The Effect of Temperature Stress on Hemodynamics: The Mechanisms of Cold Angina, Heat Shock, Tachycardia of Fever and Compensatory Hypertension

Sıcaklık Stresinin Hemodinamikler Üzerine Etkisi: Soğuk Anjinası, Sıcak Şoku, Ateşe Bağlı Taşikardi ve Kompensatuvar Hipertansiyonun Mekanizmaları

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A pioneer study (of 17 Cases) was performed for determining the suitable conditions and the methods of experiments, and it was published as an abstract: "Çınar Y. Body temperature, blood pressure, viscosity and pulse relations. J Gen intern Med 2001;(16):122." The data of these 17 cases is not combined with this new study of 36 cases. While these two studies were performing, the three authors were studying at the same Hospital.

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ABSTRACT Objective: The aim of the study is to investigate the relationships between temperature, blood viscosity and blood flow rate, heart rate, blood pressure and pulse pressure by using temperature stress of cooling and warming on the blood circulatory system. **Material and Methods:** The volunteer and healthy 36 cases were selected using the Simple Random Sampling Method. The study group remained in a cooled room (15°C) for one hour and then were allowed to warm up with an electric blanked (100-watt) in the next period of one-hour. At the end of these two periods, temperatures of arm, leg and axilla, blood pressures, heart rates and blood viscosities of the subjects were measured immediately. **Results:** The results showed that there were clinically and statistically important relationships among blood pressure, heart rate, blood viscosity and pulse pressures during temperature stress of cooling and warming (p= 0.001). However, the pulse pressures of healthy individuals remained constant despite temperature stress related systolic and diastolic pressures changed at rest position, which may resulted from circulatory compensation. **Conclusion:** The system analysis of the data and relationship between the parameters provided explanations for mechanisms of cold angina, tachycardia of fever, heat shock, hypertension and circulatory burden based on temperature stress and blood viscosity.

Key Words: Blood viscosity; angina pectoris; cold temperature; hot temperature; vascular resistance

ÖZET Amaç: Çalışmanın amacı, sıcak ve soğuk ortamlarda ısı stresi uygulayarak, sıcaklık, kan viskositesi ve kan akım hızı, kalp hızı, kan basıncı ve nabız basıncı arasındaki ilişkileri araştırmaktır. **Gereç ve Yöntemler:** Sağlıklı 36 denek rastgele örnekleme yöntemiyle seçilerek aydınlatılmış onayları alındı ve bir saat süreyle ısısı 15°C olan ortamda ve daha sonra bir saat elektrikli battaniye (100-watt) altında yatarak bekletildiler. Her iki sürenin hemen sonunda deneklerin kol, bacak ve aksillalarının sıcaklıkları, kan basınçları, kalp hızları ve kan viskositeleri ölçüldü. **Bulgular:** Bedeni soğutmaya ve ısıtmaya bağlı stresler sırasında; kan basıncı, kalp hızı, kan viskositesi ve nabız basınçları arasında klinik ve istatistiksel olarak önemli ilişkiler olduğu belirlendi (p=0.001). Sistolik ve diastolik kan basınçları istirahat pozisyonundaki ısı stresi ile değişmesine karşılık, muhtemelen kompanzasyon makenizmaları nedeniyle nabız basınçları sabit kaldı. **Sonuç:** Verilerin sistem analiziyle incelenmesi ve parametreler arasındaki ilişkilerin değerlendirilmesi; soğuk anjinası, ateşe bağlı taşikardi, sıcak şoku, hipertansiyon, sıcaklık stresi ve kan viskositesine bağlı dolaşım yükünün mekanizmaları ile ilgili açıklamalar sağlamıştır.

Anahtar Kelimeler: Kan viskozitesi; anjina pektoris; koroner dolaşım; soğuk ısı; yüksek ısı derecesi; damar direnci

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The major discussed effects of increased temperature on the circulatory system are the increased heart rate and vasodilation in the skin that causes heat loss. At rest, high temperature can markedly incre-

ases cardiac output and blood flow of the skin.[1-6] In stress of cold environment, peripheric resistance increases due to the constriction of skin arteries and increased blood viscosity.[7] However, cold angina is explained by increased myocardial oxygen demand, and cold weather hyperventilation can be used to diagnose of vasospastic angina.[7-11]

FACTORS AFFECTING BLOOD VISCOSITY AND HEMODYNAMICS

Viscosity can be defined as resistance of fluid to flow.[12] A common method in determining the viscosity is "the capillary tube viscometer", and it depends on the measurement of time for free flow of the fluid due to gravitational force through a vertical tube that has a constant length and volume. The most commonly defined factors of blood viscosity are dehydration, internal viscosity and flexibility of plasma fibrinogen and protein, deformability and mass quantity of erythrocyte and flexibility of leukocytes in the clinical practice.[13-15] Blood contents, absorbed nutrients and several drugs affect blood viscosity which, in turn, changes the blood pressure and homeostasis at a considerable amount.[16-28] A compensatory increase in the common carotid artery diameter is seen only in the young control groups but not in the elderly hypertensives.[29-31] Therefore, increasing the blood pressure for compensation is the main physiological way whereas decreasing the viscosity to increase the *Q* (flow rate) with hemodilution is a noninvasive way to rescue the tissue from ischemia for the atherosclerotic region.[26,27,30,32]

The aim of this study is to investigate the relationships among temperature stress, blood and plasma viscosity, blood flow rate, heart rate, blood pressure, and pulse pressure by using a flow chart of semi closed system analysis to evaluate the results.

MATERIAL AND METHODS

The investigation confirms the principles outlined in the Declaration of Helsinki and local ethical standards. The informed consents of patients were collected prior to admission of them into the study. Thirty-six healthy subjects who had no medical complaints and had not taken any medication for the last ten days were selected for this study using single random sampling method among different individuals of same populations. The study group includes 18 males and 18 females and the mean age of the population is 34.2 ± 4 years.

The vessel diameter, stroke volume and the hemodynamical parameters are calculated and evaluated according to Poiseuille's equation. *L* (length of the vessel), (viscosity), *v* (flow velocity), *a* (radius of the vessel), *r* (distance from the center of the vessel for a flowing particle), F_1 (initial pressure) and F_2 (final pressure at the end of the distance) are all defined as the variables of that equation [$v=1/4\eta L\ (F_1-F_2)\ (a^2-r^2)$], from which flow rate (Q) can be calculated as $Q=\pi a^4\ (F_1-F_2)\ /8\eta L$.[32]

The limits of temperature stress, case number and other experimental conditions of the study were planned according to the result of a pilot study (another 17 cases), but this new study does not cover that old data.

In the morning, after a 12-hour fasting period, each subject was admitted in a separate room and asked to rest with standard pyjamas for 20 minutes at 22°C of room temperature. Subjects were not allowed to smoke, drink, eat, or exert excessive effort during the period of the study. Their temperatures of arm, leg, axilla, and BP (blood pressure) were measured at the end of the 20-minute rest, and then immediately to measure the blood viscosity, a 9.9 mL of blood was drawn through a plastic cannula containing 0.1mL (500 IU) of heparin sodium. Blood pressure was measured with mercury sphygmomanometer from the right arm and the right leg, and the temperature was determined by ± 0.1°C sensitive digital electronic "Huger Model no: SA880SSX-CE" thermometer in axilla, poplitea and inner side of elbow. Heart rate was recorded with a chronometer for a period of one minute. After that, the subject was taken in to the experiment room that was constantly cooled to 15°C by an air conditioner for one hour and after that, all the parameters were measured again immediately. Then, the air conditioner was stopped and the temperature of those subjects was increased

with insulator and heater effect of a 100-Watt/h (86.042 kcal/h) traditional and standard electrical blanket up to their neck for one hour. Subsequently, all the parameters were measured again immediately. The experiment room is next to the clinic laboratory that all viscosity measurements were done here without any delay.

Blood and plasma viscosity were measured with a temperature controlled type of a simple capillary tube viscometer.[11,15-17] Free flow times (in seconds) of the samples were considered as the data instead of the relative viscosity value in statistical assessments and graphic drawings to achieve more accurate results and to prevent rounding down the numbers. The viscosity of the samples were measured with 37°C and 28°C reciprocal to initial temperature of axilla and extremities respectively; 25°C and 33°C reciprocal to cooling and warming temperature of extremities respectively. In order to make more accurate presentation in the graphic and statistics, the flow time was used as the data instead of "the relative viscosity value" calculated by dividing the flow time of the sample by the flow time of the distilled water.

The results of the study were analyzed statistically using the Paired Sample t test, Wilcoxon Sign test, Spearman's Correlation test and Pearson's Correlation test. Figures were drawn with Statistical Package for Social Scientists 10.0 version.

RESULTS

Distributions of systolic (SBP) and diastolic blood pressures (DBP) by cooling and SBP by warming for the arm are nonparametric, and the others are parametric. Distilled water free flow times in the viscometer at different temperatures were 3.8 (25°C), 3.8 (28°C), 3.7 (33°C) and 3.6 (37°C) seconds. Relationship between temperature and blood pressure in the arm (Figure 1), relationship between arm blood pressure and blood viscosity (Figure 2), relationship between heart rate and systolic blood pressure in the arm (Figure 3), and relationship between pulse rate and blood viscosity increases with temperature (Figure 4) are represented by graphics on coordinate systems, and the table.

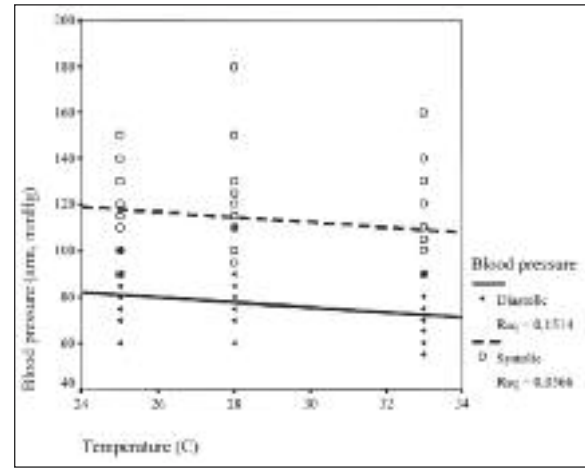

FIGURE 1: Relationship between temperature and blood pressure in the arm. When all systolic and diastolic blood pressure measures of three temperatures (25, 28 and 33 °C) are evaluated together, there is negative correlations between temperature and systolic (r= -0.238, R^2= 0.0566, p= 0.014) or between temperature and diastolic pressure (r= -0.389, R^2= 0.1514, p= 0.000).

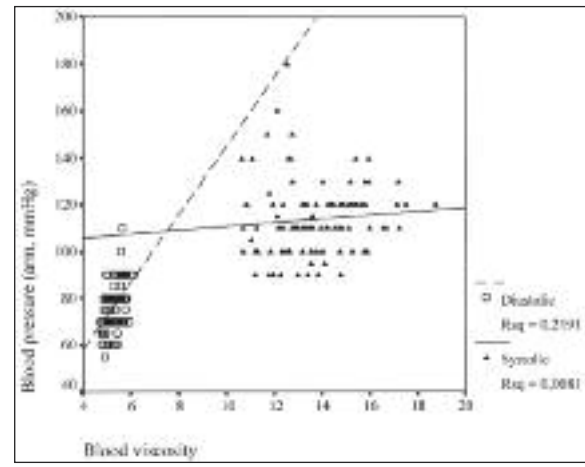

FIGURE 2: Relationship between arm blood pressure and blood viscosity. For arm; if all measured systolic and diastolic pressures and viscosity of blood values are evaluated together in the same diagram, there are positive correlation between viscosity and systolic pressure (r= 0.090, R^2= 0.0427, p= 0.361) and diastolic pressure (r= 0.468, R^2= 0.1516, p= 0.000). Free flow time (seconds) of blood was used as data of blood viscosity in statistical assessments and the graphic drawings in order to achieve results that are more accurate.

The measured results of the in vivo study are represented by the prefix of "M" (measured). When an amount of a compensatory answer was calculated as an algebraic function of one parameter (viscosity) of Poiseuille's equation, it is represented by prefix of "E" (estimated) in the text and figures.

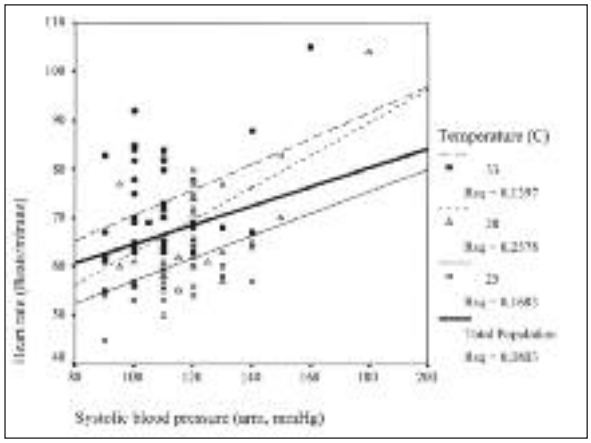

FIGURE 3: Relationship between heart rate and systolic blood pressure in the arm. There are positive correlations between systolic blood pressure and heart rate for every temperature groups (for 33 °C, r= 0.374, R^2= 0.1397, p= 0.027; for 28 °C, r= 0.508, R^2= 0.2578, p= 0.002 and for 25°C, r= 0.410, R^2= 0.1683, p= 0.014). And there is a correlation if all measurements of heart rate and pressure are evaluated in a total population within the same diagram (r= 0.283, R^2= 0.0803, p= 0.003).

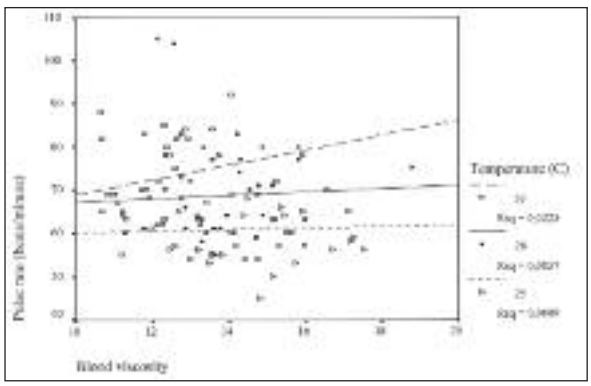

FIGURE 4: . Relationship between pulse rate and blood viscosity increases with temperature. According to increasing temperature of the arm there are increasing correlations between pulse rates and blood viscosities which 28°C of the arm is normal condition of the body and r= 0.061 (p= 0.727), and at the temperature stress of cooling to 25°C, r= 0.031 (p= 0.861), and at the warming to 33°C of the arm r= 0.150 (p= 0.389).

DISCUSSION

Mathematical estimation and evaluations of circulatory compensations are performed according to Poiseuille's Equation and principle of physiology. When blood viscosity increases 5.56% due to temperature decrease, blood flow rate decreases as 5.27%, and for the physiological compensation of this state, a 5.56% of blood pressure increase or 1.36% vasodilatation is needed. If the viscosity (denominator) increases from 100 to 105.56 (5.56%), flow rate would decrease from 100 to 91.7% (100/105.56=0.94.73 and 100%-94.73%=5.27%). If viscosity h increases 5.56% in the denominator, to keep the equation constant, multiplier, the pressure (F_1-F_2) value must be increased with the same percentage.

When the viscosity increases 5.56% in order to keep the constant flow rate (Q), the a^4 value must increase 5.56% and for the a^4_{inital}, the new value is calculated as a^4_{final} =1.0556 x $a^4_{initial}$. From this, $a_{final} = \sqrt[4]{1.0556} x a_{initial} = 1.0136$ and using this value, 1.36% vasodilatation can be found. According to the same equation, when the blood viscosity decreases 13% due to the temperature increase, a 14.9% blood flow rate increase or 13% decrease in blood pressure or 3.1% vasoconstriction is needed for the physiological compensation. When blood viscosity increases, and the body prefers to keep the pressure constant, compensation would be seen as an increased heart rate. When stroke volume is constant, the increasing amount of compensatory heart rate is 13.5% that increases the blood flow rate as 13.5%. The relationships between the parameters and system analysis of the data provided explanations for mechanisms of circulatory compensations could be represented by flow charts according to temperature stress of cooling (Figure 5) and warming (Figure 6).

CHANGING BLOOD PRESSURE AND HEART RATE WITH TEMPERATURE

The results point out that the observed in vivo changes and relations of the circulatory parameters cannot be completely explained by statistical analysis whereas most of them can be estimated in reciprocal relationship of two parameters and via the equation of hydrodynamics exactly. This is not a basic conflict for the hypothesis of this study or inequality between measured data and mathematically estimated probability that estimation of each parameter is based on the changing amount of one parameter (viscosity) for in vitro study. However, blood pressure, heart rate, blood viscosity, and the radius of vessel could change all together due to the alterations of two parameters (temperature and viscosity) in vivo simultaneously.

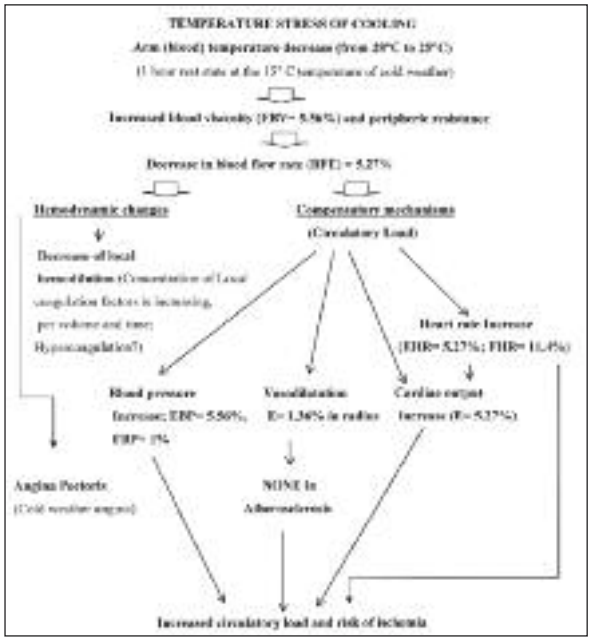

FIGURE 5: Stoichiometric relationships between decreased blood temperature (from 28°C to 25°C), increased viscosity and compensatory changes of the blood pressure (BP), blood flow rate, heart rate (HR) and vessel diameter can be shown as a system analysis on the flow chart. Estimated (E) parameters by Poiseuille's' equation are different from the measured (M) results of the in vivo study that are represented by prefix of E and M respectively
MBV: measured blood viscosity, EBF: estimated blood flow rate, EHR: estimated heart rate, MHR: measured heart rate, EBP: estimated blood pressure, MBP: measured blood pressure, E: estimated. Estimation of each parameter is based on the changing amount of only one parameter of the equation. However, same circulatory parameters could change all together in the body simultaneously.

When the patients were exposed to the temperature stress of warming, blood viscosity decreased 13% while systolic blood pressure of the arm and the leg decreased 7.93% and 10.48% respectively. Besides, the heart rate increased 5.77%. However, when viscosity reduces 13%, the blood pressure should reduce 13% for keeping the flow rate constant according to the Poiseuille's equation. Skin vasodilatation is observed in warm environment despite decreasing BP compensated by tachycardia partially, and this state can be evaluated that protection from fever is more important than hypotension. Exothermic reactions are inhibited by heat; therefore, tachycardia could not only be caused by stimulated myocardial metabolism by warmth, but also a physiological response to increase the cardiac output and blood pressure.

When the patients were exposed to the temperature stress of cooling; blood viscosity increased 5.56%, while systolic blood pressure increased 2.14% and 1% for the leg and arm respectively. In fact, when viscosity increases 5.56%, the blood pressure at the extremities should increase at the same rate to keep the Poiseuille's equation (or *Q*) constant. So, the unknown percentage of this compensation is at least 3.42% (5.56-2.14=3.42) for blood pressure. Increase in blood viscosity (also increase in total peripheral resistance) due to cooling is responsible for the rise in blood pressure at the leg at a rate of 4.2% (r=0.207) for systolic phase, and 15% (r=0.389) for the diastolic phase. To obtain a fate for this unknown part of the compensation to keep the Q constant with a simultaneous vasodilatation; 100+3.42=103.42; $a^4_{final}=1.0342xa^4_{initial}$; $a^4_{final}=\sqrt[4]{1.0342xa_{nitial}}$ = 1.0084 =0.84%, it is found out that 0.84% increase in the radius of the vessel is needed.

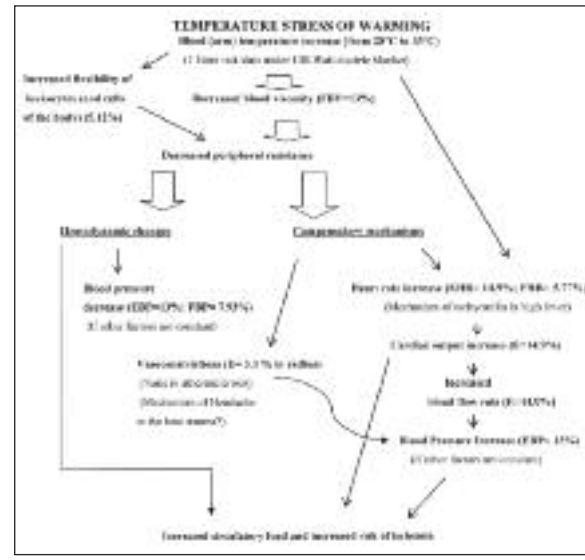

FIGURE 6: Stoichiometric relations between increased blood temperature (from 28°C to 33°C), decreased viscosity and compensatory changes of the blood pressure (BP), blood flow rate, heart rate (HR) and vessel diameter can be shown as a system analysis on the flow chart. Estimated (E) parameters by Poiseuille's' equation are different from the measured (M) results of the in vivo study that are represented by prefix of E and M respectively.
MBV: measured blood viscosity, EBF: estimated blood flow rate, EHR: estimated heart rate, MHR: measured heart rate, EBP: estimated blood pressure, MBP: measured blood pressure, E: estimated. Estimation of each parameter is based on the changing amount of the only one parameter of the equation. However, same circulatory parameters could change all together in the body simultaneously. The intersections of the arrows are meaningless.

CONCEPT OF THE TOTAL PERIPHERAL RESISTANCE

Conventional concept of the circulatory resistance is expressed as vascular resistance or the pressure drop divided by the blood flow rate.[10] However, according to Poiseuille's equation and the results of this study, the concept of (total) peripheral resistance should be considered as a common result of vascular resistance, blood temperature and blood viscosity.

MECHANISM OF CORONARY ANGINA IN COLD ENVIRONMENT

The results of the study show that when the temperature decreases, blood viscosity increases and blood flow rate decreases, and to compensate this, a significant blood pressure increase takes place. According to the calculations of the study, the effects of cold weather that the blood temperature decreases at the arms, legs, face, ears and lungs will cause cool and viscous blood perfusion, increasing peripheral resistance and decreasing blood flow rate in vessels. These could provide an additional explanation for the mechanism of coronary angina seen in cold environment. According to the results of the study, when it is accepted that temperature and viscosity relationship is linear between 36.5°C and 22°C (throughout the 14.5 °C that 36.5-22= 14.5) and the increase of blood viscosity as 29.05%, only a 1°C decrease of temperature results in 2% (29.05 /14.5= 2) increase of viscosity and 1.97% (100/102= 0.9803; 100-98.03= 1.97) decrease of blood flow rate in coronary vessel. On the other hand, a 2% increase of systemic peripheral resistance appears (when viscosity of blood increases 2%), and 2% increase of systemic blood pressure is required for the compensation of blood flow rate that produces additional circulatory burden and risk of coronary ischemia. Therefore, circulatory burden based on temperature and viscosity of blood can be calculated in clinical practice, and these are the evidences that coronary angina of cold weather can be triggered by increasing the viscosity of blood together with cold vasospasm.

AN ADDITIONAL MECHANISM FOR THE HEAT TRAUMA

Mechanisms of tachycardia and hypotension seen in hot weather and heat shock are explained by decreased "vascular resistance" due to vasodilatation.[1,2] According to the Poiseuille's equation, when blood viscosity decreases 13%, the blood pressure decrease 13% due to decreased peripheral resistance. For physiological compensation of this state, a 14.9% cardiac output increase is required. As a result, high temperature related blood viscosity (resistance to flow) decrease must be an important factor in the mechanism of heat shock, and performing a viscosity decreasing therapy (glycerol, nifedipine, nitroglycerine, isosorbite dinitrate, hemodilution, decreasing the hematocrit etc.) must be evaluated carefully during heat shock and the conditions of decreased peripheral resistance.[11,15-17]

MECHANISM OF TACHYCARDIA AND FEVER

According to the Poiseuille's equation, when blood viscosity decreases 13% due to temperature increase (from 28°C to 33°C), blood pressure decreases 13%, and for physiological circulatory compensation, 14.9% of the blood flow rate or cardiac output increase is needed. If stroke volume is constant, 14.9% increase of cardiac output is needed to increase heart rate by the same proportion as 14.9 % (Figure 6). A 14.9% increase of 80 heartbeats per minute is equal to 98.6 beats per minute. This evaluation provides an explanation for the mechanism of temperature dependent tachycardia based on temperature related change of viscosity.

EFFECT OF TEMPERATURE STRESS ON PULSE PRESSURE

Pulse pressure (difference of systolic pressure from diastolic pressure) was nearly constant despite of changing temperature, systolic blood pressure, diastolic blood pressure, viscosity and pulse rate (Table 1). Even though blood viscosity and blood pressure increased and heart rate decreased in cold environment, the same parameters changed in the opposite direction in warm environment. Thus, the increased heart rate could be a response to the decreased peripheral resistance and blood pressure in warm environment, and inversely, the decreased heart rate could be a compensatory response to the increased peripheral resistance and blood pressure in cold by decreased temperature at rest. However, this compensatory state of myocardium should not be confused by decreasing heart rate with the inhibited metabolism in hibernation. Because the long

TABLE 1: Changing in blood viscosity and circulatory parameters of the body with the temperature stress of cooling or warming.

Parameter	Initial value	Effect	Result	Difference %	Significance
Axilla temp.	36.12 ± 0.39	Cooling	35.68 ± 0.5 1	1.22 ↓	t= 6.075. p= 0.000
Arm temp	30.80 ± 1.04	Cooling	26.03 ± 1.66	15.49 ↓	t= 15.080. p= 0.000
Leg temp	30.18 ± 0.67	Cooling	25.78 ± 1.36	14.58 ↓	t= 19.055. p= 0.000
Axilla temp	36.12 ± 0.39	Warming	36.48 ± 0.32	0.99 ↑	t= -8.078. p= 0.000
Arm temp	30.80 ± 1.04	Warming	32.15 ± 1.18	4.38 ↑	t= -7.017. p= 0.000
Leg temp	30.18 ± 0.67	Warming	31.84 ± 0.99	5.50↑	t= -10.38. p= 0.000
Arm SBP	116.14 ± 14.96	Cooling	117.14 ± 15.06	1.0↑	z= -0.387. p= 0.698
Arm DBP	78.71 ± 8.57	Cooling	80.29 ± 8.22	2.0↑	z= -0.773. p= 0.440
Leg SBP	192.86 ± 31.28	Cooling	197 ± 33.17	2.14↑	z= -0.719. p= 0.472
Leg DBP	122.57 ± 20.45	Cooling	127.109 ± 18.3	3.85↑	t= -1.455. p= 0.155
Arm SBP	116.14 ± 14.96	Warming	107.86 ± 15.50	7.93↓	z= -3.600. p= 0.000
Arm DBP	78.71 ± 8.57	Warming	71.71 ± 9.15	8.9↓	z= -3.804. p= 0.000
Leg SBP	192.86 ± 31.28	Warming	172.71 ± 26.72	10.48 ↓	t= 4.162. p= 0.000
Leg DBP	122.57 ± 20.45	Warming	107.29 ± 18.04	15.72↓	t= 4.489. p= 0.000
Heart rate	68.69 ± 9.92	Cooling	60.86 ± 8.39	11.4↓	t= 7.251. p= 0.000
Heart rate	68.69 ± 9.92	Warming	72.66 ± 10.94	5.77↑	t= -3.497. p= 0.001
Pulse press. arm	37.43	Cooling	36.85	≈ stable	t= 1.576. p= 0.124
Pulse press. leg	70.29	Cooling	69.71	≈ stable	t= 0.51. p= 0.881
Pulse press. arm	37.43	Warming	36.15	≈ stable	t= 1.876. p= 0.069
Pulse press. leg	70.29	Warming	65.42	≈ stable	t= 1.301. p= 2.02
Blood viscosity	14.01 ± 1.50 (28°C)	Cooling	14.79 ± 1.42 (25 °C)	5.56↑.	t= -4.208 p= 0.000
Blood viscosity	14.01 ± 1.50 (28°C)	Warming	12.19 ± 0.94 (33°C)	13↓	t= 10.886. p= 0.000

Effect [increase (↑) or decrease (↓)] of temperature change (cooling or warming) on parameters [Temperature °C (Temp.), Systolic (SBP) and diastolic (DBP) blood pressure (mmHg), Pulse pressure mmHg (Pulse press.), heart rate beat/min, viscosity (flow time/ second)]. Note: The mean of ≈ is approximately stable.

lasting rest position inhibits the metabolism and the circulation that body goes to sleeping (parasympathetic dominancy) condition, and the aimed balance of circulatory compensation could be different in the rest state.

The in vivo "measured blood pressure" increase (MBP= 1%) is lower than the mathematically "estimated blood pressure" increase (E=5.56%) for compensation of blood flow rate decrease by temperature stress of cooling. On the other hand, the in vivo "measured heart rate" (MHR=11.4%) increase is higher than mathematically estimated (E=5.72%) blood pressure increase (Figure 5). Therefore, we conclude that the in vivo compensations are provided by the increased blood pressure and heart rate together, and the heart rate increase is the basic compensatory way in cold weather. However, compensatory changing amount of vessel radius is unknown for the in vivo condition that affects the whole parameters of blood circulation.

When the body temperature increased, decrease in blood pressure (MBP= 7.93%) is lower than mathematically estimated blood pressure decrease (E= 13%). Similarly, in vivo measured heart rate increase (MHR= 5.77%) is lower than the estimated heart rate increase (E= 14.9%) by the temperature stress of warming (Figure 6). We conclude that changing the amount of blood temperature may be inefficient by 100 watt/hour electric blanket, and the in vivo compensation is provided by common changing of blood pressure, heart rate and vessel radius. However, compensatory changing amount of stroke volume and radius of vessels are not known here. Since the atherosclerotic vessels cannot vasodilate, it could be proposed that increasing heart rate and blood pressure become dominant ways to compensate the circulatory burden when atherosclerosis grows up in the body.[29, 33-37] This state could be called as the compensatory hypertension of atherosclerosis, but not as the essential hypertension.

The results (Table 1) also suggest that keeping pulse pressure constant is more important than the stability of systolic or diastolic blood pressure for resting state of healthy people despite temperature stress. The mathematical evaluation provides additional evidences. When myocardium works in hypertensive condition as 190/140 mmHg instead of 120/70mm Hg, pulse pressures are constant as 50 mmHg but increase of diastolic pressure is one fold and resistance ($R=F_1-F_2/Q$ or $R=8\eta L/\pi a^4$) of aorta raises approximately by one fold (relationship between diastolic pressure and resistance is 1/1). Thus, blood flow rate (Q) remains stable because of the constant pulse pressure via increasing systolic pressure.

BLOOD VISCOSITY AND HYPERTENSION

According to the calculation above, when blood viscosity increases 13%, a 13% of blood pressure increase is needed for physiological compensation that is very important in clinical practice. These conditions should be evaluated by the data of blood viscosity and pressure, and so the increased blood viscosity could come out as an important factor for the increased blood pressure and ischemia.[15] The idea that a long lasting period of compensatory hypertension due to hyperviscosity produces an essential hypertension is not clear yet. However, there is an old idiom that hypertension makes hypertension.[29, 33-36]

CONCLUSION

Temperatures affect the pressure and the parameters of blood circulation in distinctive ratios. However, the pulse pressure of healthy individuals remained constant despite temperature stress related to the systolic and diastolic changes at rest position, which may resulted from circulatory compensation. The concept of total peripheric resistance considered as blood temperature, viscosity and vascular resistance should be taken into account altogether.

We present a new approach to the relationship between the hemodynamical parameters and suggest that the flow charts and additional explanations given for the mechanisms of cold angina, tachycardia of fever, heat shock, and concept of total peripheral resistance should take their places in the analysis of the hemodynamic control mechanism of the human body and therapeutic process of atherosclerosis, ischemia, hyperviscosity and hypertension.

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