Personality Type and Neural Circulatory Control

Kathleen E. Schroeder, Krzysztof Narkiewicz, Masahiko Kato, Catherine Pesek, Bradley Phillips, Diane Davison, Virend K. Somers

Abstract—Psychosocial factors, including type A personality, anger, hostility, and anxiety, have been implicated in the pathogenesis of cardiovascular disease. Abnormal sympathetic responses to stress may help explain the link between certain behavior patterns and cardiovascular disease. We tested the hypothesis that in normal humans, type A personality characteristics are associated with exaggerated heart rate, pressor, and sympathetic nerve responses to mental and physical stress. We measured heart rate, blood pressure, and muscle sympathetic nerve activity (obtained with direct intraneural recordings) at rest and during stress in 45 healthy subjects (19 men and 26 women, age 29.2±8.7 years) who had no chronic diseases and were taking no medications. Subjects were divided into tertiles based on type A scores. There were no significant differences in sympathetic or hemodynamic reactivity among the 3 different intensity levels of type A characteristics. Baseline measures and responses to stress tests were similar across the 3 groups. Sympathetic and hemodynamic changes during stress tests were also similar in subject groups stratified according to anger scale and cynicism scale. Sympathetic nerve and hemodynamic measurements at rest and during stress were not different in normal subjects with type A characteristics. Abnormalities in sympathetic or cardiovascular reactivity are unlikely to be implicated in any excess of cardiovascular disease in people with type A personality characteristics. (Hypertension. 2000;36:830-833.)

Key Words: blood pressure ■ heart rate ■ nervous system, autonomic ■ personality ■ behavior

Type A personality behavior has been associated with an increased risk of cardiovascular disease, including hypertension, myocardial infarction, and coronary heart disease. Recent data have also linked type A behavior to left ventricular hypertrophy, an independent risk factor for cardiovascular complications and death. The type A person may respond to stress with hostility or aggression, feel a sense of time pressure, and be competitive and ambitious. Previous studies suggest that type A behavior, anger level, and cynicism level increase the risk of cardiovascular disease. This increased risk may be due to a greater cardiovascular reactivity to stress in the type A personality, evidenced by exaggerated sympathetic and hemodynamic responses.

The sympathetic nervous system is an important contributor to cardiovascular stress responses and may also be implicated in the development of cardiovascular disease. Increased activity of the sympathetic nervous system has been documented in patients with heart failure and hypertension. Experimental and clinical evidence points to a central neural origin of the heightened sympathetic drive. Cardiovascular adrenergic accompaniments of behavioral states, particularly anger, may be implicated in stress-induced cardiac ischemia and arrhythmias. Activation of the sympathetic nervous system, leading to tachycardia, increased blood pressure, and impaired myocardial perfusion, may be an important link between stress, affective state, and cardiovascular events.

Increased sympathetic activity at baseline and during stress is an attractive potential mechanism that may link personality characteristics to cardiovascular disease. We therefore tested the hypothesis that sympathetic activation, blood pressure, and heart rate at rest and during stress are related to type A behavior and levels of anger and cynicism in normal humans.

Methods

We studied 45 normotensive subjects (26 women and 19 men, mean±SD age 29.2±8.7 years). Mean body mass index was 24.2±3.5 kg/m². All subjects were healthy, and none were receiving any medication. The study was approved by the University of Iowa Institutional Human Subjects Review Committee, and written informed consent was obtained from each subject.

Blood pressures were measured every minute with an automatic sphygmomanometer (Lifestat 200; Physio-Control). ECG and respiration (Biottach and Pneumotrace; Gould Electronics) were recorded continuously. Intraneural measurements of postganglionic muscle sympathetic nerve activity (MSNA) were obtained from microneurographic recordings of the peroneal nerve, posterior to the fibular head. The neural signals were amplified, filtered, rectified, and integrated to obtain a mean voltage display of sympathetic nerve activity. We were unable to obtain stable nerve recordings in 3 subjects.

Received March 8, 2000; first decision April 24, 2000; revision accepted May 8, 2000.

From the Department of Internal Medicine (K.S., D.D., V.K.S.), Mayo Clinic, Rochester, Minn; Medical University of Gdansk, Gdansk, Poland (K.N.); Tottori University, Tottori, Japan (M.K.); and Department of Internal Medicine (C.P.) and the Division of Clinical and Administrative Pharmacy (B.P.), University of Iowa (Iowa City).

Correspondence to Virend K. Somers, MD, PhD, Department of Internal Medicine, Mayo Clinic, 200 First Street Southwest, Rochester, MN 55905. E-mail somers.virend@mayo.edu

© 2000 American Heart Association, Inc.

Hypertension is available at http://www.hypertensionaha.org
Subjects were studied in the supine position on a comfortable bed. Baseline measurements of heart rate (ECG), blood pressure, respiration, and MSNA were obtained during 10 minutes under carefully standardized conditions. Each subject then experienced 3 laboratory stressors: mental stress (oral testing of serial subtraction), sustained isometric handgrip (at one third of maximal voluntary contraction) with posthandgrip muscle ischemia (with inflation of a pneumatic cuff on the upper arm to suprasystolic pressure levels just before the end of contraction), and a cold pressor test (immersion of the hand in ice). The order of the first 2 stressors was randomized; the cold pressor test was performed last, due to the sustained effects of the stress. Each stressor event lasted 2 minutes, with a 15-minute rest period between events. At least 20 minutes separated the end of 1 test from the beginning of the next.

The self-report, Revised Version of the Minnesota Multiphasic Personality Inventory (MMPI-2),\(^5\) was given to each subject on completion of the laboratory protocol. This questionnaire includes 3 components: type A, anger, and cynicism. The MMPI-2 type A scale consists of 19 items that require true or false responses to questions about time urgency, competitiveness, and hostile attitudes. High scorers on the type A scale are described as hard-driving, fast-moving, and work-oriented individuals who frequently become impatient, irritable, and annoyed. The MMPI-2 anger scale includes 16 questions about expression or control of anger. High scores on the scale suggest anger-control problems.\(^16\) The cynicism scale consists of 23 items. Subjects who score high on this scale expect hidden, negative motives behind the acts of others and are likely to hold negative attitudes toward those close to them.\(^16\)

Responses to type A, anger, and cynicism scales were categorized by score into 3 groups (low, moderate, and high) according to the distribution of scores in this population.

All data analysis was completed by a single investigator (K.E.S.), who was blinded to subject and personality type. Sympathetic bursts were identified through inspection of the voltage neurogram. The amplitude of each burst was determined and sympathetic activity was calculated as bursts/min multiplied by mean burst amplitude and expressed as U/min. Measurement of nerve activity at baseline before each intervention was expressed as 100%. Changes in integrated MSNA allowed an evaluation of intrasubject changes in sympathetic activity during the same recording session. Sympathetic activity was also expressed as bursts per minute and bursts per 100 heartbeats, which allowed a comparison of sympathetic discharge between individuals at baseline.

Comparisons between groups were made by ANOVA, followed by Sheffe’s test for multiple comparisons. Results are expressed as mean±SD. A value of \(P<0.05\) was considered statistically significant.

**Results**

The average type A score was 6.0±0.4 (range 1 to 12), the average anger score was 4.2±0.3 (range 1 to 8), and the average cynicism score was 5.8±0.6 (range 0 to 16). The type A scale correlated with both the anger score \((r=0.47; P=0.003)\) and the cynicism score \((r=0.46; P=0.005)\). The correlation between the anger and cynicism scores was not significant \((r=0.33; P=0.075)\).

There were no significant differences or trends that related increased sympathetic or hemodynamic activity to more intense behavioral characteristics, either at rest or during the different stressors. Resting MSNA was similar across all tertiles whether measured as bursts per minute (Figure 1) or bursts per 100 heartbeats \(29±4, 31±4, \text{and} 25±4, \text{respectively;} \: P=0.5)\). Blood pressure, heart rate, and MSNA increased significantly in response to mental stress, handgrip, and cold pressor test, but this increase was not influenced by personality type.

**Discussion**

Psychological characteristics have been proposed as possible mechanisms that contribute to sympathetic activation and increased blood pressure in patients with essential hypertension.\(^17,18\) Behavioral responses are frequently accompanied by sympathetic activation. These behavioral responses are regulated by central control mechanisms, which are linked closely to brain stem centers that modulate autonomic outflow.\(^19\) Indeed, in patients with hypertension, increased noradrenaline release from subcortical brain regions is associated with increased peripheral sympathetic activity.\(^5\) Thus, there is precedent for supposing that behavioral characteristics may be linked to the sympathetic-hemodynamic profile at rest and particularly during stress.

This is the first study to assess directly the possible relationship between personality type and sympathetic re-
responses to stress. Our data show no link between measures of type A behavior, anger, and cynicism and sympathetic and hemodynamic reactivity. Sympathetic mechanisms are therefore unlikely to contribute to the well-documented association between cardiovascular complications and type A behavior. Our data also show that type A characteristics are correlated significantly with both anger and cynicism. Anger and cynicism, however, are not related. This suggests that these scores are not substantially influenced by each other.

Several prior studies have examined the association between type A personality (with its related hostility and anger) and cardiovascular disease. Some have shown a direct relationship between the 2, 20–22 and others have shown no interaction. 23,24 These studies have been limited by several concerns, including nonblinded analysis of pressor data, lack of control groups, indirect (or absent) estimates of sympathetic activity, and intermittent measures of hemodynamic data.

Important and unique strengths of the present study include, first, that data analysis was completed with the investigator blinded to personality type. Second, sympathetic nerve activity was recorded directly, via microneurography. Third, we studied normal subjects over a narrow range of age and body mass index, which minimized the influence of these variables on the responses we report.

It is unlikely that our negative findings can be explained by the relatively young age of our subject population. Previous studies have shown that sympathetic neural reactivity to stress does not increase with age in healthy humans. 25 However, it is possible that later in life type A personality characteristics may more significantly influence autonomic and vascular responses to stress, particularly in older subjects with underlying cardiovascular pathology. An additional consideration is that the classification of personality type is based on attitudes and responses to the stressors more closely linked to mental than to physical stress. In the present study, responses to only 1 mental stressor (serial subtraction) were examined. It is possible that if responses to several mental stress protocols were tested, a link between personality classification and mental stress response may have become evident.

In conclusion, our novel finding is that sympathetic and hemodynamic measures at rest and during stress are not influenced by personality type. Consequently, the link between personality type and cardiovascular risk is unlikely to be mediated via personality type-dependent potentiation of sympathetic and hemodynamic responses to mental and physical stressors. These findings do not speak against the compelling link between emotional stress and anger and cardiovascular events. 12–14 Nor do they exclude the possibility of a connection between personality type and cardiovascular disease. Rather, they suggest that any such connection is unlikely to be mediated via enhanced responsiveness of the sympathetic nervous system.

Acknowledgments

Dr Somers is an Established Investigator of the American Heart Association and is also supported by NIH grants HL-61560 and HL-60618 and M01-RR00585. This work was carried out with the support of NIH Fogarty Fellowship TW-05399 (Dr Narkiewicz), American Physiological Society Perkins Memorial Awards (Drs Narkiewicz and Kato), NIH grant HL-07121 (Dr Pesek), and an NIH Sleep Academic Award and NIH grant HL-14388 (Drs Phillips and Somers).

References


Personality Type and Neural Circulatory Control
Kathleen E. Schroeder, Krzysztof Narkiewicz, Masahiko Kato, Catherine Pesek, Bradley Phillips, Diane Davison and Virend K. Somers

Hypertension. 2000;36:830-833
doi: 10.1161/01.HYP.36.5.830

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2000 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://hyper.ahajournals.org/content/36/5/830

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Hypertension can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Hypertension is online at:
http://hyper.ahajournals.org//subscriptions/