Original Research Article

Comparison of cardiovascular responses with proprioceptive neuromuscular facilitation stretching on pectorals and hamstrings with valsalva maneuver in young adults

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Abstract

Introduction: According to the American College of Sports Medicine, flexibility is an important component of good physical fitness and health. Therefore, for physical activity programs, muscles stretching exercise is an important component. Sedentary or less flexible subjects may perform the VM during stretching exercises due to difficulty in reaching and sustaining extreme ranges of motion.
Materials and methods: 40 participants of age group 18-25 years having either hamstrings or pectorals tightness were included in quasi experimental study by convenient sampling technique. 20 subjects were included in each group: group A hamstrings tightness and group B pectorals tightness. 3 reps of stretching were given with 15 sec rest after each rep. Heart rate and Blood pressure was measured.
Results: PNF stretching of hamstrings with VM shows significant increase in HR, SBP and DBP compared to PNF stretching of pectorals.
Conclusion: PNF stretching of hamstrings with VM shows significant increase in HR, SBP and DBP compared to PNF stretching of pectorals.

Key words

Diastolic Blood Pressure, Heart Rate, Proprioceptive Neuromuscular Stretching, Systolic Blood Pressure, Valsalva maneuver.

Introduction

According to the American College of Sports Medicine [1], flexibility is an important component of good physical fitness and health. Therefore, for physical activity programs, muscles stretching exercise is an important component [2]. Studies have demonstrated that stretching prior to physical activity leads to intense muscle contraction and may affect muscle [2, 3] and yet, muscle stretching is still usually performed prior to physical activity [3-6].

Farinatti, et al. [7] suggested that stretching exercises performed at low flexibility levels can affect the autonomic nervous system of subjects, leading to increased sympathetic activation. McCully [8] suggested that because of the structural changes that are caused by stretching [9], the oxidative capacity and oxygen supply of muscles may be impaired.

Another issue to be considered is the increase in cardiac preload due to the Valsalva maneuver (VM) [7]. During the VM there is increase in thoracic pressure and expiratory effort which reduce the venous return and cardiac output, provokes baroreflex responses that increase the BP [8]. Sedentary or less flexible subjects may perform the VM during stretching exercises due to difficulty in reaching and sustaining extreme ranges of motion. Also, maintaining an adequate workload position for several seconds may demand sustained static contractions of reasonable intensity. Despite this, there are very few studies on acute cardiovascular responses to flexibility exercises with VM.

Physiological changes with stretching are first seen in large muscle groups like hamstrings and pectorals. Hence the purpose of this study was to evaluate in young healthy subjects with poor flexibility level, Heart Rate (HR), and BP before and after PNF stretching with VM involving large muscle groups. We hypothesized that stretching exercises involving larger muscle masses and the VM would induce greater cardiovascular responses. Additionally, post-exercise hypotension was expected to occur, at least after stretching larger muscle groups.

Materials and methods

Subjects

Young healthy adults with age group 18- 25 years having either hamstrings or pectorals tightness were included in the experimental study at tertiary care hospital.

Exclusion criteria

- Hypertensive individuals
- Orthopedic conditions
- Neurological conditions
- Athletes
- Individuals undergoing fitness training.

By convenient sampling technique 40 subjects who satisfied inclusion and exclusion criteria were included in the study. Oral explanation of procedure was given to subjects and informed consent was signed prior to study.

Experimental design

All of the subjects were instructed to avoid caffeine, alcohol, and any kind of physical exercise in the 24 hr prior to the exercise session. Subjects were divided in to 2 groups on the basis of tightness of muscle: hamstring tightness and pectorals tightness. Each group comprised of 20 subjects.

On the first day subjects were evaluated for tightness of hamstrings and pectorals muscles and were divided into groups accordingly. They were made familiarized with the techniques to be performed. Each subject has to perform a set PNF stretching exercise. It consists of 3 repetitions of stretching exercise, each with 6 sec of isometric contraction followed by 24 sec of sustained stretching. VM was performed in last second of each repetition and 15 seconds rest was given between each repetition [9].

On second day, prior to stretching session subjects rested for 10 min. Baseline HR and BP were measured after that. PNF stretching of hamstrings was performed with subject in supine with hip flexion and knee extension. So for that base line data was measured in supine position. PNF stretching of pectorals was given in high sitting position with horizontal flexion of shoulders forming 90° angle with the trunk and elbows flexed such that the hands touches the back of head. Base line data for pectorals was taken in high sitting position (**Figure – 1**, **Figure – 2**).

Figure - 1: Hamstrings stretching.

Figure - 2: Pectorals stretching.

Immediately after completion of a set of stretching exercises, HR and BP were measured. To maintain the ecological validity of the results, the measurements were conducted within 30 sec after stretching. Measurements were measured again after 1 min and 3 min.

Blood pressure and heart rate measurement

BP was measured with the sphygmomanometer and heart rate was measured manually at radial artery for 15s. Outcome measures were heart rate systolic blood pressure and diastolic blood pressure.

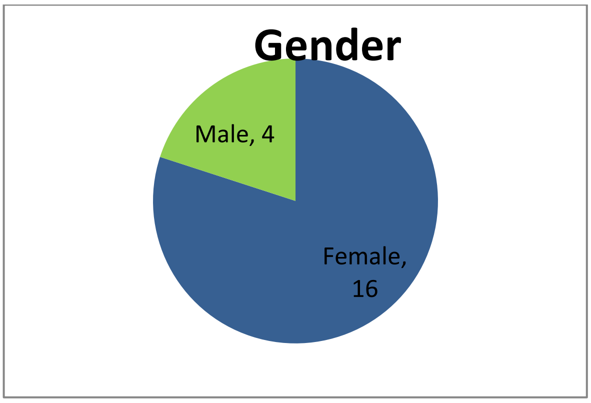
Statistical analysis

Data were not normally distributed so Non Parametric test Wilcoxon Signed Rank test was used for within group analysis and Mann-Whitney 'U' test was used for between group analysis with level of significance p<0.005 with SPSS version 16.

Results

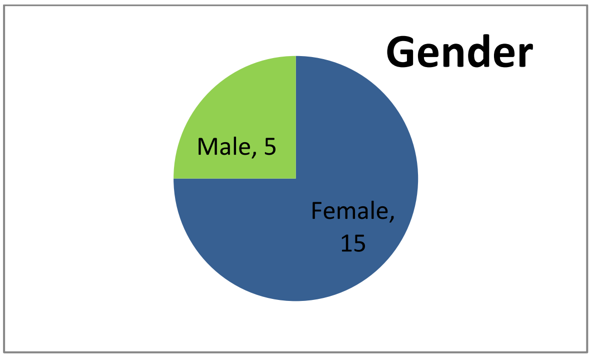
40 participants were included in study 20 in each group. In Hams stretch group 16 females and 4 males while pectorals stretch had 15 females and 5 males (**Graph – 1**, **Graph – 2**).

Graph - 1: Gender distribution in Hams stretch group.



According to the analysis done, for within group analysis for group A, HR post immediately, SBP post 1 min and post 3 min are significant with p values as 0.000, 0.000 and 0.005 respectively and for group B, HR post immediately, post 1 min, SBP post immediately, post 1min and DBP post immediately and post 1 min are significant with p values 0.004, 0.002, 0.000, 0.003, 0.000 and 0.421 respectively (**Table – 1**, **Table – 2**).

Graph - 2: Gender distribution in pectorals stretch group.



For inter group analysis, group A showed significant difference in HR, SBP and DBP than group B as per **Table - 3**.

Discussion

Present study aimed to observe the cardiovascular responses to multiple sets of stretching exercises involving different muscles masses and VM. Primary findings revealed that there was significant increase in HR, SBP and DBP.

Table - 1: Within Group analysis: Wilcoxon Signed Rank Test - Group A (Hamstrings).

Statistics	HR			SBP			DBP		
	Post-Pre	Post1-Pre	Post3-Pre	Post-Pre	Post1-Pre	Post3-Pre	Post-Pre	Post1-Pre	Post3-Pre
W	**-3.764**	-0.595	-1.028	-2.486	**-3.599**	**-2.793**	-2.686	-0.913	-0.538
p	**0.000**	0.552	0.304	0.013	**0.000**	**0.005**	0.007	0.361	0.591

Post 1=Post 1 min; Post 3=Post 3 min

Table - 2: Within Group analysis: Wilcoxon Signed Rank Test - Group B (Pectorals).

Statistics	HR			SBP			DBP		
	Post-Pre	Post1-Pre	Post3-Pre	Post-Pre	Post1-Pre	Post3-Pre	Post-Pre	Post1-Pre	Post3-Pre
W	**-2.858**	**-3.033**	-2.632	**-3.491**	**-2.948**	-2.963	**-3.544**	-0.805	-2.484
p	**0.004**	**0.002**	0.008	**0.000**	**0.003**	0.007	**0.000**	0.421	0.130

Post 1=Post 1 min; Post 3=Post 3 min

The sustained muscle tension may have favored the HR and SBP increases due to the activation of muscle and tendon mechanoreceptors. Mechanical stimuli such as tendon stretch and light, non-noxious probing of receptive fields have been known for some time to stimulate group III muscle afferents [10-12]. In addition, tendon stretch has been shown to reflexly increase arterial pressure and heart rate in animals [13-15] as well as heart rate in humans [16]. Moreover, compression of muscles in humans, a mechanical stimulus, has also been shown to increase reflexly arterial pressure and heart rate [17, 18]. Tendon stretch in animals was reported to not increase metabolism in the muscle [14], and in humans it was reported not to be painful [16].

Mechanical stress caused by flexibility training can affect hemodynamic responses [19]. Stretched muscle fibers activate mechanoreceptors, which elicit cardiovascular adjustments through parasympathetic withdrawal and sympathetic activation [20]. In this context, previous studies with animal models have demonstrated that the muscle tension produced while stretching increases cardiovascular responses, particularly the heart rate (HR) [21]. Other studies have demonstrated that small muscle fiber receptors also react to stretching in humans [22], with a significant impact on the initial HR acceleration. In addition, sustained contractions of large muscle groups increase the peripheral vascular resistance and therefore influence the cardiac output and blood pressure (BP) [23].

Table - 3: Inter group analysis: Mann-Whitney U Test.

Outcome Measures		Statistics	
		U	p
HR	Pre	123.500	0.036
	Post	193.500	0.859
	Post1	188.000	0.742
	Post3	141.000	0.108
SBP	Pre	158.000	0.164
	Post	173.000	0.946
	Post1	147.500	1.000
	Post3	158.000	0.721
DBP	Pre	150.000	0.164
	Post	197.500	0.946
	Post1	200.000	1.000
	Post3	187.000	0.721

Post1=Post 1min; Post3=Post 3min

Another issue to be considered is the increase in cardiac preload due to the Valsalva maneuver (VM) [7]. Depending on the body position, it is not unusual for respiration to be blocked while stretching. The expiratory effort and increase in thoracic pressure during the VM reduce the venous return and cardiac output, which provokes baroreflex responses that can increase the BP [8].

Present study, showed significant increase in cardiovascular responses on stretching with VM in both the groups. The execution of the VM may enhance the intra-abdominal and intra-thoracic pressures, which would influence the both SBP and DBP. The combination of prolonged static contractions and the VM is known to amplify the increase in BP and the pressure load on the heart [24, 25]. Sedentary or less flexible subjects may perform the VM during stretching exercises due to difficulty in reaching and sustaining extreme ranges of motion. In addition, maintaining an adequate workload position for several seconds may demand sustained static (isometric) contractions of reasonable intensity.

The physiological mechanisms underlying cardiovascular responses during flexibility exercises are not completely known. In the present study, the maximal range of motion was reached and held passively, and the stretched muscle was certainly contracted because of the muscle spindle reflex. The sustained muscle tension may have favored the HR and SBP increases due to the activation of muscle and tendon mechanoreceptors [26, 27]. The sustained static contraction combined with stretching to the maximal range of motion may also have occluded muscle vessels, leading to an increase in the SBP [28, 29]. In simultaneous muscle stretching and contraction, type III fibers and metaboreceptor activation may induce vagal inhibition and baroreflex stimulation and contribute to an increase in the overall cardiovascular response [30, 31].

In the present study, the HR was assessed during exercise sessions performed with a static component sustained for 30 s, with four repetitions. Because the subjects had low flexibility levels, the contraction intensity can be considered as at least moderate, representing a non-negligible muscle workload. Additionally, the HR and BP in each set increased proportionally to the duration of the muscle contraction. This issue evokes the importance of controlling the stimulation duration to prevent undesirable cardiac stress.

On comparison between two groups, hamstrings stretching showed significant increase in responses compared to pectorals. The mass of the stretched muscle influenced the HR, regardless of the presence of the VM. The SBP was influenced by the VM, at least in the exercises that engaged a larger muscle mass. The influence of respiratory blocking on the cardiovascular response during flexibility exercises is greater when larger muscle groups are stretched. Higginbotham MB [32] proposed that the VM combined with static exercises may significantly increase the BP in normotensive subjects. It is also possible that the VM interferes with the transition from rest to exercise, perhaps contributing to the BP increase [33]. Our findings are consistent with this idea; the combination of incorporating a larger muscle mass and the VM produced greater increases in the BP, regardless the number of sets done.

Conclusion

PNF stretching of hamstrings with VM shows significant increase in HR, SBP and DBP compared to PNF stretching of pectorals.

Limitation

The VM intensity was not controlled, so the expiratory pressure was set individually. The force applied during isometric contractions in the PNF stretching protocol was not measured.

Further recommendation

Study can be carried out with large sample size and in cardiac patients to see the effect.

References

1. American College of Sports Medicine Position Stand (ACSM). Quantity and quality of exercise for developing and maintain cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: Guidance for prescribing exercise. Med Sci Sport Exerc., 2011; 43: 1334-1359.
2. Rubini EC, Costa ALL, Gomes PSC. The effects of stretching on strength performance. Sport Med., 2007; 37: 213-224.
3. Costa PB, Graves BS, Whitehurst M, Jacobs PL. The acute effects of different durations of static stretching on dynamic balance performance. J Strength Cond Res., 2009; 23: 141-147.
4. Franco BL, Signorelli GR, Trajano GS, Costa PB, Oliveira CG. Acute effects of three different stretching protocols on the Wingate test performance. J Sport Sci Med., 2012; 11: 1-7.
5. Gomes TM, Simão ER, Marques MC, Costa PB, Novaes JS. Acute Effects of Two Different Stretching Methods on Local Muscular Endurance Performance. J Strength Cond Res., 2011; 25: 745-752.
6. Wong PL, Lau PW, Mao de W, Wu YY, Behm DG, Wislof U. Three days of static stretching within a warm-up does not affect repeated-sprint ability in youth soccer players. J Strength Cond Res., 2011; 25: 838-845.
7. Farinatti PTV, Brandão C, Soares PPS, Duarte AFA. Acute Effects of Stretching Exercise on the Heart Rate Variability in Subjects with Low Flexibility Levels. J Strength Cond Res., 2011a; 25: 1579-1585.
8. McCully KK. The Influence of Passive Stretch on Muscle Oxygen Saturation. Adv Exp Med Biol., 2010; 662: 317-322.
9. Kubo K, Kanehisa H, Kawakami Y, Fukunaga T. Influence of static stretching on viscoelastic properties of human tendon structures in vivo. J Appl Physiol., 2001; 90: 520-52.
10. Korner PI, Tonkin AM, Uther JB. Reflex and mechanical circulatory effects of graded Valsalva maneuvers in normal man. J Appl Physiol., 1976; 40: 434-40.
11. Khayat RN, Przybylowski T, Meyer KC, Skatrud JB, Morgan BJ. Role of sensory input from the lungs in control of muscle sympathetic nerve activity during and after apnea in humans. J Appl Physiol., 2004; 97: 635-40.
12. Silva GC, et al. Effects of Proprioceptive Neuromuscular Facilitation Stretching and Static Stretching on Cardiovascular Responses. Journal of Exercise Physiology online, 2013; 16(1): 117-125.
13. Kaufman MP, Longhurst JC, Rybicki KJ, Wallach JH, Mitchell JH. Effects of static muscular contraction on impulse activity of groups III and IV afferents in cats. J Appl Physiol., 1983; 55: 105–112.
14. Mense S, Stahnke M. Responses in muscle afferent fibers of slow conduction velocity to contractions and ischemia in the cat. J Physiol., 1983; 342: 383–397.

15. Paintal AS. Functional analysis of group III afferent fibres of mammalian muscles. J Physiol., 1960; 152: 250–270.
16. Hayes SG, Kaufman MP. Gadolinium attenuates exercise pressor reflex in cats. Am J Physiol Heart Circ Physiol., 2001; 280: H2153–H2161.
17. Stebbins CL, Brown B, Levin D, Longhurst JC. Reflex effect of skeletal muscle mechanoreceptor stimulation on the cardiovascular system. J Appl Physiol., 1988; 65: 1539–1547.
18. Wilson LB, Wall PT, Pawelczyk JA, Matsukawa K. Cardiorespiratory and phrenic nerve responses to graded muscle stretch in anesthetized cats. Respir Physiol., 1994; 98: 251–266.
19. Gladwell VF, Coote JH. Heart rate at the onset of muscle contraction and during passive muscle stretch in humans: a role for mechanoreceptors. J Physiol., 2002; 540: 1095–1102.
20. Gaffney FA, Thal ER, Taylor WF, Bastian BC, Weigelt JA, Atkins JM, Blomqvist GG. Hemodynamic effects of medical anti-shock trousers (MAST garment). J Trauma, 1981; 21: 931–937.
21. Williamson JW, Mitchell JH, Olesen HL, Raven PB, Secher NH. Reflex increase in blood pressure induced by leg compression in man. J Physiol., 1994; 475: 351–357.
22. Rassier DE, Macintosh BR, Herzog W. Length dependence of active force production in skeletal muscle. J Appl Physiol., 1999; 86: 1445-57.
23. Drew RC, Bell MP, White MJ. Modulation of spontaneous baroreflex control of heart rate and indexes of vagal tone by passive calf muscle stretch during graded metaboreflex activation in humans. J Appl Physiol., 2008; 104: 716-23.
24. Yamamoto K, Kawada T, Kamiya A, Takaki H, Shishido T, Sunagawa K, et al. Muscle mechanoreflex augments arterial baroreflex-mediated dynamic sympathetic response to carotid sinus pressure. Am J Physiol Heart Circ Physiol., 2008; 295: 1081-9.
25. Gladwell VF, Fletcher J, Patel N, Elvidge LJ, Lloyd D, Chowdhary S, et al. The influence of small fibre muscle mechanoreceptors on the cardiac vagus in humans. J Physiol., 2005; 567: 713-21.
26. MacDonald J, MacDougall J, Hogben C. The effects of exercising muscle mass on post exercise hypotension. J Hum Hypertens., 2000; 14: 317-20.
27. MacDougall JD. Blood pressure responses to resistive, static and dynamic exercise. In: Fletcher GF, editor. Cardiovascular Response to exercise. Mount Kisco: Futura Publishing Company; 1994, p. 155-173.
28. Longhurst JC, Stebbins CL. The power athlete. Cardiol Clin., 1997; 15: 413-29.
29. Hayes SG, Kindig AE, Kaufman MP. Comparison between the effect of static contraction and tendon stretch on the discharge of group III and IV muscle afferents. J Appl Physiol., 2005; 99: 1891-6.
30. Drew RC, McIntyre DB, Ring C, White MJ. Local metabolite accumulation augments passive muscle stretch-induced modulation of carotidcardiac but not carotidovasomotor baroreflex sensitivity in man. Exp Physiol., 2008; 93: 1044-57.
31. Fisher JP, Bell MP, White MJ. Cardiovascular responses to human calf muscle stretch during varying levels of muscle metaboreflex activation. Exp Physiol., 2005; 90: 773-81.
32. Higginbotham MB. Cardiac performance during sub maximal and maximal exercise in health persons. Heart Failure, 1988; 4: 68-76.
33. Kaufman MP, Hayes SG. The exercise pressor reflex. Clin Auton Res., 2002; 12: 429-39.