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Awareness of Lifestyle Modifications Among High Stress Professionals Prone For Cardio-Metabolic Derangement

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Abstract

Introduction: Stress has become an integral part of our lifestyles. Stress to some extent improves management skills, job performance but when crosses the limit results adversely on health. Recent studies have mentioned majority of software professionals and bank employees are stressed and suffer from related disorders. Work duration, workload and mental stress have a great impact on the functioning of cardio-metabolic system and thereby evaluation of these functions helps to detect any health hazards and take appropriate preventive measures at the earliest. We carried out this study to find out the stress levels among working middle-aged male professionals through a questionnaire-based analysis; study the effects of work-related stress on Blood Pressure (BP), Body Mass Index (BMI), Waist-Hip ratio (W/H), Blood glucose and lipid profile and to find out their awareness about lifestyle modifications. *Materials and Methods:* The study consisted of 133 male participants of which 60 were bank employees and 73 software professionals. A pretested and validated questionnaire on work stress divided the participants into non-stressed and stressed groups. Their age, work experience, BP, BMI and Waist/Hip ratio, Random Blood Sugar, Lipid parameters and Atherogenic index were analyzed. Their awareness on lifestyle modifications was assessed. These parameters were compared by unpaired *t*-test. All tests were two-tailed and $p < 0.05$ was considered significant. *Results and Conclusions:* The study showed that significant stress levels are observed in these professions and as a result, there is derailment of cardio-metabolic parameters among them. There exists a difference in awareness about the need for yoga and exercise among them. Hence while addressing the complications of job stress, a more aggressive working health policy has to be implemented at all workplaces.

Keywords: Lifestyle; Occupational; Professionals; Stress; Yoga; Workplace.

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Introduction

Stress has become an active part of every occupation. Stress to some extent improves management skills and job performance but when it crosses the limit, adversely affects health; physical and mental. Work-related stress has been known to cause many ill-effects on the body or accentuate already existing morbidities. There is no standard procedure to track back and pin-point the causation and underlying mechanism of ill health as "Stress".[1] But a rough estimate can be done by observing the health of people conditioned under stress. Such knowledge will help us to classify stress as an 'etiologic agent' or a 'predisposing factor'. By this, we can confer jobs with work-related stress as a potential health hazard.

Job stress has been known to potentiate the onset and the progress of diabetes mellitus, hypertension, bronchial asthma, and metabolic syndromes.[2,3]

Of all the morbidities; hyperacidity, obesity, cardiovascular manifestations, and depression are the most common complaints of stressed workers.

World Health Organization (WHO) defines work-related stress as "is the response people may have when presented with work demands and pressures that are not matched to their knowledge and abilities and which challenge their ability to cope".[4] Stress effects have been observed in almost all professions, more so in the banking sector and software industry as per the recent survey.

The banking sector forms an important contributor to the growth of the country's economy. India had 14 nationalized banks in 1969. Now scheduled commercial banks, private banks, public sector banks, regional rural banks, and foreign banks are added. The number of employees working in this sector has increased as well as the competition and associated stress.

Such organizational stress along with the sedentary lifestyle of a bank employee has made him our ideal candidate for studying cardio-metabolic risk factors that have been conditioned under work stress. World Health Organization report shows that 17.3 million deaths per year globally are due to cardiovascular disease.[5] There has been an emerging trend of cardiovascular disease leading to mortality in developing nations.[6,7] In developing countries, 80% of deaths are due to cardiovascular disease.[8] Workplace stress has a major impact on personal, professional, organizational and national development. According to Botnia study in a population of Western Finland, finance, work, and social relationships had a direct effect on the prevalence of insulin resistance, obesity, and altered lipid levels.[9] Work stressed employee suffers psychological strain and is more prone to develop cardiovascular disease according to a German study.[10] It was observed that work duration, workload, and mental stress alter the functioning of cardiac[11] and autonomic nervous system.[12]

Stress can be observed in all fields of work; big or small[13] Earlier what was observed in heavy workers involving more of stressful activities at workplaces, has now become a common entity observed even in sedentary work atmospheres. Due to rapid changes in globalization, economic liberalization, financial progress, technological advancements, stress has entered banking industry. As there has been a rapid spurt of private banks into the arena in recent years, occupational stress in these employees is crippling their performance and health. Levels of stress depend on working conditions, workload, management, leadership, strict deadlines to achieve ambitious targets and also on the type of banks whether Government or private. It has been observed that stress levels are more in the employees of non-nationalized banks compared to those working in Nationalized banks.[14] Long hours of working conditions in the banking sector create a stressed mind that adversely affects their health and performance according to Jamshed et al.[15]

Corporate Indian adults are exposed to stressful life in the software profession. Increased work stress, strict deadlines, high expectations, soaring competition are some of the causes of this. A recent study by the Association Chambers of commerce and Industry of India (ASSOCHAM) has mentioned that 54% of workers in software industry are suffering from stress and its disorders like hypertension, diabetes, obesity, spondylosis, depression, and headaches. It has been shown that work duration, workload, mental stress, and stress-related disorders have a greater impact on the functioning of autonomic, cardio-metabolic systems and thereby evaluation of these functions helps to detect any health hazards.

A clear understanding of stress, its pathophysiology and its effects on the health of a bank employee and software professionals will help us find better remedies to manage work stress in banking sector and software industry effectively and add more years to the lives of the human resource of our country.

Objectives

1. To find out the stress levels among working middle-aged male professionals through a questionnaire-based analysis.
2. To study the effects of work-related stress on Blood Pressure (BP), Body Mass Index (BMI), Waist-Hip ratio (W/H), Blood glucose and lipid profile.
3. To find out their awareness about lifestyle modifications.

Materials and Methods

The present cross-sectional study consisted of 133 male participants by convenience sampling method of which 60 were bank employees 35–60 years age group and 73 software professionals 21–45 years age group. Ethical approval was obtained for this study from the Institute's Ethical review committee. Written informed consent was taken from each participant after describing in full

detail the procedure and purpose of the study.

A validated self-administered Likert Scale questionnaire on work stress was used for the study. Responses were collected from the participants. The questionnaire consisted of 20 questions; each with a minimum score of 0 and maximum of 4 per question, scores 0 to 25 indicate that the person is probably coping adequately with his job. If 26 to 40, there is job stress and a need to take preventive action. If scores are 41 to 55, employee needs to take appropriate action to avoid job burnout. And any score from 56 to 80, requires a comprehensive job stress management plan to be started at the earliest.

Based on the analysis of their job stress levels, study group was divided into control (Non-stressed) and test/experimental group (Stressed). Their age, Blood pressure (BP), Body Mass Index (BMI) and Waist-Hip Ratio (WHR) were recorded. General physical examination and, complete systemic examinations were done. A detailed history which included the work history, diet history, family and drug history were taken.

We included 35-60 year-old male bank employees, 21-45 year-old software professionals with stress level scores 26 and above as the test group. Healthy, non-stressed age-matched employees formed the controls. Those with a history of neurological disorders and any systemic illness were excluded.

Blood sample was collected under all aseptic conditions and blood glucose levels were measured by Glucoseoxidase-peroxidase endpoint by Trinder's method using glucose reagent. (Transasia Bio-Medicals Ltd, Solan, Himachal Pradesh, India). Their lipid profile parameters; Total Cholesterol (TC), High-Density Lipoprotein (HDL), Low-Density Lipoprotein (LDL), Triglycerides (TG), Very Low-Density Lipoprotein (VLDL) were measured. Lipid ratios TG/HDL, TC/HDL, LDL/HDL, Atherogenic index in Plasma (AIP) by Log (Triglycerides/HDL-Cholesterol) were calculated. AIP values of -0.3 to 0.1 are associated with low, 0.1 to 0.24 with medium and above 0.24 with high cardiovascular risk. Another questionnaire was administered to assess their awareness of lifestyle modifications. Data were analyzed for normal distribution. Age, work experience, BP, BMI, Waist-Hip ratio, Blood glucose, and Lipid profile parameters among stressed and non-stressed middle-aged male professionals were analyzed statistically by using the statistical software SPSS and MS Excel. All tests were two-tailed and $p < 0.05$ is considered as significant.

Results

Stress analysis of Bank employees

Based on the analysis of the questionnaire, there were 21 stressed and 39 non stressed male bank employees in the study group. The stressed group had a mean stress score of 34 whereas in the nonstressed group it was 22. The mean age of the stressed and nonstressed males was 50.71 ± 7.03 and 50.36 ± 8.04 years respectively (Table 1) 30.8% of the stressed were aged below 50 years. 38.2% were above 50 years old (Table 1). The most common responses from questionnaire analysis showed that 78% are callous nature about others' problems, 68% have dissatisfaction, 61% suffer from forgetfulness, 56 % are irritable/impatient and 55% have lost their time, energy.

Systolic Blood Pressure and Diastolic Blood Pressure were significantly more in stressed individuals. (p = 0.035 and 0.031 respectively) (Table 1). BMI was statistically more in the test group. (p = 0.04) Mean RBS (mg/dl) in the stressed group was 139.62 ± 37.45. Non-stressed males had 121.43 ± 24.78 with a significance value of p 0.02. Waist/Hip ratio was not significant among the study group. Lipid parameters like Total Cholesterol, HDL, VLD Lipoprotein levels were not significantly altered except the Triglyceride and LDL levels which were elevated with p-value of 0.034 and 0.037 respectively (Table 1). TG/HDL was highly significant with p 0.0004. Atherogenic Index, a predictor for cardiovascular risk was significant p-value of 0.0002.

Stress analysis of Software professionals

There were 21 stressed and 53 non stressed in the study group. Stressed group had a mean stress score of 36 where as in the non stressed group it was 24. Mean age of the stressed and non stressed males were 28.53 ± 6.25 and 27.62 ± 5.24 years respectively (Table 3). The most common responses from questionnaire analysis showed that they are physically, emotionally, spiritually depleted, have irritability, impatience, eating more or less, drinking more coffee, smoking more cigarettes, or using more alcohol or drugs to cope with job, feel a sense of dissatisfaction, of something wrong or missing and have less than usual decision-making ability.

Systolic Blood Pressure and Diastolic Blood Pressure were significantly more in stressed individuals p-value 0.0001 (Table 3). No difference was seen with respect to obesity parameters among the group probably due to their young age.

Random blood sugar was elevated in the stressed group (p-value =< 0.0009). Lipid parameters like LDL, VLD Lipoprotein levels were not significantly altered except Total Cholesterol, Triglycerides and HDL levels which were elevated with *p* – value of 0.026 and 0.0001 and 0.0016 respectively. TC/HDL, TG/HDL and LDL/HDL were more in stressed group and highly significant with *p* 0.0001. Atherogenic Index, a predictor for cardiovascular risk was more in stressed group with significant *p* – value of 0.0001 (Table 3).

When the responses of bank employees on

Table 1: Baseline and cardio-metabolic risk parameters of bank employees.

Parameter	Stressed	N	Mean ± SD	*p*
Age (Years)	Yes	21	50.71 ± 7.03	1.534
	No	39	50.36 ± 8.04	1.288
Work experience (yrs)	Yes	21	20.67 ± 6.45	0.53
	No	39	22.13 ± 9.56	
Stress score	Yes	21	34	
	No	39	22	
Systolic BP mmHg	Yes	21	137.33 ± 12.17	0.035*
	No	39	128.29 ± 20.15	
Diastolic BP mmHg	Yes	21	87.69 ± 9.24	0.031*
	No	39	81.14 ± 11.76	
BMI kg/m2	Yes	21	29.62 ± 7.45	0.04*
	No	39	26.43 ± 4.78	
WHR	Yes	21	0.93 ± 0.05	0.703
	No	39	0.93 ± 0.05	
RBS mg/dl	Yes	21	139.62 ± 37.45	0.02*
	No	39	121.43 ± 24.78	
TC mg/dl	Yes	21	211.44 ± 34.70	0.259
	No	39	200.86 ± 33.54	
TG mg/dl	Yes	21	258.95 ± 80.72	0.034*
	No	39	220.18 ± 57.12	
HDL mg/dl	Yes	21	46.97 ± 12.78	0.492
	No	39	49.48 ± 14.41	
LDL mg/dl	Yes	21	117.31 ± 37.59	0.037*
	No	39	101.24 ± 21.08	
TG/HDL	Yes	21	5.50 ± 1.41	0.0004***
	No	39	4.44 ± 0.88	
TC/HDL	Yes	21	4.50 ± 2.71	0.5116
	No	39	4.06 ± 2.32	
LDL/HDL	Yes	21	2.49 ± 0.94	0.21
	No	39	2.04 ± 1.46	
Atherogenic index	Yes	21	0.74 ± 0.14	0.0002***
	No	39	0.64 ± 0.05	

*Statistically significant

Table 2: Awareness of bank employees on lifestyle modifications

Item	Stressed	N	Strongly Agree	Agree	Neither Agree nor Disagree	Disagree	Strongly Disagree
Can Yoga/ exercise can improve the wellbeing	Yes	21	19 (90%)	02 (10%)	–	–	–
	No	39	29 (74%)	07 (18%)	03 (8%)	–	–
Is yoga and exercise to be done after 40 years of age	Yes	21	–	–	–	10 (48%)	11 (52%)
	No	39	–	–	01 (3%)	20 (51%)	18 (46%)
Yoga and exercise must be practiced by people of all age group	Yes	21	11 (52%)	06 (29%)	03 (14%)	01 (5%)	–
	No	39	22 (57%)	13 (33%)	–	04 (10%)	–

lifestyle modifications were analyzed it was observed that 90% of the stressed and 74% of the non stressed strongly agreed that yoga/exercise improves the wellbeing of a person. Majority of them disagreed that yoga and exercise to be done after 40 years of age. They were of the opinion that yoga and exercise must be practiced by people of all age groups. Around 10% of the nonstressed group disagreed to this (Table 2).

The responses of software professionals on lifestyle modifications showed that there was a difference in opinion compared with the bank employees on the fact that yoga and exercise to be done after 40 years of age. Only 10% of the non stressed group strongly agreed that yoga and

Table 3: Baseline and cardio-metabolic risk parameters of software professionals.

Parameter	Stressed	N	Mean ± SD	*p*
Age (Years)	Yes	21	28.53 ± 6.25	0.526
	No	53	27.62 ± 5.24	
Work experience(yrs)	Yes	21	2.69 ± 2.31	0.14
	No	53	3.58 ± 2.33	
Stress score	Yes	21	24	
	No	53	36	
Systolic BP mmHg	Yes	21	142 ± 16.09	0.0001***
	No	53	124 ± 16.03	
Diastolic BP mmHg	Yes	21	96 ± 11.10	0.0001***
	No	53	80 ± 11.16	
BMI kg/m2	Yes	21	24.72 ± 3.07	0.8334
	No	53	24.57 ± 3.64	
WHR	Yes	21	0.89 ± 0.05	1.0000
	No	53	0.89 ± 0.05	
RBS mg/dl	Yes	21	123.61 ± 28.78	< 0.0009***
	No	53	91.83 ± 19.79	
TC mg/dl	Yes	21	198.79 ± 38.34	0.026**
	No	53	177.85 ± 28.16	
TG mg/dl	Yes	21	125.39 ± 19.56	< 0.0001***
	No	53	87.86 ± 14.48	
HDL mg/dl	Yes	21	34.56 ± 4.78	0.0016***
	No	53	42.71 ± 5.27	
LDL mg/dl	Yes	21	91.29 ± 7.07	0.071
	No	53	89.46 ± 3.12	
TG/HDL	Yes	21	3.55 ± 0.05	0.0001***
	No	53	2 ± 0.1	
TC/HDL	Yes	21	5.65 ± 0.25	0.0001***
	No	53	4.13 ± 0.18	
LDL/HDL	Yes	21	2.5 ± 0.3	0.0001***
	No	53	2.1 ± 0.2	
Atherogenic index	Yes	21	0.55 ± 0.07	0.0001
	No	53	0.3 ± 0.11	

*Statistically significant

Table 4: Awareness of software professionals on lifestyle modifications

Item	Stressed	N	Strongly Agree	Agree	Neither Agree nor Disagree	Disagree	Strongly Disagree
Can Yoga/ exercise can improve the wellbeing	Yes	21	17 (80%)	4 (19%)	—	—	—
	No	53	34 (64%)	17 (32%)	02 (4%)	—	—
Is yoga and exercise to be done after 40 years of age	Yes	21	—	1 (4%)	01 (4%)	10 (48%)	09 (44%)
	No	53	—	6 (11%)	09 (17%)	23 (44%)	15 (28%)
Yoga and exercise must be practiced by people of all age group	Yes	21	11 (52%)	04 (19%)	01 (5%)	05 (24%)	—
	No	53	10 (19%)	40 (75%)	—	03 (6%)	—

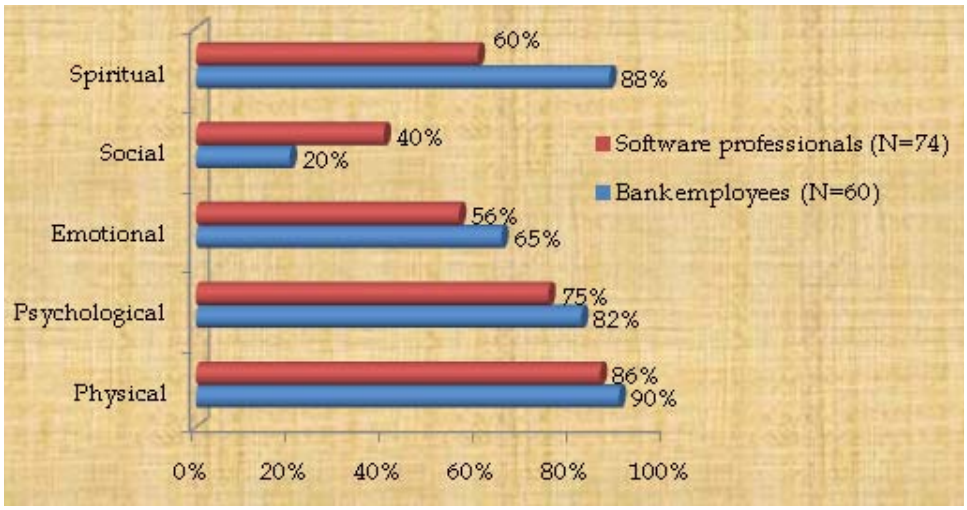

Fig. 1: The perception of the study group towards Yoga activity

exercise must be practiced by people of all age groups (Table 4).

We asked about their perception of Yoga activity: Physical/Psychological/Emotional/Social/Spiritual. Each individual had different perception about it. Participants selected the activities which were applied to them. The majority of them selected almost all the above options. But it was observed that many of them think that yoga is a physical activity. It was considered less of a social activity among bank employees (20%) compared to 40% among software professionals (Fig. 1).

Discussion

This study revealed the impact of job stress on cardiometabolic risk factors in male professionals. Stressed participants of the two groups had elevated BP, BMI, glucose and lipid levels. It has been shown from the earlier works that prolonged exposure to work stress affects autonomic, cardiovascular[16] and neuroendocrine activity directly, contributing to the development of various disorders. Obesity, hypertension, diabetes mellitus, hyperlipidemia form important components of metabolic syndrome. Various factors like genetic, environmental, biological play interconnecting role in its pathogenesis. Stressful condition whether personal or professional leads to the activation of the hypothalamo-pituitary-adrenal (HPA) axis, leading in turn to endocrine abnormalities, such as high cortisol, low sex steroid levels and increase in visceral adiposity. High cortisol stimulates gluconeogenesis and glycogenolysis in skeletal muscle which inhibit insulin sensitivity and glucose uptake contributing to insulin resistance.[17] Our results are in accordance with the studies by Chandola et al. that showed that stressful work results in hypertension, obesity, hyperlipidemia and coronary vascular diseases.[18]

Stressed workers had significantly high BP both systolic and diastolic. Job stress is more prevalent under 50 years of age further developing cardiovascular diseases.[19] Autonomic dysfunction in stressed workers was observed as a result of sympathetic predominance and vagal withdrawal that increases the BP.[20] Earlier studies have shown that stress induces the activation of sympathetic system releasing catecholamines which increases Blood Pressure.[21] Obesity leading to insulin resistance has also been linked to the dysfunction of autonomic system and sympathetic predominance acting through the hypothalamus that increases Blood Pressure.[22]

Our study showed that BMI, an important obesity marker is elevated in stressful work atmosphere among bank employees and not among software professionals probably due to their lesser mean age. White hall II study has depicted the association between work stress, autonomic activity, cortisol levels, weight gain, blood glucose levels and hence the emergence of metabolic syndrome.[23]

Lipid parameter, LDL, Triglyceride levels, TG/HDL levels were elevated in the stressed individuals as per the present study. Qureshi et. al., have discussed the role of routine mental stress in cardiovascular derangement and lipolytic changes even in healthy young individuals in the age group of 18-23 years. There were increased BP, Heart rate and lipid levels in 114 medical students.[24]

Young men had increased lipid levels like TG, LDL, VLDL and elevated Total cholesterol count as a result of stress.[25] Job stress has been associated

with altered cholesterol, HDL and LDL levels.[26] Cortisol and epinephrine have been linked to the pathogenesis of dyslipidemia.[27] The sympathetic nervous system innervating the adipose tissue activates the process of lipolysis. Elevated LDL, Triglyceride levels promote the development of atherosclerosis. Atherogenic Index is a better marker to test the atherogenic dyslipidemia and can identify increased coronary artery disease risk compared to cholesterol ratios which was more in the stressed employees in our study. The atherogenic index of plasma was calculated to predict cardiovascular risk. The value should be below 0.1.[28]

Our study showed that though the study participants are aware of the lifestyle modifications that are required in their lives, majority of them are not following them strictly. There is difference in perception of the young professionals about the yoga activity which needs to be looked into. Previous studies have shown that young individuals should be informed about the benefits of yoga and exercise and they need to be motivated to practice these lifestyle modifiers which increase the overall quality of an individual's life.[29,30]

Limitations of not including female employees as the available sample size was limited as this was a cross-sectional study. Hence forms the future scope of the study. Further, with a larger sample, across different work atmosphere, age group, in both the genders, measures to increase their awareness and practice of yoga and exercise, correlating with hormonal analysis the effect of stress and de-stress activities on the above discussed parameters will be studied.

Conclusion

Work stress alters cardio metabolic parameters which are mainly due to autonomic and neuroendocrine alterations. Stressed professions though are aware of life style modifications fail to practice it regularly. This study indicates that while addressing the complications of job stress, a more aggressive working health policy has to be implemented at all work places. All professionals irrespective of their age should develop active and healthy lifestyles. This includes amongst others, the practice of yoga and regular exercise which soothe the mind and body to perform the duties disease-free effectively. Well planned awareness programs to prevent development of risk factors and complications in working employees should be encouraged.

References

1. Brunner EJ, Marmot MG. Social organisation, stress and health. In: Marmot MG, Wilkinson RG, eds. Social determinants of health. Oxford: Oxford University Press 2006;6–30.
2. Chandola T, Brunner E, Marmot M. Chronic stress at work and the metabolic syndrome:prospective study. BMJ 2006 Mar 4;332(7540):521–5.
3. Kang MG, Koh SB, Cha BS, et al. Association between job stress on heart rate variability and metabolic syndrome shipyard male workers. Yonsei Med J 2004;45(5):838–46.
4. World Health Organization. Occupational health http://www.who.int/occupational_health/topics/stressatwp/en/ Accessed 2019 November 15.
5. World Health Organization. Cardiovascular Diseases. Available online: https://www.who.int/health-topics/cardiovascular-diseases/ Accessed 2019 November 15.
6. Reddy KS. Cardiovascular Disease in Non-Western Countries. N Engl J Med. 2004 Jun 10;350(24):2438–40.
7. Gaziano TA. Reducing the growing burden of cardiovascular disease in the developing world. Health Aff (Millwood). 2007 Jan-Feb;26(1):13–24.
8. Mezue K. The increasing burden of hypertension in Nigeria - can a dietary salt reduction strategy change the trend? Perspect Public Health. 2014; 134(6):346–52.
9. Pyykkönen AJ, Räikkönen K, Tuomi T, et al. Stressful life events and the metabolic syndrome: the prevalence, prediction and prevention of diabetes (PPP)-Botnia Study. Diabetes Care 2010;33:378–84.
10. Emeny RT, Baumert J, Zierer A, et al. Job Strain, Inflammatory Biomarkers and Coronary Events in Healthy Workers of the MONICA/KORA Augsburg Case-Cohort Study. Psychother Psych Med 2011;61–A017.
11. Trudel X, Brisson C, Milot A. Job strain and masked hypertension. Psychosom Med. 2010 Oct;72(8):786–93.
12. Yu SF, Zhou WH, Jiang KY, et al. Effect of occupational stress on ambulatory blood pressure. Zhonghua Lao Dong Wei Sheng Zhi Ye Bing ZaZhi 2009;27:711–15.
13. Rose M. Good Deal, Bad Deal? Job Satisfaction in Occupations. Work Employment Society 2003;17(3):503–30.
14. Katyal S, Katyal R. Prevalence of Occupational Stress among Bankers. International Journal of Humanities and Social Science Invention 2013;2(4): 53–56.
15. Khattak JK, Khan MA, Haq AU, et al. Occupational stress and burnout in Pakistan's banking sector.

African Journal of Business Management 2011;5(3):810–17.

16. Esquirol Y, Bongard V, Mabile L, et al. Shift work and metabolic syndrome: respective impacts of job strain, physical activity, and dietary rhythms. ChronobiolInt 2009;26(3):544–59.
17. Innes KE, Vincent HK, Taylor AG. Chronic stress and insulin resistance-related indices of cardiovascular disease risk, part I: neurophysiological responses and pathological sequelae. AlternTher Health Med 2007;13(4):46–52.
18. Chandola T, Britton A, Brunner E, et al. Work stress and coronary heart disease:what are the mechanisms? Eur Heart J. 2008 Mar;29(5):640–8.
19. Kivimaki M, Leino-Arjas P, Luukkonen R, et al. Work stress and risk of cardiovascular mortality: prospective cohort study of industrial employees. BMJ. 2002 Oct 19;325(7369):857.
20. Vrijkotte TGM, van Doornen LJP, de Geus EJC. Effects of work stress on ambulatory blood pressure, heart rate, and heart rate variability. Hypertension 2000;35:880–86.
21. Brunner EJ, Hemingway H, Walker BR, et al. Adrenocortical, autonomic, and inflammatory causes of the metabolic syndrome: nested case-control study. Circulation 2002 Nov 19;106(21):2659–65.
22. Reaven GM, Lithell H, Landsberg L. Hypertension and associated metabolic abnormalities—the role of insulin resistance and the sympathoadrenal system. N Engl J Med. 1996 Feb 8;334(6):374–81.
23. Kuper H, Marmot M. Job strain, job demands, decision latitude, and the risk of coronary heart disease within the Whitehall II study. J Epidemiol Community Health 2003 Feb;57(2):147–53.
24. Qureshi GM, Seehar GM, Zardari MK, Pirzado ZA, Abbasi SA. Study of blood lipids, cortisol and haemodynamic variations under stress in male adults. J Ayub Med Coll Abbottabad 2009;21(1): 158–61.
25. Le Fur C, Romon M, Lebel P, Devos P, Lancry A, Guédon- Moreau L, et al. Influence of mental stress and circadian cycle on postprandial lipemia. Am J Clin Nutr. 1999 Aug;70(2):213–20.
26. Alfredsson L, Hammar N, Fransson E, de Faire U, Hallqvist J, Knutsson A, et al. Job strain and major risk factors for coronary heart diseases among employed males and females in a Swedish study on work, lipids and fibrinogen. Scand J Work Environ Health. 2002 Aug;28(4):238–48.
27. Sternberg EM. Neuroendocrine regulation of autoimmune and inflammatory disease. J Endocrinol. 2001 Jun;169(3):429–35.
28. Dobiášová M, Frohlich J. The plasma parameter log (TG/HDL) as an atherogenic index: correlation with lipoprotein particle size and esterification rate in apoB-lipoprotein-depleted plasma (FER HDL). Clinical Biochemistry 2001 Oct;34(7):583–8.
29. Smith JA, Greer T, Sheets T, Watson S. Is there more to yoga than exercise? Altern Ther Health Med 2011;17(3):22–9.
30. Woodyard C. Exploring the therapeutic effects of yoga and its ability to increase quality of life Int J Yoga 2011;4(2):49–54.

Original Research Article

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Comparison of Handgrip Strength in Male and Female Medical Students: A Cross Sectional Study

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Abstract

Introduction: Handgrip muscle strength is the maximum force developed during maximal voluntary contraction under a given set of conditions. It can be quantified by measuring the amount of static force that a person's hand can squeeze around a dynamometer. It is widely accepted that hand grip strength provides an objective index of functional integrity of the upper extremity. The present study aimed to compare maximal hand grip strength on the basis of gender. *Materials and Methods:* Present study was conducted on 120 healthy medical students in 18–24 years age group (60 male and 60 female) of DVVPF'S Medical College Maharashtra. The grip strength of dominant hand was measured twice at an interval of 1 minute, and higher reading recorded was considered to be the maximum hand grip strength for each student. *Results:* Data was analyzed by paired t test using SPSS. We noted that handgrip strength in male students was statistically significant ($p < 0.001$) as compared to female students. *Conclusion:* Hand grip muscle strength is more in male students as compared to female students in 18–24 years age group.

Keywords: Dynamometer; Female; Hand grip muscle strength; Male.

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Introduction

Handgrip muscle strength is the maximum force developed during maximal voluntary contraction under a given set of conditions.[1] It is the muscle strength and force that a person can generate with their hands and can be quantified by measuring the amount of static force that a person's hand can squeeze around a dynamometer.[2] Assessment of muscle strength and function plays a distinctive role is field of sports. It is often used as an indicator of the overall physical strength.

Handgrip strength testing has been extensively employed in a number of human movement related disciplines. Assessment of hand grip strength may be used in the investigations and follow up of patients with neuromuscular disorders.[3] It is also used as a functional index of nutritional status.[4] It is widely accepted that grip strength provides an objective index of functional integrity of the upper extremity.[5]

Aim

1. To measure hand grip muscle strength in healthy first year medical students.
2. To compare hand grip strength amongst male and female students.

Materials and methods

This is a cross sectional study conducted in research lab of Physiology department of Dr. Vithalrao Vikhe Patil Foundation's Medical College, Ahmednagar. By convenient sampling method, 120 healthy subjects from first year student population of our institute (60 male and 60 female) in the age group of 18–24 years participated in our study. Those

students suffering from any major illness in past or present, history of injury/nerve damage to upper limbs, history of any medications affecting motor function and those with musculoskeletal disorder were excluded from the study. The study was approved by the Institutional Ethical committee. Duration of study was 3 months.

At the beginning, the purpose and procedure of this study was explained to all the participants. Informed written consent was taken from all the subjects. Maximum handgrip strength was measured with handgrip dynamometer (INCO Ambala India). The most common and easiest method of assessment for grip strength is the use of handheld dynamometer. The subjects exerted grip strength using the handgrip device while sitting in chair with their elbow straight and close to the body.[6] The maximum grip strength of the dominant hand was measured twice within one minute time interval and greater value amongst the two was considered for further analysis. For Statistical analysis, Paired t test was applied using SPSS software. p – Value < 0.05 was considered statistically significant and < 0.001 was statistically considered to be highly significant.

Results

Present study included 120 healthy first year medical students. Amongst these, 60 were male and 60 were female students. Age and Physical characteristics like height (cm) and weight (Kg) were recorded for all the students.

Table 1: Mean values of physical characteristics in male and female medical students

Sr. No	Parameters	Male	Female
1	Age (years)	20.89 ± 2.50	20.32 ± 2.12
2	Height (cm)	167 ± 5.98	163 ± 5.10
3	Weight (Kg)	65 ± 9.10	61 ± 9.30

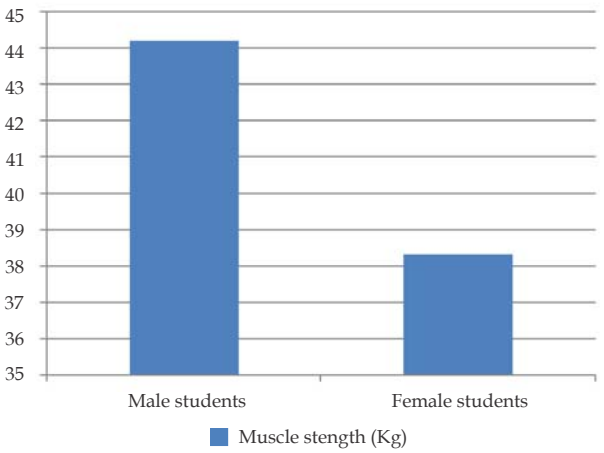
*p < 0.05 statistically significant **p < 0.001 statistically highly significant

Statistically, no significant difference was noted in mean values of age, height and weight between male and female medical students (Table 1).

Table 2: Maximum handgrip strength in male and female medical students

	Muscle strength in Kg (Mean ± SD)	p – value
Male students (n = 60)	44.21 ± 5.0	<0.001 (significant)**
Female students (n = 60)	38.30 ± 4.21	

*p < 0.05 statistically significant **p < 0.001 statistically highly significant



Graph 1 Comparison of muscle strength in male and female medical students

Discussion

Handgrip strength is one of the most commonly used tests for assessing muscular fitness in adults and it is also used as an important indicator of sports efficiency.

In present study, handgrip strength was compared amongst 60 male and 60 female medical students. This can provide baseline data/handgrip reference values in 18–24 years age group medical students.

In our study, Handgrip strength was noted to be statistically significant in male students as compared to female students. (Table 2 and Graph 1)

Christine described that, Females of age 16 years and older have muscle strength about two third less as compared to males of same age group. Gender differences in muscle strength may be because of observed variations in their physical activities of daily life.[7] In addition gender difference was found in maximum handgrip strength due to sex difference in muscle mass.

Leyk D et al. Showed that mean maximal hand grip strength in men was more than in women.[8] Heyward VH et al. noted that sex-related strength difference is more pronounced in the upper body due to muscle mass in men as compared to women.[9] Study conducted by Shah et al showed that mean handgrip strength in healthy adult males is more as compared to females.[10]

Similarly, Shyamal Koley and Shrikant Goud have also noted handgrip strength is maximum in males as compared to females.[11]

However, our study was restricted to medical students of 18–24 years of age group. Different age groups need to be studied.

Conclusion

We concluded that this study gives a baseline of normative data in sample population of MBBS students at DVVPF's Medical College Maharashtra. In the present study hand grip strength in male might have increased due to physiological maturation found in muscle growth.

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References

1. Sale DG. Testing strength and power In Mac Dougal JD, Wenger HA, Green HJ editors. Physiological testing of the high performance athlete. (IL) Human kinetics, Champaign III 1991. pp.21–75.
2. Massy-Westropp N, Gill TK, Taylor AW Bohannon R and Hill CL. Hand grip strength: Age and Gender stratified normative data in a population based stdy.BMC Research Notes 2011 April 14;4:127–33.
3. Wiles CM, Karni Y, Nicklin J. Laboratory testing of muscle function in the management of neuromuscular disease. J Neurol Neurosurg Psychiatry. 1990 May;53(5):384–387.
4. Jeejeebhoy KN. Nutritional assessment. Nutrition. 2000 Jul-Aug;16(7-8):585-90.
5. Mayers DB, Grennan DM, palmar DG. Handgrip function in patients with rheumatoid arthritis. Arch Phys. Med. Rehabil 1982;61:362–72.
6. Ramkumar B, Chittibabu B. Evaluation of dominant and non dominant hand grip strength among cricket, handball and Volleyball players. International Journal of physical education sports and yogic sciences 2012;2(1):41–3.
7. Hiroshi K, Demura S. Gender differences and laterality in maximal handgrip strength and controlled force exertion in young adults. Health journal 2011;3(11) 684–8.
8. Leyk D et al. Hand grip strength of young men, women and highly trained female athlets. Eur J Appl Physiol. 2007 Mar;99(4):415–21.
9. Heyward VH, Johannes-Ellis SM, Romer JF. Gender differences in strength. Res Quart 1986;57(2):154–9.
10. Shah S, Nahar P, Vaidya S, Salvi S. Upper limb muscle strength and endurance in chronic obstructive pulmonary diseases. Indian J med Res 2013;138(4):492–6.
11. Shyamal Koley and Srikanth Goud B. Correlations of Handgrip Strength With Selected Anthropometric Variables in Indian Junior and Senior Badminton Players. International Journal of Recent Scientific Research. 2016;7(4);10351–5.

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Hematocrit and its Correlation with Fasting Blood Sugar of Type II Diabetes Mellitus Patients: A Cross-Sectional Study

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Abstract

Introduction: Diabetes mellitus (DM) is a non-communicable disease with increasing prevalence worldwide. In DM, hematological indices such as WBC count, Hematocrit, platelet count, erythro-cyte aggregation, and erythrocyte deformability, are disturbed, which can lead to the development of inflammation and a tendency for coagulation and microvascular complications. So the current study was aimed at correlating RBC count, PCV and RDW with Fasting blood sugar of Type 2 Di-abetes patients. *Methodology:* A total of 96 participants (47 cases and 47 healthy controls) were selected using a systematic random sampling technique. Data is retrieved from the Medical Records Department. Parameters are lab investigation values that are already done on patients who came to DM WIMS. FBS was estimated using (Cobas Integra 400 plus) automated clinical chemistry analyzer and hema-tological parameters using a fully automated (Sysmex XT-1800i) analyzer. *Statistical analysis:* The statistical analysis was done using SPSS 15.0 version. After checking for normality Pearson's or Spearman correlation analysis is carried out to study the correlation. *Results:* PCV and RBC count was significantly higher in diabetics when compared to controls. Even though there was a negative correlation between PCV, RBC count and RDW with FBS (fasting blood sugar) in diabetics it was not statistically significant. *Conclusion:* The routine hematological profile checking of patients with T2DM may help to prevent complications associated with aberrations in hematological values.

Keywords: Fasting blood glucose; Red cell distribution width; PCV; type 2 diabetes mellitus.

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Introduction

Diabetes mellitus (DM) is a non-communicable disease with increasing prevalence worldwide.[1] Poorly controlled diabetes leads to various complications such as nephropathy, retinopathy, neuropathy and oxidative stress causing oxidative damage to tissues and cells.[2] Altered level of many hematological parameters such as red blood cells (RBCs), white blood cells (WBC), and the platelet function has been observed in patients with the diabetes.[3,4]

Many studies have advocated the importance of raised levels of WBC and RBC count in the diagnosis of metabolic syndrome.[5,6] Many epidemiological studies have also suggested a close relationship between hematological parameters and different components of metabolic syndrome.[7,8]

In DM, hematological indices such as WBC count, Hematocrit, platelet count, erythrocyte aggregation, and erythrocyte deformability, are disturbed, which can lead to the development of inflammation and a tendency for coagulation and micro vascular complications.[9] Researchers have been demonstrated that higher or even normal reference range of RDW (red cell distribution width) was strongly associated with increased risk of cardiovascular disease (CVD) events in middle-aged and older adults.[10,11]

Patients with T2DM have an increased risk of atherogenic dyslipidemia and cardiovascular disease (CVD) and the enhanced blood viscosity adversely affect the microcirculation in diabetes patients, leading to microangiopathy.[12] Also, increased levels of hematocrit and blood viscosity contribute to the development of insulin resistance and are independent predictors of type 2 diabetes.[13]

So the current study was aimed at correlating RBC count, PCV and RDW with Fasting blood sugar of Type 2 Diabetes patients.

Aims and Objectives

To determine the Hematocrit, RBC count and Red cell distribution width. To correlate Hematocrit, RBC count and Red cell distribution width with FBS of type II diabetic patient.

Materials and Methods

The study done was a comparative cross-sectional study at DM Wayanad Institute of Medical Sciences, Kerala, India. Data including Fasting blood sugar and hematological parameters like Platelet count and platelet distribution width of patients aged between 25 and 70 years were collected from hospital records of the above-mentioned institute. The duration of the study was from the Ist of January 2018 to the 31st of June. Parameters are lab investigation values those are already done on patients who came to DM WIMS central lab and procedure was done by collecting 2 ml Fasting blood sample and FBS was estimated using (Cobas Integra 400 plus) automated clinical chemistry analyzer. 2 ml of venous blood was collected for hematological parameters using fully automated (Sysmex XT-1800i) analyzer.

Sample size

Hematological parameters of 47 patients with FBS below 126 mg/dl are collected and considered as a control group.

Hematological parameters of 47 patients with FBS above or equal to 126 mg/dl are collected and considered as the study group. Age and sex were matched.

Inclusion criteria

a) Control group includes the data of patients whose FBS < 126 mg/dl and is apparently healthy individuals who had no previous history of chronic diseases.

b) The study group includes the data of patients whose FBS ≥ 126 mg/dl.

Exclusion criteria

Severely ill patients, infected patients, pregnant women, on antihypertensive treatment, on antiplatelet drugs, on statins, and who had other chronic diseases were excluded from the study.

Statistical Analysis

The sample size required to study the correlation is 90 at 5% level of significance and 80% power assuming the population correlation to be .3 (moderate correlation).

The statistical analysis was done using SPSS 15.0 version. After checking for normality Pearson's correlation analysis was carried out to study the correlation.

Ethical consideration

Ethical clearance was obtained from the Research and Ethical Committee of DM Wayanad Institute of Medical Sciences, Kerala, India. A permission letter was also taken from the Hospital Superintendent head for collecting data from the hospital record. For maintaining the confidentiality of the study participant's information, the data was stored in a password-protected computer of a principal investigator.

Results

Out of 47 diabetic patients 28 (59.57%) were females & 19 (40.42%) were males. PCV and RBC count was significantly higher in diabetics when compared to controls. RDW was marginally higher but statistically not significant shown in Table 1. Even though there was a negative correlation between PCV, RBC count and RDW with FBS (fasting blood sugar) in diabetics it was not statistically significant as shown in Table 2.

Table 1: Ematological parameters in study and control group

Variables	Diabetics (study group)	Nondiabetics (control group)	*t* value	*p* value
PCV (%)	38.61 ± 7.67	36.32 ± 5.22	1.68	0.04
RBC (millions/cumm)	4.74 ± 0.85	4.42 ± 0.76	1.88	0.03
RDW	14.70 ± 6.26	14.11 ± 2.61	0.59	0.27

$p < 0.05$ considered as significant

Table 2: Pearson's correlations(r) of Hematological parameters with FBS among T2DM patients and healthy controls

Variables	*r* value	*p* value
PCV (%)	-0.13	0.38
RBC(millions/cumm)	-0.11	0.46
RDW	-0.03	0.84

$p < 0.05$ considered as significant

Discussion

An increase in blood glucose levels is one of the factors that change the erythrocyte morphology. The extent of change in the shape of erythrocyte depends on the level of blood glucose level. All this affects the flow property of blood due to alteration and deformation.[14] The present study compares the hematological parameters between type II diabetics and nondiabetics. Our results showed that there is a significant increase in RBC count and PCV in diabetics when compared to nondiabetics this finding was similar to various previous studies.[15–17] Increased PCV, RBC count may be due to a variety of morphological changes exhibited by RBCs and compositional changes in plasma of diabetics.[18]

In contrast to this study, a study conducted on Chinese patients with T2DM reported that a decreased RBC count is associated with micro vascular complications.[19] Likewise, a study performed in Tobago (Caribbean) reported that RBC count, Haemoglobin concentration, and Hematocrit levels in T2DM patients are lower than in the control group.[20] The possible hypothesis for this difference might be that chronic hyperglycemia causes non-enzymatic glycosylation of RBC membrane proteins leading to accelerated aging of RBCs. A similar study on the middle-aged and elderly Chinese population in Taiwan also contradicts our findings as it is reported a reduced RBC count in patients with insulin resistance. Another study observed that diabetics are prone to anemia due to reduced kidney functions and decreased the production of erythropoietin hormone, which ultimately leads to decreased RBC count in the body.[21]

In our study even though there was a slight increase in RDW in diabetics than nondiabetics it was not statistically significant which us contradictory to other studies which showed a significant difference in RDW in diabetics and nondiabetics.[22–24] But few studies have shown results similar to our study.[25–27] Differences in study design and ethnic and cultural differences across the study populations may account for the variability of RDW across studies. High RDW indicates a high degree of anisocytosis which is associated with distortion and degradation of erythropoiesis reflecting chronic inflammation and an increased level of oxidative stress.[28]

The life span of red blood cells could be decreased in diabetes patients. So, RBC's are affected by various disturbances in the hematopoietic milieu. These disturbances lead to elevated internal viscosity and increased membrane rigidity in these blood cells. So, the RBC count is calculated as an increase.[29]

Conclusion

Hematological parameters like RBC count, RDW and PCV can be a predictor of good glycemic control diabetics. Unfortunately, our study didn't show any significant correlation between these parameters and FBS, this may be due to small sample size. The routine hematological profile checking of patients with T2DM may help to prevent complications associated with aberrations in hematological values.

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Conflict of interest: None declared

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References

1. Whiting DR, Guariguata L, Weil C, et al. IDF Diabetes Atlas: Global estimates of the prevalence of diabetes for 2011 and 2030. Diabetes Res Clin Pract 2011;94(3):311–21.
2. Comazzi S, Spagnolo V, Bonfanti U. Erythrocyte changes in canine diabetes mellitus: in vitro effects of hyperglycemia and ketoacidosis. J Comp Clin Path 2004;12(4):199–205.
3. Mbata Christian A, Adebayo A, Chinyere N, et al. Some Haematological Parameters in Diabetic Patients in Port Harcourt Nigeria. AJMS 2015;3(2):2348–7186.
4. Mirza S, Hossain M, Mathews C, et al. Type 2-diabetes is associated with elevated levels of TNF-alpha, IL-6 and adiponectin and low levels of leptin in a population of Mexican American: a cross-sectional study. Cytokine 2012;57(1):136–42.

5. Barbieri M, Ragno E, Benvenuti E, et al. New aspects of insulin resistance syndrome: impact on haematological parameters. Diabetologia. 2001 Oct;44(10):1232–7.
6. Grundy SM. Hypertriglyceridemia, insulin resistance, and the metabolic syndrome. Am J Cardiol. 1999 May 13;83(9B):25F–29F.
7. Friedman GD, Teskawa I, Grimm RH, et al. The leukocyte count: correlates and relationship to coronary risks factors: the CARDIA study. Int J Epidemiol 1990;19:889–93.
8. Shukla DK, Chandra KP, Pawah AK. Study of hematological indices in patients with diabetes mellitus and hypertensive diabetes mellitus. International Journal of Medicine Research 2016;1(4):28–31.
9. Demirtas L, Degirmenci H, Akbas EM, et al. Association of hematological indices with diabetes, impaired glucose regulation and microvascular complications of diabetes. Int J ClinExp Med. 2015;8(7):11420–5. PMID: 26379958, PMCID: PMC4565341.
10. Anderson JL, Ronnow BS, horne BD, et al. Usefulness of a complete blood count-derived risk score to predict incident mortality in patients with suspected cardiovascular disease. Am J Cardiol. 2007 Jan 15;99(2):169–74.
11. Ani C, Ovbiagele B. elevated red blood cell distribution width predicts mortality in persons with known stroke. J Neurol Sci. 2009 Feb 15;277(1-2):103–8.
12. Cho YI, Mooney MP, Cho DJ. Hemorheological disorders in diabetes mellitus. J Diabetes Sci Technol. 2008;2(6):1130-8. doi: 10.1177/193229680800200622. PMID: 19885302, PMCID: PMC2769810.
13. Tamariz LJ, Young JH, Pankow JS, et al. Blood viscosity and hematocrit as risk factors for type 2 diabetes mellitus: the atherosclerosis risk in communities (ARIC) study. Am J Epidemiol. 2008 Nov 15;168(10):1153-60. doi: 10.1093/aje/kwn243. PMID: 18931370, PMCID: PMC2581671.
14. Singh M, Shin S. Changes in erythrocyte aggregation and deformability in diabetes mellitus. Indian Journal of Experimental Biology 2009;47(1):7–15.
15. Chen LK, Ming-Hsien L, Zhi-Jun C, et al. Association of insulin resistance and hematologic parameters: study of a middle-aged and elderly Chinese population in Taiwan. Chin Med Assoc 2006;69(6):248–53.
16. Charles LE, Fekedulegn D, McCall T, et al. Obesity, white blood cell counts, and platelet counts among police officers. Obesity 2007;15(11):2846–54.
17. Farhangi MA, Keshavarz SA, Eshraghian M, et al. White blood cell count in women: relation to inflammatory biomarkers, haematological profiles, visceral adiposity, and other cardiovascular risk factors. J Health PopulNutr 2013;31(1):58–64.
18. Marcinkowska-Gapinska A, Kowal PA. Blood fluidity and thermography in patients with diabetes mellitus and coronary artery disease in comparison to the healthy subject. Clin Hemorheol Microcir. 2006;35:473.
19. Wang ZS, Song ZC, Bai JH, et al. Red blood cell count as an indicator of microvascular complications in Chinese patients with type 2 diabetes mellitus. Vasc Health Risk Manag. 2013;9:237–43.
20. Ezenwaka CE, Jones-Le Cointe A, et al. Anemia and kidney dysfunction in Caribbean type 2 diabetic patients. Cardiovasc Diabetol 2008;7:25.
21. Cawood TJ, Buckley U, Murray A, et al. Prevalence of anemia in patients with diabetes mellitus. Ir J Med Sci 2006;175:25–27.
22. Biadgo B, Melku M, Abebe SM, Abebe M. Hematological indices and their correlation with fasting blood glucose level and anthropometric measurements in type 2 diabetes mellitus patients in Gondar, Northwest Ethiopia. Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy 2016;9:91–99.
23. Malandrino N, Wu WC, Taveira TH, et al. Association between red blood cell distribution width and macrovascular and microvascular complications in diabetes. Diabetologia. 2012 Jan;55(1):226-35.
24. Sherif H, Ramadan N, Radwan M, et al. Red cell distribution width as a marker of inflammation in type 2 diabetes mellitus. Life Sci J. 2013;10(3):1501–7.
25. Cakir L, Aktas G, Enginyurt O, Cakir S. Mean platelet volume increases in type 2 diabetes mellitus independent of HbA1c level. Acta Medica Mediterranea. 2014;30:425.
26. Dada OA, Uche E, Akinbami A. The relationship between red blood cell distribution width and blood pressure in patients with type 2 diabetes mellitus in Lagos, Nigeria. J Blood Med. 2014 Sep 19;5:185–9.
27. Evans TC, Jehle D. The red blood cell distribution width. J Emerg Med. 1991;9 Suppl 1:71–4.
28. Md. Sadikuj Jaman et al. Diabetes and red blood cell parameters Annals of Clinical Endocrinology and Metabolism 2018;1:1–9.

Review Article

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Neurochemical Regulation of Veins

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Abstract

Neural control of the venous tone is provided by the adrenergic innervation. In some venous territories, a cholinergic innervation or a non-adrenergic non-cholinergic one were identified. Hormones are also involved in the control of venous tone. Catecholamines and angiotensin II are most important venoconstrictors. The relaxing action of estrogen and progesterone was also studied insome venous territories. Vasopressin, somatostatin, insulin, and thyroid hormones have actions in pathophysiologic states. Local control of venous tone includes: metabolic regulation; humoral control; ions and endothelium-dependent regulation through vasodilators and vasoconstrictors. Veins exhibit a less pronounced endothelium-dependent control and a different response profile to endogenous vasoactive substances than arteries. Other factors such as reactive oxygen species, cytokines, fibrinogen, thrombin, oxidized LDL (low density lipoprotein) and vasostatins also play role in venous regulation. Myogenic control of veins is less important than arterial one. Pharmacological agents can also modulate venous tone. Innervation, hormones, metabolic factors, ionic environment, humoral factors, endothelium-derived vasoactive factors, and even reactive oxygen species and cytokines act directly on venous smooth muscle and endothelial cells. In addition, to their vasoconstrictor or vasodilator actions, some of these factors may be involved in other important physiological (vascular hypertrophy, intimal hyperplasia, and venular permeability) and pathological mechanisms such as venous graft pathology or varicose veins.

Key words: Adrenergic innervation; Catecholamines; Endothelium derived vasoactive factor; Nitric oxide; Reactive oxygen species; LDL.

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Introduction

Neural control of the venous tone is provided by the adrenergic innervation. In some venous territories, a cholinergic innervation or a non-adrenergic non-cholinergic one were identified. Hormones are also involved in the control of venous tone.[1] Catecholamines and angiotensin II are most important venoconstrictors.[2,3] The relaxing action of estrogen and progesterone was also studied in some venous territories.[4] Vasopressin, somatostatin, insulin, and thyroid hormones have actions in pathophysiologic states.[5,6] Local control of venous tone includes: metabolic regulation; humoral control (which involves vasoconstrictor substances, vasodilator factors and factors with both constrictor and relaxing actions); ions; endothelium-dependent regulation (venous endothelial cells produce vasodilators – Nitric oxide, Prostacyclin (PGI_{2}) and Endothelium Derived Hyperpolarising Factor (EDHF) – and also vasoconstrictors –Endothelin-1 (ET-1), Thrombaxane A2 (TxA2) and Prostaglandin H2 (PGH2).[7,8] Veins exhibit a less pronounced endothelium-dependent control and a different response profile to endogenous vasoactive substances than arteries.[9–11] Other factors such as reactive oxygen species, cytokines, fibrinogen, thrombin, oxidized LDL and vasostatins also

play role in venous regulation.[12,13] Myogenic control of veins is less important than arterial one. Pharmacological agents can also modulate venous tone.[14]

Neural control

Neuralcontrol of veins is through sympathetic control, vasomotor center and neurotransmitters released by the sympathetic nervous fibers i.e norepinephrine, adenosine triphosphate, neuropeptide Y and calcitonin gene related peptide.[2,3]

Norepinephrine

Adrenergic varicosities contain the enzymatic apparatus necessary for Norepinephrine (NE) biosynthesis, storage and release. NE is synthesized through hydroxylation of tyrosine to desoxy phenylalanine (DOPA), followed by DOPA decarboxylation to dopamine. The final step involves the 3-hydroxylation of dopamine to norepinephrine by dopamine 3-hydroxylase present in storage vesicles in adrenergic varicosities. NE enters the synaptic cleft and activates the adrenoceptors on the vascular cells. Removal of norepinephrine is by uptake in the nerve endings, degraded by the intraneuronal monoamine oxidase (MAO), diffusion to the capillaries, uptake by the effector cells and enzymatic degradation by MAO and catechol-O-methyl transferase (COMT) to inactive metabolites.[2,3] NE activates post junctional receptors mainly α1 and α2 for control of vascular tone.[1] α2 A *and* α2 Bfor arterial contraction and α2 C for venous vasoconstriction.[1]

Adenosine triphosphate (ATP)

It is released from the vesicles in the sympathetic nerve terminals, acts locally and post-junctionally being hydrolyzed by ecto ATP- ase.Induces an inward current via ligand-gated channels and causes both vasoconstriction and vasodilation. Purinoreceptors (P2X) receptors are responsible for the vasoconstrictor response and Purinorceptors (P2Y) receptors mediate the relaxing response.[15]

Neuropeptide Y (NPY)

Acts on receptors Y1 and Y2 subtypes. Y1 receptor is located mainly post-junctionally and are involved in vasoconstriction. Y2 receptors were detected at pre- and post-junctional sites and mediate the pre-junctional inhibition of norepinephrine release. [16,17]

Calcitonin gene-related peptide (CGRP)

It is contained in both adrenergic and NANC (non-adrenergic non-cholinergic) fibers distributed to different components of the cardiovascular system.[18,19]

Cholinergic Innervation

Studies have revealed two separate cholinergic systems in the vascular wall. Endothelial cells represent the intrinsic, intimal system; regulator of basal vascular tone. Perivascular autonomic nerve fibers represent the extrinsic, adventitial system. Acetylcholine (ACh) can contract and relax vascular tissue. Vasodilation being endothelium-dependent and mediated via muscarinic M3 receptors.[20,21]

Non-Adrenergic Non-Cholinergic Innervation (NANC)

ATP, VIP (vasoactive intestinal peptide), CGRP, and NO were designed as neurotransmitters released by the NANC fibers.[20,21]

Hormonal control

Hormones responsible for maintenance of venous tone are Catecholamines, Angiotensin, Estrogen and progesterone. Other hormones like (vasopressin, somatostatin, insulin, thyroid hormones)also contribute.

Catecholamines are synthesized and released from the chromaffin cells of the adrenal medulla in response to emotional stress. The most important catecholamine secreted is epinephrine.[2,3] α2 and β adrenoceptors exist on endothelial cells and contribute to the regulation of vasomotor tone. α2 adrenoceptors stimulates the release of NO.[22]

Angiotensins play an important role inrenin angiotensin system (RAS). All components of RAS except renin, are produced in several tissues, including vessel wall.[23] Angiotensinogen mRNA and protein was identified in vascular smooth muscle, endothelium, and perivascular fat. ACE was found in the adventitia, endothelial cells and vascular smooth muscle cells in culture. Angiotensin II exerts two types of effects: vasoconstriction and aldosterone release. It also causes pressor effect at very low doses.[23]

Estrogens mediates its beneficial actions via

nuclear receptors, named ERα and ERβ which decrease vascular tone, has cytoprotective action on the vascular wall, increase of HDL cholesterol, decrease of LDL cholesterol, causes inhibition of LDL-oxidation and decreases plasminogen and fibrinogen levels. The relaxing effect is caused by:increased synthesis of endothelium-dependent relaxing factors (NO, PGl2, EDHF)[4], decreased production of vasoconstrictor factors, such as endothelin[24] or superoxide anion [25], decreased calcium entry into vascular smooth muscle cells[26] and decreased α-adrenergic responsiveness in venous smooth muscle cells, thereby decreasing venous tone and contributing to the pathogenesis of varicosities[27]. There is enhancement of venous compliance after ingestion of oral estrogen-containing contraceptives and occurrence of varicose veins.[28,29]

Progesterone produce a dose-dependent relaxation mediated by a receptor-activated cAMP mechanism.[30]

Other Hormones which have a role to play include vasopressin, somatostatin, insulin and thyroid hormones. The constrictor action of vasopressin seems to be lower in veins, compared to arterial preparations, due to a low population or sensitivity of receptors sites for this peptide.[5,31] Somatostatin presents both venoconstrictor and venodilator actions. The dilator effect of somatostatin was reported in the venous portal tree. A combination of somatostatin, vasopressin, and nitroglycerin seems to be very effective in patients with portal hypertension[6,32]. Insulinplays a role in the regulation of vascular tone in human vessels, including venous tone by attenuating vasoconstrictor responses to pressor agonists and increasing the vasorelaxing effect. Mechanism of insulin-induced vasodilation are decreased vascular sensitivity to α-adrenergic agonists and an enhanced sensitivity to β-adrenoceptor agonists[33]; stimulation of nitric oxide release from endothelial cells[34,35]; activation of ATP-dependent potassium channels.[36,37] Insulin has Long term actions in vascular smooth muscle cells, such as increase of Na^+-K^+-ATP-ase and Na^+-Ca^{2+} exchanger.[38,39] Thyroid Hormones have positive inotropic and vasodilator effects.[40]

Local control

Local control of veins is achieved by Metabolic, Humoral, Ions and Endothelium derived factors. PO_2 represents an important factor in local metabolic regulation venous tone. Chronic hypoxia impairs venous smooth muscle contractility.[41] Acute hypoxia increases venous smooth muscle contractility. Anoxic constriction is mediated by ET-1 release.[7] Lowered pH and increased PCO_2 decreases smooth muscle contractility.[42] Lactateis produced by the venous wall, even in normoxic conditions; in case of local hypoxia, lactate release is elevated, because LDH (lactate dehydrogenase) subunit composition in venous tissue is better suited for transformation of pyruvate into lactate [43]. In pre-eclampsia, absence of lactate induced dilatation of placental vessels may contribute to the fetal complications due to impaired blood flow and vasospasm.[44] Adenosine is well known for itspowerful relaxing effects in vascular beds. P1-purinoceptors present an agonist potency and their activation induces changes in intracellular cAMP mechanism. ATP and ADP (adenosine diphosphate) are components of platelets and erythrocytes, and can also be released from endothelial cells and smooth muscle cells.[8] Activation of P2-purinoceptors can produce either vasoconstriction or vasodilation. Endothelial P2 receptors usually mediate relaxation via production of NO.

Humoral Control involves vasoconstrictors asangiotensin II, PGF2α, Thromboxane A2, bradykinin, histamine (H1), serotonin and vasodilators as ANP (atrial natriuretic peptide), BNP (brain natriuretic peptide), CNP (c-type natriuretic peptide), PGI2, PGE2, Bradykinin, Histamine (H2), Substance P. Angiotensins exist in vascular tissue including venous wall.[45–47] Angiotensin-converting enzyme (ACE) activity was found in the endothelium and mast cells in the adventitia of the vessel wall.[48] Angiotensin I caused a marked venoconstriction. The contractile response was inhibited by AT1 receptor antagonist losartan.[49] Angiotensin II potentiates the venoconstrictorresponse via release of NE from the nerve terminals.[50,51] Ang II, Ang III, and Ang IV produced concentration-dependent contractions. The ACE inhibitor enalaprilat augmented endothelium-dependent relaxations.[52] In varicose veins, the reactivity of venous smooth muscle to angiotensin II is impaired.[53]

Natriuretic Peptidesas ANP, BNP and CNP are encoded by genes with similar structure. They exert their affects through NP receptors. Two of them stimulating guanylate cyclase, whereas the third appears mostly as a clearance receptor. ANP (mainly synthesized within the atrial myocytes), BNP (predominantly produced in atria and brain) and CNP from the vascular endothelium is involved in the control of vascular tone.[54–56]

Venous tissue is capable of producing

eicosanoids. Both endothelium and smooth muscle take part in production of both prostacyclin and thromboxane.[57] There are two forms of COX: the constitutive form COX-I, located in the endothelial layer, a cytokine-inducible isoform, COX-2, in vascular smooth muscle by IL-1β, TNF-α, bacterial LPS (lipo-polysaccharides), growth factors, and phorbol esters. Venous smooth muscle expressed higher amounts of COX-2 than the arterial one.[58] Bradykinin can be derived from a number of different sources, including endothelium and are considered local hormones acting in a autocrine-paracrine manner. ACE inactivates bradykinin and thereby blunt sendothelium-dependent relaxations to this peptide.[59] Histamine presents venoconstrictor and venodilator actions. The H1 receptors mediate their effects via products of inositol phospholipid hydrolysis and increasing intracellular calcium. H2 receptors are coupled via Gs protein to adenylate cyclase and stimulate cAMP formation, inducing a decrease of Ca^{2+}[60]. Serotonin is mainly derived from platelets acton specific 5-HT receptors, may induce both contraction and relaxation[61–63]. The relaxing action is due to inhibition of NE release from sympathetic nerveendings, by direct vascular smooth muscle relaxation, and by release of EDRF[64]. Substance Pis an endothelium-dependent vasodilator neurokinin effect being mediated by NKI receptors.[65,66]

Ions such as Sodium, Potassium, Calcium, Magnesium, and Chloride also have a role to play in local control of venous tone.Sodium distribution is regulated by [67] Na^+- K^+ pump.[68] Na^+/Ca^{2+} exchanger transports Ca^{2+} out of the cell at expense of Na influx, which then will be removed by the Na^+- K^+ pump. Sodium also takes part in- Na^+-H^+ exchanger, Na^+/K/2Cl, and Fast sodium channels. Potassium presents important role through direct and indirect mechanisms, through the electrogenic Na^+- K^+ pump, by alterations in cell membrane permeability and bymodulation of norepinephrine release by sympathetic nerves.[69] A moderate K^+ elevation (5-10 mM) produced relaxation, while a pronounced increase, above 15 mM induced venous contraction.[70] Different types of potassium channels are Ca^{2+} activated K^+ channels (BK Ca^{2+}), ATP sensitive K^+ channels (KATP) andvoltage gated K^+ channels (KV). Calcium plays a major role in the initiation of contraction, which depends on the increase of myoplasmic concentration of Ca^{2+}Calcium influx from the extracellular pool occurs through receptor-activated, voltage-dependent, or stretch-activated calcium channels. Release of Ca^{2+} from the sarcoplasmic reticulum is linked to binding of the second messenger IP3 (ionositol triphosphate).[71]

Magnesium represents a natural calcium antagonist; it inhibits voltage-dependent, receptor-dependent, and leak channels.[72,73] Reduction of extracellular Mg^{2+} level increases Ca^{2+} influx with subsequent elevation in tension and reactivity to vasoconstrictors.[72–74]

Chloride transport across cell membrane is elicited via:co-transporters, such as Na^+-Cl, K^+-Cl, and Na^+-K^+-2Cl [75], exchangers, such as Na^+-HCO_3/ Cl and HCO_3/Cl; andchloride channels.[76]

Endothelium-Derived Factors are also responsible forshort term regulation ofvenous tone. It produces vasodilator substances, such as nitric oxide (NO), prostacyclin (PGI2) and EDHF andvasoconstrictor factors, such as endothelin-1, thromboxane A2 and prostaglandin H2.

Nitric oxide (NO) is formed from the guanidine-nitrogen terminal of L-arginine by endothelial NO synthase (eNOS). Itdiffuses into the underlying vascular smooth muscle to mediate vascular relaxation by a cGMPdependent process.[11,78] Pathophysiological use of NO are in Venous graft spasm, Primary varicose veins and, Raynaud's disease. Nitrovasodilators including Glyceryl trinitrite (GTN) and other organic nitrates, are prodrugs, acting by the release of nitric oxide.[79] The venoselectivity of these NO-donating drugs presents both advantages (efficacy in heart failure and angina therapy) and disadvantages (postural hypotension, headache).[80] Endothelium-derived hyperpolarizing factors (EDHFs) induces relaxation by reducing the open probability of voltage -dependent calcium channelsand by the activation of potassium channels on the vascular smooth muscle.[10,81] Important stimuli for ET-1 release are, hypoxia and low shear stress, vasoactive substances (epinephrine, angiotensin II, vasopressin, bradykinin), growth factors (transforming growth factor)[82], and cytokines (lL-1).[83]

Other factors involved in the modulation of venous tone are Reactive oxygen species, Cytokines, Fibrinogen, thrombin, Oxidized Low-Density Lipoprotein (ox-LDL) and Vasostatins.

Reactive Oxygen Species (ROS) are generated mainly at sites of inflammation and injury. They function as signalling molecules. At higher concentration, they can endanger all cellular macromolecules.[12] Main ROS are Superoxide Anion, Hydrogen peroxide and OH (hydroxyl ions). Major ROS producing systems include[84]: NADH/NADPH oxidases, xanthine oxidase, lipo-oxygenases; cyclooxygenase; P-450 monooxygenases; the enzymes of mitochondrial oxidation, NO synthase. Défense against ROS are superoxide dismutases

(SOD), catalasesand peroxidases.[85,86] Cytokines likeIL-1 may influence venous contractility by inducing NO synthase. Fibrinogen endothelium-dependent relaxation effect is reversed at higher concentrations of fibrinogen.[13]

Myogenic control

Venous intrinsic tone play an insignificant role in overall ability of veins to regulate vascular capacity.[14]

Pharmacological aspects

Include Venodilator Drugs, Calcium antagonists, Nitro-vasodilators, ACE inhibitors. Venotonic Drugs are derived from Aesculus hippocastannum (Horse chestnut), Ruscus aculeatus (Butcher's broom), Centella Asiatica (Gotu Kola), Bioflavinoids:Diosmin and Hesperidin.

Conclusion

Innervation, hormones, metabolic factors, ionic environment, humoral factors, endothelium-derived vasoactive factors, and even reactive oxygen species and cytokines act directly on venous smooth muscle and endothelial cells. In addition, to their vasoconstrictor or vasodilator actions, some of these factors may be involved in other important physiological mechanisms, suchas vascular hypertrophy, intimal hyperplasia, and venular permeability.

References

1. Civantos Calzada B, Aleixandre de Artinano A: Alpha-adrenoceptor subtypes. Pharmacol Res Pharmacol Res. 2001 Sep;44(3):19–208.
2. Vanhoutte PM: The role of the systemic veins: an update. Phlebology 1988; 3(suppl.l): 13–25.
3. Weiner N, Molinoff PB: "Catecholamines" In Basic Neurochemistry: Molecular, Cellular and Medical Aspects, 4th edition. Siegel GJ et aI., ed. Raven Press NY, 1989; 233–51.
4. Foegh ML, Ramwell PW. Cardiovascular effects of estrogen: implications of the discovery of the estrogen receptor subtype beta. Curr Opin Nephr Hypertens 1998;7:83–89.
5. Martinez MC, Aldasoro M, Vila M, et al. Responses to vasopressin and desmopressin of human cerebral arteries. J Pharmacol Exp Ther 1994;270:622–27.
6. Rice TL. Treatment of esophageal varices. Clin Pharm. 1989 Feb;8(2):122–31.
7. Vanhoutte PM, LUscherTF, Gr: Endothelium dependent contractions. Blood vessels 1991;28:74–83.
8. Ralevic V, Bumstock G: Roles of PrPurinoceptors in the Cardiovascular System. Circulation 1991;84: I-JO.
9. Feletou M, Vanhoutte PM: The alternative: EDHF. J Mol Cell Cardiol. 1999 Jan;31(1):15–22.
10. Corriu C, Feletou M, Canet E, et al. Endothelium-derived factors and hypepolarizations of isolated carotid artery of the guinea pig. Br J Pharmacol 1996;119:959–64.
11. Murad E, Waldman S, Molina C, et al. Regulation and role of guanylate cyclase-cyclic GMP in vascular relaxation. Prog Clin Bioi Res 1987; 249:65–76.
12. Lum H, Roebuck KA. Oxidant stress and endothelial cell dysfunction. Am J Physiol Cell Physiol. 2001 Apr;280(4):C719–41.
13. Hicks RCJ, Golledge J, Mir-Hasseine R,et al. Vasoactive effects offibrinogen on saphenous vein. Nature 1996;379:818–20.
14. Vanhoutte PM, Janssens WJ: Local control of venous function. Microvasc Res 1978;16:144–46.
15. Flavahan NA, Vanhoutte PM. Sympathetic purinergic vasoconstncnon and thermosensitivity in acanine cutaneous vein. J Pharm Exp Ther 1986;239:784–89.
16. Kuriyama H, Kitamura K, Nabata H. Pharmacological and Physiopathological significance of ion channels and factors that modulate them in vascular tissues. Pharm Rev 1995;47:387–573.
17. Franco-Cereceda A, Liska J. Neuropeptide Y YI receptors in vascular pharmacology. Eur J Pharmacol. 1998 May 15;349(1):1–14.
18. Preibisz 11: Calcitonin Gene-Related Peptide and regulation of human cardiovascular homeostasis. Am Hypertens 1993 May;6(5 Pt 1):434–50.
19. Bell D, McDermott BJ. Calcitonin Gene-related Peptide in the Cardiovascular System: Characterization of Receptor populations and Their (patho) Physiological Significance. Pharm Rev 1996; 48:254–82.
20. Eglen RM, Hedge SS, Watson N. Muscarinic receptor subtypes and smooth muscle function. Pharm Rev 1996;48:532–56.
21. Caulfield MP, Birdsall N1: International Union of Pharmacology. XVII. Classification of Muscarinic Acetylcholine Receptors. Pharm Rev 1998 Jun;50(2):279-90.
22. Haefeli WE, Srivastava N, Kongpatanakul S, et al. Lack of role of endothelium-derived relaxing factor in effects of alpha-adrenergic agonists in cutaneous

veins in humans. Am Physiol 1993;264:364–69.

23. Danserl AH. Local Renin-angiotensin systems. Moll Cell Biochem 1996;157:211-16.
24. Orirno A, Inoue S, Ouchi Y, et al. Vascular smooth muscle cells possess estrogen receptor and respond to estrogen. Ann NY Acad Sci 1995;748:592-94.
25. ArnallF, Tack 1, BesombreslP, Pipy B, Negre-Salvayre A: Nitric oxide and superoxide anion production during endothelial cell proliferation. Am J Physiol 1996;271:C1521–26.
26. Collins P, Meale CM, Rosano Me. Oestrogen as a calcium channel blocker. Eur Heart J 1996; 17(supplD):27–31.
27. Jilma B, Wolzt H, Wagner OF, et al. In vivo study of the effect of exogenous estradiol on c-adrenoceptor responsiveness of human veins. J Cardiovasc Pharmacol 1994;23:859–63.
28. Goodrich SM, Wood JE. Peropheral venous distensibility and velocity ofvenous blood flow during pregnancy or during oral contraceptive therapy. Am J ObstetGynecol 1964;90:740–44.
29. Cohen 1: Insufficienceveineuse et contraception orale. Rev Fr GynecolObstet 1991;86:187–89.
30. Omar HA, Ramirez R, Gibson M. Properties of a progesterone-induced relaxation in human placental arteries and veins. J Clin Endocrinol Metab. 1995 Feb;80(2):370–3.
31. Aldasoro M, Medina P, Vila M, et al. Endothelium dependent relaxation of human saphenous veins in response to vasopressin and desmopressin. 1Vase Surg 1997;25:696–703.
32. Burroughs AK, McCormick PA. Randomized trial for variceal bleeding. Z Gastroenterol 1988;26:24–35.
33. Creager MA, Liang C, Coffman JD. Beta-adrenergic mediated vasodilator response to insulin in the human forearm. JPharmacol Exp Ther 1985;235:709–14.
34. Steinberg HM, Brechtel G, Johnson A,et al. Insulin-mediated skeletal muscle vasodilation is nitric oxide dependent.J Clin Invest. 1994 Sep;94(3):1172–1179.
35. Scherrer U, Randin D, Vollenweider P, et al. Nitric oxide release accounts for insulin effects in humans. 1Clin Invest 1995;94:2511–15.
36. McKay MK, Hester RL. Role of nitric oxide, adenosine and ATP-sensitive potassium channels in insulin-induced vasodilation. Hypertension 1996 Aug;28(2):202-8.
37. Izhar U, Hasdai D, Richardson DM, et al. Insulin and insulin-like growth factor-I cause vasorelaxation in human vessels in vitro. Coron Artery Dis 2000;11:69–76.
38. Tirupattur PR, Ram JL, Standley PR, et al. Regulation of Na+'K+-ATPase gene expression by insulin in vascular smooth muscle cells. Am J Hypertens. 1993 Jul;6(7 Pt 1):626-9.
39. Ozttirk Y, MelihAltan V, Yildizoglu-Ari N. Effects of Experimental Diabetes and Insulin on Smooth Muscle Functions. Pharm Rev 1996 Mar;48(1):69-112.
40. Krasner U, Wendling WW, Cooper SC, et al. Direct effects of triiodothyronine on human internal mammary artery and saphenous veins. J Cardiothorac Vase Anesth 1997 Jun;11(4):463–6.
41. Zhao Y, Packer CS, Rhoades RA. Chronic hypoxia impairs pulmonary venous smooth muscle contraction. Respir Physiol. 1995 Apr;100(1):75–82.
42. Vanhoutte PM, Janssens WJ. Local control of venous function,. Microvasc Res 1978;16:144–46.
43. Malmqvist U, Amer A, Uvelius B. Lactate dehydrogenase activity and isoform distribution in normal and hypertrophic smooth muscle tissue from the rat. Pflugers Arch. 1991 Oct;419(3-4):230–4.
44. Figueroa R, Martinez E, Fayngersh RP, et al. Absence of relaxation to lactate in human placental vessels of pregnancies with severe preeclampsia. Am J Obstet Gynecol. 1995 Dec;173(6):1800-6.
45. Mizuno K, Niimura S, Tani SM, et al. Direct prooffor local generation and release of angiotensin II in peripheral human vascular tissue. Am 1 Hypertens 1991;4(suppl):S67–72.
46. Belz GG, Berrmann J, Schloos J, KleinBloesem CH. The effect of oral clazapril and prazosin on the constrictor effects oflocally infused angiotensin I and noradrenaline in human hand veins. Br J Clin Pharmacol 1989;28:608–11.
47. Paul M, Wagner J, Dzau VJ. Gene expression of the renin-angiotensin system in human tissues. Quantitative analysis by the polymerase chain reaction. J Clin Invest. 1993 May;91(5):2058–64.
48. Borland JA, Chester AH, Morrison KA, et al. Alternative pathways of angiotensin II production in the human saphenous vein. Br J Pharmacol. 1998 Oct;125(3):423–8.
49. Borland JA, Chester AH, Crabbe S, et al. Differential action of angiotensin II and activity of angiotensin-converting enzyme in human bypass grafts. J Thorac Cardiovasc Surg 1998;116:206–12.
50. Webb DJ, Benjamin N, Cockcroft JR, et al. Augmentation of sympathetic venoconstriction by angiotensin II in human dorsal hand veins. Am J Hypertens 1989;2:721–23.
51. Molderings OJ, Likungu L, Hentrich F, et al. Facilitatory presynaptic angiotensin receptors on the sympathetic nerves of the human saphenous vein and pulmonary artery. Potential involvement in beta-adrenoceptor-rnediated facilitation of noradrenaline release. NaunynSchmiedebergs Arch Pharmacol 1988;338:228–33.
52. Yang Z, Arnet U, Von Segesser L, et al. Different effects of angiotensin-converting enzyme inhibition in human arteries and veins. J Cardiovasc Pharmacol 1993;22 (suppI5):S17–22.

53. Drouet L, Baudin B, Baumann FC, Caen lP. Serum angiotensin-converting enzyme. An endothelial cell marker. 1Lab Clin Med 1988;112:450–57.
54. Curtis SB, Hewitt J, Yakubovitz S. et al. Somatostatin receptor subtype expression and function in human vascular tissue. Am J Physiol Heart Circ Physiol. 2000 Jun;278(6):H1815–22.
55. Vesely DL. Atrial Natriuretic Peptides. Kidney: A Current Survey of World Literature, 1994;3:4–8.
56. Banks M, Wei CM, Kim CH, et al. Mechanism of relaxations to C type natriuretic peptide in veins. Am J Physiol 1996;217:H1907–11.
57. Nadasy GL, Szekacs B, Juhasz I, et al. Role of endothelium, oxygen and ionic milieu in the prostacyclin and thromboxane production of rat aortic tissue slices. Acta Physiol Hung 1991;78:77–87.
58. Bishop-Bailey D, Pepper JR, Larkin SW, et al. Differential induction of cyclooxigenase-2 in human arterial and venous smooth muscle. Role of endogenous prostanoids. ArterioscIerThrombVaseBioi 1998;18(10):1655–61.
59. Yang Z, LUscherTF. Basic cellular mechanisms of coronary bypass graft disease. EurHeartJ 1993;14 (suppl 1):193–97.
60. Monos E, Berczi V, Nadasy G. Local Control of veins: Biomechanical, Metabolic and Humoral Aspects, Physiological Reviews 1995;75(3):616–19.
61. Vashisht R, Sian M, Sharp EJ, et al. Serotonin-induced contractility in human saphenous vein is inhibited by naftidrofuryl. Br J Surg 1992 Nov;79(11):1154–56.
62. Yang Z, LUscherTF. Basic cellular mechanisms of coronary bypass graft disease. EurHeartJ 1993;14 (suppl 1):193-97.
63. Barber DA, Rubin JW, Zumbro GL, et al. The use of methylene blue as an extravascular surgical marker impairs vascular responses of human saphenous vein. J Thorac Cardiovasc Surg. 1995;109:21–29.
64. Monos E, Berczi V, Nadasy G. Local Control of veins: Biomechanical, Metabolic and Humoral Aspects. Physiological Reviews 1995 Jul;75(3):611–66.
65. Edvinsson L, Hakanson R, Steen S, et al. Innervation of human omental arteries and veins and vasomotor response to noradrenaline, neuropeptide Y, substance P and vasoactive intestinal peptide. Regul Pept 1985;12:67–79.
66. Bodelsson G, Sandstrom K, Wallerstedt SM, et al. Effects of propofol on substance P-induced relaxation in isolated human omental arteries and veins. Eur J Anaesthesiol. 2000 Dec;17(12):720–8.
67. Somlyo AP, Somlyo AV, Shuman H. Electron probe analysis of vascular smooth muscle. Composition of mitochondria, nuclei, and cytoplasm.J Cell Biol. 1979 May;81(2):316–35.
68. Allen lC, Kahn AM, Navran S. Na+-K+-ATP-ase in vascular smooth muscle. Am J Physiol. 1986 Apr;250(4 Pt 1):C536–9.
69. Sparks HV. Effect of local metabolic factors on vascular smooth muscle. In Handbook of Physiology. The Cardiovascular System. Vascular Smooth Muscle. Bethesda, MD: Am Physiol Soc 1980;11:475–513.
70. Nguyen DH, Voss U, Brecht K. The effect of potassium on isolated bovine facial and human saphenous veins. Experientia Basel 1980;36:975–76.
71. Karaki H, Ozaki H, Hori M, et al. Calcium movements, distribution, and functions in smooth muscle. harmacol Rev. 1997 Jun;49(2):157–230.
72. Altura BM, Altura BT. Magnesium ions and contraction of vascular smooth muscles relationship to some vascular diseases. Fed Proc. 1981 Oct;40(12):2672–9.
73. Altura BM, Altura BT. Interactions of Mg and K on blood vessels-aspects in view of hypertension. Magnesium 1984;3(4-6):175–94.
74. Szabo C, Berczi V, Schneider F, et al. Role of endothelium in the response of the vein wall to magnesium withdrawal. Pflugers Arch 1992; 420:140–45.
75. Brock TA, Brugnara C, Canessa M, et al. Bradykinin and vasopressin stimulate Na+-K+-Cl- cotransport in cultured endothelial cells.Am J Physiol. 1986 Jun;250(6 Pt 1):C888–95.
76. Matsumoto T, Tomita T. Intracellular alkalinization caused by chloride removal in the smooth muscle of guinea-pig vena cava. Jpn J Physiol. 1993;43(1):67–73.
77. Wang Q, Hogg RC, Large WA. Properties of spontaneous inward currents recorded insmooth muscle cells isolated from the rabbit portal vein. J PhysiolLond 1992;451(1):525–37.
78. Ignarro LJ. Heme-dependent activation of guanilate-cyclase by nitric oxide: a nivel signal transduction mechanism. Blood Vessels. 1991;28(1-3):67–73.
79. Feelisch M. Biotransformation to nitric oxide of organic nitrates - comparison to other nitrovasodilators. Eur Heart J. 1993 Nov;14 Suppl I:123–32.
80. Henderson AH. Endothelium in control. Br Heart J 1991;65:116–25.
81. Corriu C, Feletou M, Canet E, et al. Inhibitors of the cytochrome p. 450 monooxygenase and endothelium-dependent hyperpolarizations in the guinea pig isolated carotid artery. Br J Pharmacol 1996;107:607–10.
82. Escobales N, Longo E, Cragoe EJ, et al. Osmotic activation of NaH exchange in human endothelial cells. Am J Physiol. 1990 Oct;259(4 Pt 1):C640–6.

83. Rubanyi GM, Polokoff MA. Endothelins: Molecular Biology, Biochemistry, Pharmacology, Physiology, and Pathophysiology. Pharmacol Rev. 1994 Sep;46(3):325–415.

84. Griendling KK, Sorescu D, Lassegue B, et al. Modulation of Protein-kinase activity and gene expression by reactive oxygen species and their role in vascular physiology and pathophysiology. Arterioscler Thromb Vasc Biol. 2000 Oct;20(10):2175–83.

85. Fridovich I. Fundamental aspects of reactive oxygen species, or what's the matter with oxygen? Ann N Y Acad Sci. 1999;893:13–8.

86. Lum H, Roebuck KA. Oxidant stress and endothelial cell dysfunction. Am J Physiol Cell Physiol. 2001 Apr;280(4):C719–41.

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[1] Flink H, Tegelberg Å, Thörn M, Lagerlöf F. Effect of oral iron supplementation on unstimulated salivary flow rate: A randomized, double-blind, placebo-controlled trial. J Oral Pathol Med 2006; 35: 540-7.

[2] Twetman S, Axelsson S, Dahlgren H, Holm AK, Källestål C, Lagerlöf F, *et al*. Caries-preventive effect of fluoride toothpaste: A systematic review. Acta Odontol Scand 2003; 61: 347-55.

Article in supplement or special issue

[3] Fleischer W, Reimer K. Povidone iodine antisepsis. State of the art. Dermatology 1997; 195 Suppl 2: 3-9.

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[4] American Academy of Periodontology. Sonic and ultrasonic scalers in periodontics. J Periodontol 2000; 71: 1792-801.

Unpublished article

[5] Garoushi S, Lassila LV, Tezvergil A, Vallittu PK. Static and fatigue compression test for particulate filler composite resin with fiber-reinforced composite substructure. Dent Mater 2006.

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[6] Hosmer D, Lemeshow S. Applied logistic regression, 2nd edn. New York: Wiley-Interscience; 2000.

Chapter in book

[7] Nauntofte B, Tenovuo J, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O,

Kidd EAM, editors. Dental caries: The disease and its clinical management. Oxford: Blackwell Munksgaard; 2003. p. 7-27.

No author given

[8] World Health Organization. Oral health surveys - basic methods, 4th edn. Geneva: World Health Organization; 1997.

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[9] National Statistics Online—Trends in suicide by method in England and Wales, 1979-2001. www.statistics.gov.uk/downloads/theme_health/HSQ20.pdf (accessed Jan 24, 2005): 7-18. Only verified references against the original documents should be cited. Authors are responsible for the accuracy and completeness of their references and for correct text citation. The number of reference should be kept limited to 20 in case of major communications and 10 for short communications.

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