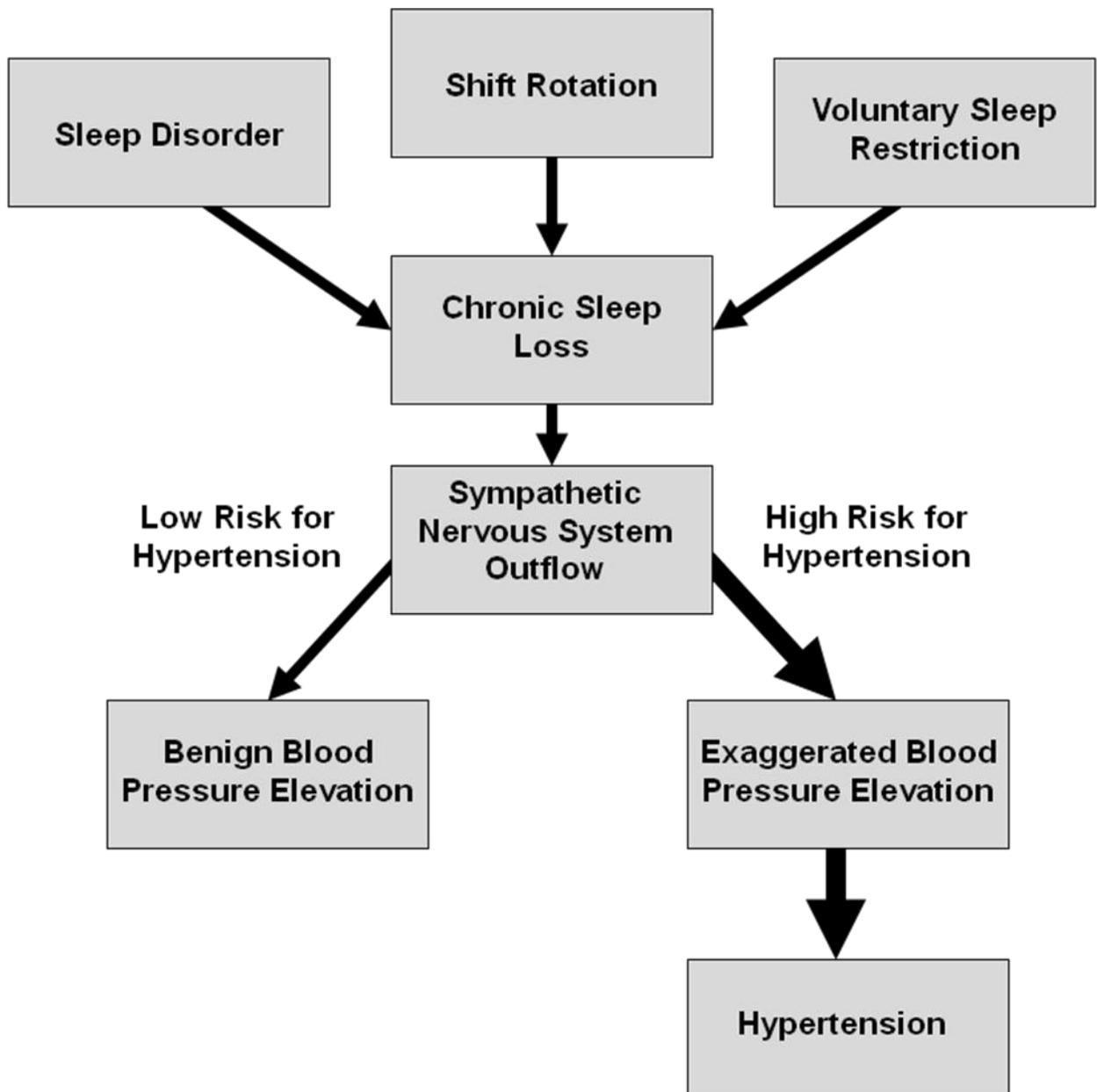
	<b>Normal</b>	<b>Prehypertension</b>
<b>Male</b>	114 (1.65) n=25	122.4 (2.92) n=8
<b>Female</b>	101.9* (2.13) n=15	126.2 (5.85) n=2

\*  $p<.001$  versus Normal Male

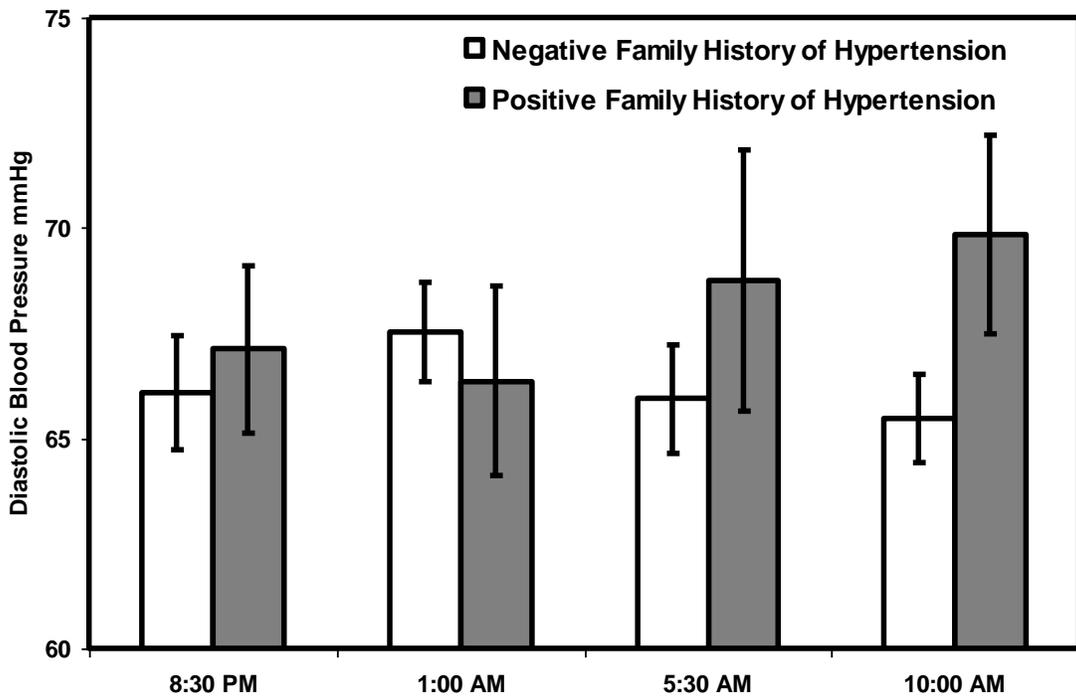
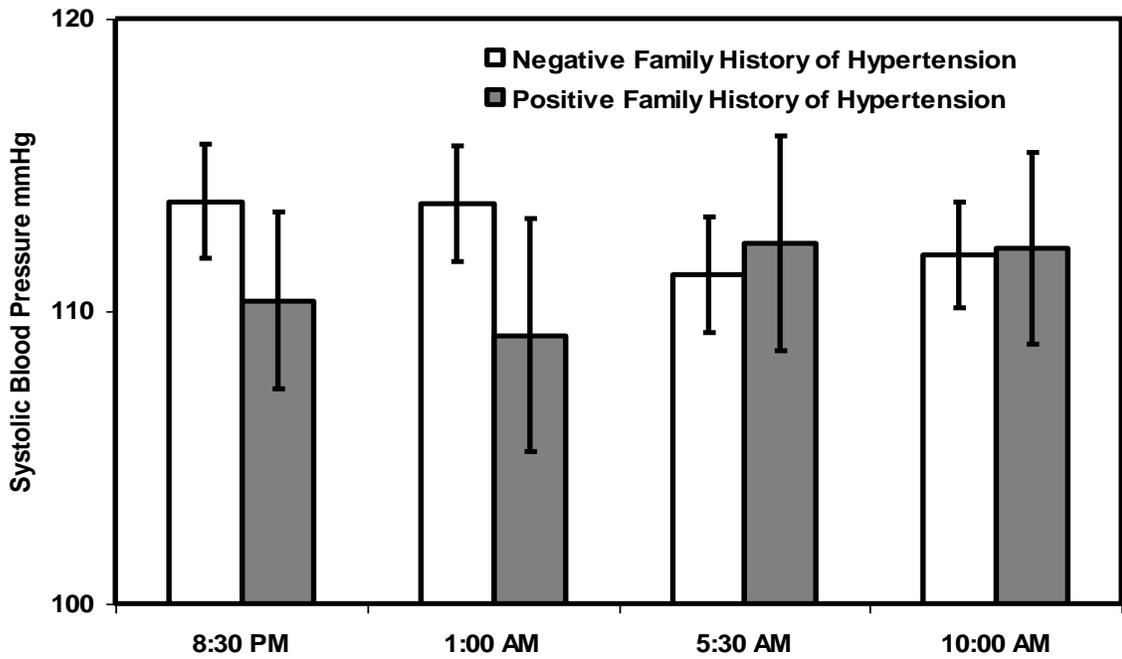
### Figure Captions

Figure 1. Causal model of effects of sleep deprivation on blood pressure control and progression of hypertension in persons at risk.

Figure 2. Means and standard errors for resting systolic (2a) and diastolic (2b) blood pressure throughout a night of sleep deprivation in groups with positive versus negative family history of hypertension. The Family History X Time interaction multivariate  $F(3,46)=4.574$ ,  $p=.007$ ,  $\eta^2 = .230$ .



**Figure 1**



Figures 2a and b