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Mini Review

Computing the Reflexes Regulating Blood Pressure and it's Components in Response to Various Pathological and Physiological Factors in Healthy Adolescents

Shantala S. Herlekar*

Department of Physiology, Jawaharlal Nehru Medical College, Belgaum, Karnataka, India

ABSTRACT

An observational study was directed on healthy youth in the age group of 18-22yrs to assess the influences of various physiological and pathophysiological components on individual element of blood pressure. Cardiovascular reflexes were re-gauged to explain probable mechanism for observations made. The results from 120 normotensive subjects helped us revalidate following hypothesis on blood pressure regulatory mechanisms; gender difference showed lower BP elements in females probably due to estrogens protective role on both cardia and vasculature through nitric oxide induction and more. Significant negative correlation of age was seen with RPP hypothesising early and reduced cardiac oxygen extraction. BMI correlated positively with BP proposing changes within circulating blood volume, cardiac output and large vessels. No correlation was seen between handedness and BP element. The extended biopsychosocial model and active baroreceptor reflex have been used to explain influences of acute perceived stress on BP unlike baroreceptor resetting seen during acute physical stress. Rumination, cardiovascular refashioning, reflex adaptation and resetting may be probable explanations for the BP changes seen with chronic perceived stress.

Keywords: Rate pressure product; Acute perceived stress; Acute physical stress; Chronic perceived stress; Blood pressure components

Abbreviations: TPR: Total Peripheral Resistance; RPP: Rate Pressure Product; STAI: State Triat Anxiety Inventary; E2: Estrogen; RAAS: Renin Angiotensin Aldosterone System; SNS: Sympathetic Nervous System

INTRODUCTION

Adequate tissue perfusion is maintained due to lateral pressure exerted by the column of blood within the major arterial system termed Blood Pressure. Arterial pressure=cardiac output*peripheral resistance. Systolic pressure is the maximum pressure in the aorta during the ventricular systole of cardiac cycle and is a measure of "cardiac function", (approximately 120 mm Hg) while, diastolic pressure is the minimum pressure within the aorta, corresponding to diastole of cardiac cycle and signifies TPR. It is a measure of "vascular function"; (approximately 80 mm Hg). The Mean Arterial Pressure (MAP) is the average arterial pressures over a period of time; (85-115 mm Hg). The difference between systolic and diastolic pressure is pulse pressures; (40-50 mm Hg). Pulse pressure measures efficient tissue perfusion. Two major components affect pulse pressure: (1) directly proportional to the stroke volume and (2) inversely proportional to the compliance of the arterial tree. Rate Pressure Product (RPP) is the product of heart rate and systolic blood pressure. It is an comfortably measurable index which associates well with cardiac myocyte oxygen demand and defines the how the coronary circulation reacts to myocardial metabolic demands [1].

The elements of blood pressure are affected by numerous physiological and pathophysiological components. A complex set of reflex mechanisms are involved to maintain all blood pressure elements within the physiological range and are well established. However, re-visiting and re-evaluation them has a large scope.

Correspondence to: Dr. Shantala S. Herlekar, Department of Physiology, Jawaharlal Nehru Medical College, Belgaum, Karnataka, India, Tel: +919900260310; E-mail: shantala.herlekar@gmail.com

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Objectives

To weigh various cardiovascular reflex mechanisms involved in blood pressure modulations in response to various physiological and pathophysiological components. Evaluating and gauging the physiological and pathophysiological reflex mechanisms for the above.

MATERIALS AND METHODS

After obtaining institutional ethical clearance and written informed consent, 120 healthy youth aged 18-22 yrs, with gender number equality, were recruited for the study. Following recordings were taken (Tables 1 and 2).

Blood pressure: SBP, DBP were recorded using Semi - Automatic (HEM-4030) Omron BP Monitor machine in upper right arm. PP, MAP and RPP were calculated using standard formulas (PP=SBP-DBP; MAP=DBP+1/3PP; RPP=SBP*HR)

Gender: male/female.

Age in years: calculated from date of birth.

Height (mts): By commercial stadiometer

Weight (kgs): By digital weighing scale (seca) (accuracy of ± 100 grms).

Body mass index (Kg/mts^2): weight (kg) / height (mts)^2

Handedness: Annett Handedness Questionnaire was used to determine handedness.

Acute and chronic perceived stress: State Triat Anxiety Inventary (STAI) questioner based assessment was done.

BP elements	Male	Female
SBP	128.45 ± 14.90	112.45 ± 9.75
DBP	73.35 ± 9.74	72.17 ± 8.29
PP	55.10 ± 11.77	40.28 ± 8.40
MAP	91.72 ± 10.32	85.60 ± 7.86
RPP	9146.82 ± 1882.68	8944.13 ± 1492.55
HR	70.78 ± 10.08	79.38 ± 9.65

Table 1: Gendre difference in BP and its various elements.

Physiological and pathological components	BP element	Pearson's correlation	p value
Age	RPP	-.153*	0.048
Bmi	SBP	.207*	.012*
	DBP	.166*	.035*
	MAP	.210*	.011*
Handedness	none	-	-
Acute perceived stress	SBP	-.172*	.030*
	DBP	-.249**	.003**
	MAP	-.244**	.004**
	RPP	-.169*	.032*
Chronic Perceived stress	SBP	.181*	.024*
	MAP	.165*	.036*

** Correlation is significant at the 0.01 level (1-tailed).

* Correlation is significant at the 0.05 level (1-tailed).

Table 2: shows significant correlation values of various physiological and pathological components with different blood pressure element.

RESULTS AND DISCUSSION

Literature review for obtained results

Credible and plausible mechanisms for the obtained results have been put forth as follows

Gendre difference

From Tables 1 and 2, it is well established that all BP elements SBP, DBP, PP, MAP and RPP are higher in males. All female subjects had regular menstrual cycles. Hence the oestrogen influences on both cardiac and vessel wall can be used to explain the obtained results and are briefly described herewith.

Vascular validities

Estrogen acts across 2 receptors, α and β. Animal experiments have shown ER action on α enhances basal NO production in the aorta which causes vascular relaxation. It activates endothelial NOS, inhibits vascular muscle proliferation and prevents medial thickening. Estrogen is also known to slow atherosclerosis and improves stress relaxation within the arterial system. These and other effects of ER on vascular tress probably leads to decrease in TPR and in turn DBP in females as compared to males.

Cardiac validities

By its effect on both α and β receptors, estrogen has cardio-shielding influences. It downgrades apoptosis by stabilizing the mitochondrial membrane and prevents apoptosome generation. It eases hypertension associated pathological hypertrophy of the cardia, reducing the chances of further injury.

Other factors like improved parasympathetic activity, lower nor-adrenalin levels, smaller make of the heart, lesser circulating blood volume, responsive blood vessels in females compared to

males, may act as contributory factors. Marys law explains the HR readings being higher in females [2].

Age

Averaging 3rd -5th decade of life, the vascular system undergoes gradual thickening and large arteries slowly begin to enlarge, medial layer hypertrophies, extracellular matrix adds up, with deposits of calcium and endothelium dysregulates. All these add to vascular stiffness and pulmonary arterial refashioning. While in the heart, myocytes thicken due to increase in size and the interventricular septum thickens. The total number of cardiomyocytes reduces.

Our study showed a significant negative correlation with Rate Pressure Product (RPP) even in early 20s. It might direct towards early fall in myocyte mass, hypothesising premature apoptotosis or fibrotic changes or, lipofuscin / amyloid deposits within cardiac musculature. This hypothesis needs further validation [3].

BMI

Elevated BMI showed strong correlation with higher SBP, DBP and MAP. A congregation of mechanism have been well analysed to explain these effects which includes hyperglycaemic toxicity and increased sodium load caused by kidneys, leading to elevated intravascular volume in obese, directing cardiac output and SBP to rise. Untimely RAA, SNS activation along with autonomic dysregulation may be few others to name. Non-efficient endothelial, arterial hardening, extracellular matrix modulations, and abnormal vascular SM are some of the earliest changes in obesity increase in DBP. As BMI increases, baroreceptors become hyporesponsive causing elevated MAP [4].

Handedness

Both the cerebri have distinct control over various cardiac and vascular components like heart rate, blood pressure and cardiac contractility. The right cerebrum is said to control heart rate while the left hemisphere affects cardiac contractility. Researchers suggest this differential control over the heart and vasculature must happen via varied hemispheric control over autonomic nervous system. Evidence suggests right cerebri which is predominant in left handers, has superior control over sympathetic nervous system. Our analysis failed to correlate any baseline changes in blood pressure elements with hand preferences. We hypothesise that hemispheric dominance and autonomic function control may become noticeable only under stressful conditions where blood pressure needs regulation. Our study subjects were at physical rest.

Acute perceived stress

When the body is under physical stress, the baroreceptors are reset to a level higher than at rest, causing obligatory elevation in arterial pressures and HR. This is said to be caused by motor cortex through its central command and afferent inputs from mechanoreceptors. “Extended biopsychosocial model” explains the impact of acute perceived stress on BP which hypothesises that pre-existing psychological traits determines the direction of BP change. Also, contributions are through the coping mechanisms which are used. Like physical stress, Physiological rise in BP should occur with mental stress when active coping strategies are used. But in our study, significant negative correlation was seen between perceived stress and BP. This may reflect baroreceptors did not undergo the said physiological alteration [5]. This could direct towards the assumption that youth in our study used negative coping strategies as, response to their mental stress was reduction in BP components. (Baroreceptor was not reset to higher levels).

Chronic perceived stress

Baroreceptors which are reset during stress should remain so only until the stress is present. If it continuous to remain elevated beyond this point, it causes damaging effects. “Rumination” is where stress related thoughts are retained for a sizable amount of time. Youth in our study probably had a collective trait of rumination, which occurs through the dorsomedial hypothalamus and perifornical area. This might act as a precursor for hypertension [6].

CONCLUSION

This article is useful to gauge outcome of specific factor modifying BP. It also helps to understand probable underlying mechanisms for the same.

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CONFLICT OF INTEREST

None to be report

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