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Hemodynamics of Seasonal Adaptation

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To investigate the possibility that seasonal adaptation requires significant hemodynamic changes, 5 normotensive and 21 mildly hypertensive subjects were followed through 4 seasons for changes in systemic hemodynamics and sympathetic nervous activity. In the upright position wintertime blood pressures increased by 3% (P = NS) over summer values whereas cardiac output and stroke volume decreased by 18% and 21%, respectively ($P < .0017$ for each). Similarly, wintertime upright heart rate increased by 7% ($P < .017$) with larger parallel increases in systemic vascular resistance (+24%, $P < .0017$) and plasma norepinephrine (+26%, $P < .017$). The supine values followed similar trends but the magnitude of changes was about 50% less than the corresponding upright values. Thus, in the northern US, wintertime vasoconstriction is related to increased sympathetic nervous activity and decreased cardiac output. When these reciprocal changes are proportional, blood pressure remains constant. Am J Hypertens 1990; 3:405-407

KEY WORDS: **Seasonal hemodynamics, blood pressure, cardiac output.**

Arterial pressure varies widely under normal circumstances. It is well-recognized that acute stressors can elevate blood pressure transiently but much less attention has been paid to more chronic changes in hemodynamics or blood pressure. One aspect of long term blood pressure variation is seasonal change, a phenomenon well documented by the Medical Research Council of England. In their preliminary report of 51,000 treated or untreated hypertensive patients (17,000 each on placebo, diuretic, or β-blocker), blood pressure increases of approximately 7/3 mm Hg were seen in the winter compared to the summer.[1] However, no data were presented from these studies regarding the degree of heterogeneity of seasonal variation. Other preliminary studies of northern Japanese found blood pressure elevations of as much as 40/30 mm Hg in the winter in association with increased urinary catecholamine excretion.[2]

Such variations could easily confound the diagnosis of essential hypertension or complicate treatment strategies. Furthermore, the underlying hemodynamic changes that accompany such blood pressure shifts are not well understood. The present studies combined neuroendocrine monitoring with noninvasive hemodynamics to address the questions of whether significant hemodynamic seasonal adaptation occurs and whether patterns differ in normotensives as compared to hypertensives. The additional stress of postural adaptation was employed as a predictable stimulant to investigate the role of the sympathetic nervous system in these phenomena.

METHODS

Nonobese subjects were solicited by advertisement and included individuals with borderline hypertension and normotensive subjects. Borderline hypertension was defined as a minimum of 2 documented occasions where clinic or office diastolic blood pressure readings in the sitting position were greater than 90 mm Hg. Subjects whose diastolic blood pressures exceeded 105 mm Hg were excluded from the study, as were

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those with other active medical problems. All subjects were between the ages of 18 and 71 and were not taking any cardiovascular drugs.

Subject enrollment was staggered into 4 seasons, which were defined as summer (July and August), fall (October and November), winter (January and February), and spring (April and May). Each subject was thus studied on four occasions. If a data point was missing, all data for that variable in that patient were omitted from further analysis. Summer was considered the reference point for change.

On each study day, subjects reported to the Outpatient Clinic, where blood pressures were determined after 5 min of recumbency, 5 min of quiet sitting with the back unsupported, and 2 min of upright posture. Supine and sitting blood pressures were the means of 3 determinations, whereas upright blood pressures were the means of 2 determinations. Heart rate was measured immediately after each blood pressure. On each study day, patients had refrained from caffeine or nicotine after midnight.

Subjects were then escorted to the Pulmonary Laboratory, where an indwelling venous catheter was inserted into an antecubital vein. Subjects remained supine for an additional 15 min prior to blood sampling for plasma catecholamines. After blood sampling, subjects performed duplicate acetylene-helium rebreathing maneuvers for determination of cardiac output.[3] Subjects then assumed the upright position for 5 min prior to repeat blood sampling and duplicate cardiac output determinations. Blood pressures were determined before and after each cardiac output in each position and the means of the 4 determinations were used as the final supine and upright values. Heart rate was determined by electrocardiogram during the rebreathing maneuvers, with 2 determinations meaned in each position.

Plasma catecholamines were determined by a modified radioenzymatic assay that has been well-established in our laboratory.[4] Standard statistical techniques included paired Student t tests for comparison of summer with the three other seasons. A somewhat more conservative approach was adopted for judgment of statistical significance during multiple comparisons within each subject (Bonferroni modification); statistical significance was accepted at the $P < .017$ level ($P < .05/3$) for the comparison of any other season to the summer baseline.[5]

RESULTS

In this preliminary report, data were analyzed in 26 subjects during 4 seasons. Of these subjects, partial data loss occurred in 9 subjects. The final group included 10 normotensive and 16 borderline hypertensive subjects and included 4 females and 22 males. Because of data "holes," final hemodynamic data were available in 17 subjects in the supine position and 18 subjects in the upright position, with supine and upright catecholamine values obtained in 26 subjects.

A summary of supine and upright seasonal changes is presented in Table 1. There was only 3 mm Hg in mean arterial pressure variation (diastolic + 1/3 pulse pressure) across the 4 seasons, with a similar pattern in both the supine and upright positions. Hemodynamic changes in the upright position were about 50% greater than those seen for supine parameters, including a wintertime decrease of 18% in upright cardiac output ($P < .017$), a 21% decrease in stroke volume, ($P < .0017$) with corresponding wintertime increases of 24% in sys-

TABLE 1. SEASONAL HEMODYNAMICS

	Summer	Fall	Winter	Spring
A. Supine				
Mean arterial pressure (mm Hg)	89 ± 2	91 ± 2	91 ± 2	89 ± 2
Cardiac output (L/min)	7.2 ± 0.4	6.8 ± 0.3	6.3 ± 0.3*	6.6 ± 0.4
Heart rate (beats/min)	64 ± 2	69 ± 2*	68 ± 2*	64 ± 2
Stroke volume (mL)	117 ± 8	104 ± 7*	96 ± 7†	108 ± 9
Systemic vascular resistance (dyne·sec·cm^5)	1018 ± 63	1094 ± 60*	1173 ± 61†	1108 ± 78
Plasma norepinephrine (pg/mL)	235 ± 20	242 ± 18	277 ± 22	278 ± 19
Plasma epinephrine (pg/mL)	50 ± 3	54 ± 4	48 ± 3	45 ± 3
B. Upright				
Mean arterial pressure (mm Hg)	93 ± 2	95 ± 2	96 ± 2	93 ± 2
Cardiac output (L/min)	5.1 ± 0.2	4.9 ± 0.2	4.2 ± 0.2*	4.9 ± 0.3
Heart rate (beats/min)	77 ± 2	81 ± 2*	81 ± 2*	76 ± 2
Stroke volume (mL)	68 ± 4	59 ± 4*	54 ± 4†	64 ± 5
Systemic vascular resistance (dyne·sec·cm^5)	1514 ± 81	1670 ± 101*	1873 ± 70†	1619 ± 68
Plasma norepinephrine (pg/mL)	455 ± 31	469 ± 28	573 ± 45*	536 ± 33
Plasma epinephrine (pg/mL)	62 ± 4	67 ± 5	60 ± 4	60 ± 5

* $P < .017$, † $P < .0017$.

temic vascular resistance ($P < .0017$) and 26% in plasma norepinephrine (NE) ($P < .017$) compared to summer. In general, fall values were closer to winter, whereas spring values more closely approximated summer.

DISCUSSION

These results demonstrate major predictable hemodynamic changes during seasonal adaptation in the northern United States. In the summer, peripheral vasodilatation is associated with proportional increases in stroke volume and cardiac output. In the winter, peripheral vasoconstriction is associated with proportional decreases in cardiac output and stroke volume. These changes in cardiac output are the opposite of those that would have been predicted from the observed small wintertime increase in heart rate. Because of the association of increased systemic vascular resistance with increased plasma NE (reflecting increased sympathetic nervous activity), it is plausible to assume that seasonal adaptation intrinsically requires activation of the sympathetic nervous system, which mediates arterial and venous constriction. The stimulus for these changes is unknown but could include ambient temperature change or day/night light cycle changes.

Few data are available to further elucidate the mechanisms of chronic climatic adaptation but it is likely that seasonal "volume adaptation" is necessary to subserve basic thermoregulatory needs. It is known that short-term desert acclimatization (heat adaptation) often results in blood volume increases of 1 L or more.[6] In heat-adapted individuals cardiac output rises because of the increases in blood volume and stroke volume, whereas systemic vascular resistance falls.[7,8] Conversely, it is well-known that cold exposure causes diuresis and reduced extracellular fluid volume. In the summer it is necessary to supply more blood to the skin, subcutaneous tissues and extremities to allow adequate heat dissipation. In the winter vasoconstriction reduces blood flow to these same areas, allowing more effective heat conservation. To the extent that courterregulatory increases in systemic vascular resistance equal the corresponding decreases in cardiac output, blood pressure does not change. However, in an individual in whom this counterregulation is not precisely proportional, seasonal blood pressure might change as well. Preliminary data from our laboratory and others indicate that some individuals manifest a form of seasonal hypertension.[1,2]

Unfortunately, blood volume data in the present study are only fragmentary. The original protocol used Evans Blue dye to measure total blood volume during each season. Only 5 subjects had completed the study when the dye was suddenly removed from the American market. At that time it was not possible to convert the remaining subjects to another technique, so total blood volume data are unavailable. Nevertheless, summer blood volume exceeded winter in 4 of 5 subjects by approximately 500 to 1000 mL.

Clarification of the implications of present data for clinical medicine will require further studies. For example, efficacy studies of sympatholytic drugs or volume-depleting agents may require reanalysis to account for seasonal trends. Hemodynamic changes of the magnitude seen in the present study could also affect outcomes of longer term studies of patients with other cardiovascular diseases.

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