Effect of Valsalva Maneuver on Cardiovascular Reflexes

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الخلاصة:

تعتبر عملية الفاالسالفا (VM) Valsalva maneuver من الاختبارات الهامة لتقييم كفاءة الجهاز العصبي اللاإرادي (autonomic nervous system) ففي هذه العملية يتم اختبار كفاءة المستقبلات الخاصة بتغيرات ضغط الدم (Baroreceptor activity) لأنّ هذه المستقبلات تقوم بعملها الطبيعي عن طريق الجهاز العصبي الودي (Sympathetic nervous system) و اللاودي (Parasympathetic nervous system). وبهذا فإنّ إجراء اختبار الفالسالفا (VM) يستخدم لمعرفة التغيرات الحاصلة في ديناميكية القلب والأوعية الدموية (Hemodynamics of cardiovascular system) لغرض تقييم كفاءة القلب والأوعية الدموية (Cardiovascular integrity). تتكون عملية الفالسالفا من أربع مراحل يتم خلالها قياس ما يلي:

1- قياس كمية الدم التي يضخها القلب في النبضة الواحدة (SV) Stroke Volume وذلك باستعمال جهاز الإيكو(Echocardiography).

2- قياس عدد ضربات القلب(HR)Heart rate خلال العملية باستعمال جهاز تخطيط القلبECG.

3- يمكن حساب كمية الدم التي يضخها القلب في الدقيقة الواحدة Cardiac output (CO) من النقطتين 1و2 أعلاه باستعمال القانون (CO = SV × HR)، أي إنّ كمية الدم التي يضخها القلب في الدقيقة الواحدة تساوي عدد ضربات القلب في الدقيقة الواحدة مضروب في كمية الدم التي يضخها القلب في النبضة الواحدة.

4- قياس ضغط الدم (BP) Blood pressure خلال العملية باستعمال جهاز ضغط الدم الزئبقي (Mercury sphygmomanometer).

5- يمكن حساب المقاومة المحيطية للأوعية الدموية Peripheral vascular resistance (PVR) باستعمال القانون PVR = BP/CO (المقاومة المحيطية للأوعية الدموية تساوي ضغط الدم مقسوماً على كمية الدم التي يضخها القلب في الدقيقة الواحدة).

تم إجراء هذه الدراسة على سبعين شخصاً من الأصحاء، أعمارهم (20-40) سنة بمعدل 27.31 ± 5.28. وقد تم دراسة التغيرات الحاصلة في ديناميكية القلب والأوعية الدموية خلال عملية الفالسالفا أي التغيرات الحاصلة في (CO, SV, HR, BP and PVR).

6- ما يتعلق بالتغيرات في مختلف مراحل الفالسالفا فقد وجد إن هنالك زيادة مفاجئة في ضغط الدم مع تباطؤ في النبض مع بداية الجهد (المرحلة الأولى) خلال المرحلة الثانية (مرحلة الجهد). هنالك انخفاض في كمية الدم التي تضخ من القلب وانخفاض في ضغط الدم إلى أوطأ نقطة، والذي يؤدي إلى تحفيز الجهاز العصبي

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السمبثاوي، وبالتالي زيادة في عدد ضربات القلب، وزيادة في ضغط الدم (العالي والواطئ ومعدل ضغط الدم SBP, DBP and MBP), لهذا السبب فان المرحلة الثانية تقسم إلى: المرحلة الثانية المبكرة والمرحلة الثانية المتأخرة.

عنــد تحريــر الجهــد لعمليــة الفالســالفا، هنالــك انخفــاض ســريع لكميــة الــدم التي يضخها القلب مع انخفاض شديد في الضغط (المرحلة الثالثة)، لهذا فإنّ المرحلة الثانية المبكـــــــرة والمرحلـــــــة الثالثـــــــة لـــــــم تضـــــــمن فـــــــي هـــــــذه الدراسة، لأنّ تغيرات ضغط الدم تحتاج إلى الطرق المباشرة التي تسجل التغيرات الآنية خلال هاتين المرحلتين (invasive technique). أما المرحلة الرابعة، فإنّها تبدأ بثوان قليلة بعد إخراج الهواء، حيث يعود ضخ القلب للدم إلى مستواه الطبيعي مع زيادة كبيرة في ضغط الدم (العالي و الواطئ ومعدل ضغط الدم) مع تباطؤ في النبض كرد فعل لهذه الزيادة في الضغط. لهذا فإنّ كمية الدم التي يضخها القلب في الدقيقة الواحدة تنخفض لأنها تحسب من المعادلة: CO = SV × HR، أما المقاومة المحيطة (PVR) فأنها تزداد لأنها تحسب من المعادلة: PVR = BP/CO.

Abstract:

Valsalva maneuver (VM) is one of the most important tests used to investigate the integrity of the autonomic nervous system (ANS), it can be used to assess the baroreflex activity since the baroreceptors innervated by both sympathetic and parasympathetic nervous systems. Therefore (VM) used to investigate the changes in the hemodynamic variables in order to assess the integrity of cardiovascular system.

The procedure of (VM) involves four phases through these phases the following measurements take place:

1. Measurement of stroke volume (SV) which is the volume of blood pumped from the heart by each beat using echocardiographic technique.

2. Heart rate (HR) is recorded by ECG in order to count the number of heart beats per each minutes.

3. Cardiac output (CO) which is the volume of blood pumped from the heart per each minute can be calculated by the equation $CO = HR \times SV$ from the above points (1,2).

4. Blood pressure measurement during (VM) by using mercury sphygmomanometer by which measurement of SBP, DBP and MBP.

5. Peripheral vascular resistance (PVR) can be calculated from the equation $PVR = BP/CO$.

This study was carried out on seventy normal healthy subjects, their age range (20-40 years) with mean $\pm$ SD is (27.31 $\pm$ 5.28years).

In this study a totally non-invasive techniques were used during all phases of VM.

Concerning the responses in different phases of VM we found that there is sudden increase of BP with reflex bradycardia at the onset of straining(phase1). During phase2 (straining phase) there is significant

reduction of SV and decreasing of BP to the low point lead to sympathetic stimulation and reflex tachycardia and increment in BP(systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean blood pressure (MBP), so phase 2 can be divided in to phase 2E and phase 2L. At release of strain of VM, there is transient reduction of SV and BP (phase3), phase 2E and phase 3 were not included in this study as BP changes need to be measured by invasive technique. few seconds after release of strain, the SV return to premaneuver level with BP "over shoot" (increased SBP, DBP and MBP) and peripheral vascular resistance (PVR) also increased as it is calculated from this equation (PVR = MBP/CO), and there is reflex bradycardia so cardiac output(CO) is decreased (phase4).

Introduction:

Valsalva maneuver (VM):

   The (VM) is a common every day life and occurs during lifting heavy thing, coughing, vomiting and defecation. The maternal straining efforts during childbirth make extensive use of the VM.(1)

   During the maneuver, venous return to the heart is impeded, setting off a well characterized sequence of positive and negative arterial pressure changes heavily influenced by the autonomic nervous system.(2,3,4) Cardiovascular response to VM are divided into four phases, (phase 1) is the onset of strain. During this phase, the increase in intrathoracic pressure compressing the great vessels, raising the aortic pressure, heart rate (HR) is reflexively decreased as a result of baroreceptor activation. There is a little change in cardiac output (CO) at this point. In (phase 2), the increased intrathoracic pressure narrows veins at the thoracic inlet and obstructs systemic venous return. Intracardiac volumes decreased by 25% to30%, reducing cardiac preload and hence stroke volume. Arterial pressure falls to a low-point and pulse pressure narrows. Low pressure cardiac receptors are stimulated, and this leads to sympathetic afferent stimulation of vasoconstrictor region. Baroreceptor impulses originating from the carotid sinus and aortic arch decrease because of lowered mean aortic pressure and pulse pressure. The resulting sympathetic outflow increases HR and contractility and constricts peripheral arteries (increasing resistance) and veins (increasing venous pressure outside the chest). As a result, cardiac volume, stroke volume, CO, and arterial pressure stabilize and may even increase, so a biphasic response is generally observed, consisting of a reduction in aortic pressure (phase 2E), followed by a secondary rise in aortic pressure, after about five seconds, to resting values (phase 2L). At the moment the strain is released (phase 3), intrathoracic pressure falls,

relieving constraining effects on the thoracic vena cava, heart, and aorta. Immediately, there is a transient fall in systemic pressure and HR increases further in reflexive response. The sympathetically mediated vascular responses initiated and sustained during straining then produce a large venous pressure gradient between the extra thoracic capacitance vessels and the under filled low-pressure right atrium, driving an explosive venous return. Within a few seconds, intracardiac volumes return to premaneuver levels. This recruitment of the cardiac length-tension mechanisms coupled with sympathetic cardiac support increases stroke volume and translocates the increased venous return into the constricted arterial circulation. The sudden transfer of this additional volume raises arterial and pulse pressures above premaneuver levels so blood pressure "over shoot" and resulting in a baroreflex-mediated bradycardia (phase4).(3,5,6)

Material and Method:

This study was performed in Marjan hospital in Babylon city during the period May/2005 to December/2005. The subjects were from the medical staff and from the visitors to the hospital.

Seventy healthy subjects with mean age (27.31±5.28years) and age range (20-40years).

Seven subjects were not included in this study because:

1.One person was found with mitral prolapse by echocardiography.
2.Some developed abnormal beats (ectopics) that were recorded by ECG when doing VM, so HR can not be estimated.
3.Some were difficult to estimate their BP during phase2.

Mercury sphygmomanometer:

The ordinary sphygmomanometer was used to measure BP with stethoscope by auscultatory method. Another mercury sphygmomanometer was used to perform the strain part of VM. MBP was calculated according to the equation:
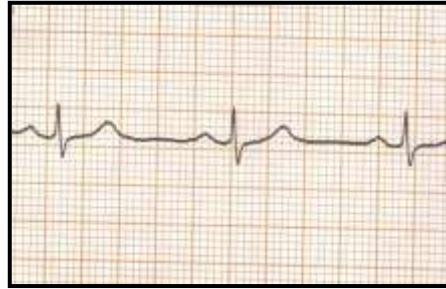
MBP = DBP +1/3 (SBP-DBP)……..(7)
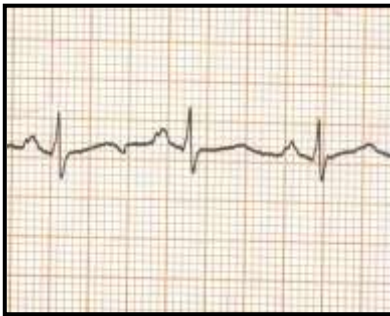
Electrocardiography(ECG):

Used the limb leads only to record strip of lead II before, during and after 2min of VM. Paper moves 1500 small divisions in a minute to measure the HR in a regular heart, the number of small square between two R-waves is counted, 1500 divided by this number gives the HR(8,9) Fig. (1).
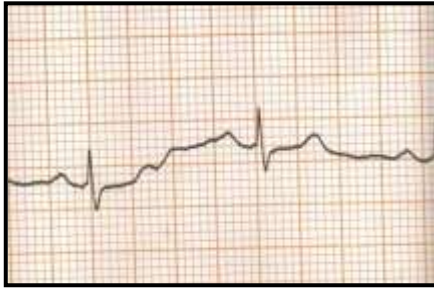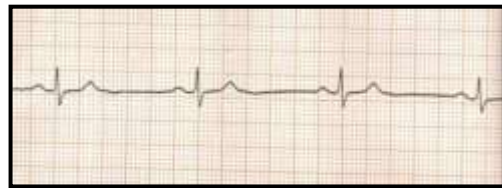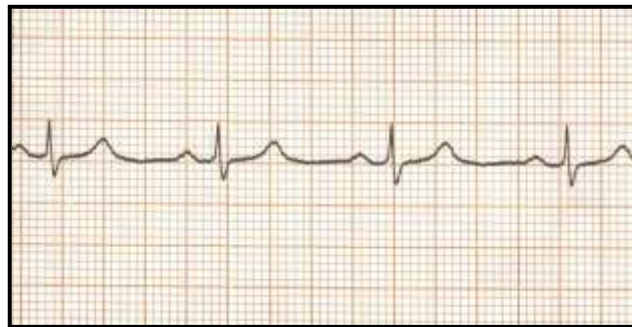
a

b

c

d

e

Echocardiography: M-mode echocardiography used to estimate SV before, during VM (during phase1, phase 2L, and phase 4) and two minutes after finishing of VM. Then CO can be calculated from the equation:

$$CO = SV \times HR\ldots\ldots\ldots\ldots\ldots\ldots\ldots..(10)$$

Phase 2E and phase 3 of VM were not recorded in this study because rapid reduction in BP (i.e. immediate changes) need to be measured by more sensitive technique (invasive technique).

Examination protocol:

The subjects who involved in this study were instructed to do the maneuver of VM and reassurance of these subjects in order to avoid any emotional excitement and try to simplified the procedure for them so that they will not frightened.

The VM was performed with subject in supine position as the quantitative changes observed may be affected by the posture of the subject(23), so it is important to standardized posture as part of the VM protocol. Subject should be in steady state before performing VM.A steady state mean that the HR in consecutive minute changing by less than 3beats/min.(11) Limb lead electrodes were connected to the subject, and tide the cuff of sphygmomanometer to his arm, then placing the transducer of the echocardiography to the chest of the subject. We measured the BP (by sphygmomanometer), SV was obtained by echocardiography, and HR from ECG. These measures were made at rest as a control measurement before VM.

Subjects were asked to exhale forcefully after a normal inspiration into tube that was connected to a sphygmomanometer. A tiny air leak was placed in the tube to ensure that airway pressure was produced from the thoracic cavity and not the pharynx(12), so a small needle is routinely placed into the hose to provide a small air leak. This prevents the subject from closing their glottis and from developing the necessary pressure with the cheek muscle.

The straining phase of VM was maintained for 15 seconds with airway pressure of 30 mmHg.(13) VM repeated for three times with a break (resting time) between each two maneuvers for about 2 minutes. So we measured the SV and BP with the beginning of the straining (phase 1), while ECG recording is continuously measuring HR. Measurement of BP during early phase 2 (phase 2E) was difficult to be assessed by auscultatory method (it needs invasive technique to measure immediate changes in BP), so we assessed the secondary rise in BP during phase2l which occurs after about 5 seconds of beginning of straining(14), at the same time SV was also assessed by echocardiography.

During phase3, there is sudden reduction of blood pressure that can not be assessed by auscultatory method, so hemodynamic variables during this phase were not included in this study.

As phase4 occurs within 3-8 seconds after the termination of VM(15), so hemodynamic variables of this phase measured within this period. After 2 min. of performing VM we also measured SV, BP, and HR were measured during all phases by continuous strip of lead II. All images and

readings were obtained by or under supervision of well-trained cardiologist. We calculated CO, MBP, and PVR from these formulas that mentioned previously:

- $CO = SV \times HR$
- $MBP = DBP + 1/3$ (pulse pressure)
- $PVR = MBP/CO$

Calculation of body mass index (BMI) was calculated as weight in Kg/height2 in(m2).(16) The range of BMI (18-25) is considered normal. All our subjects were within normal range of BMI. Body surface area (BSA) were determined for all subjects. BSA in square meter(m2) as index of body surface are calculated according to nomogram of dubios.(17) Anthropometric data for subjects are shown in table (1) and fig. (1).

Table (1): Anthropometric data for subjects included in the study.

Anthropometric data	Group1(n=70)
Age range (years)	20-40years
Mean age ± SD years	27.31 ± 5.28
Mean weight ± SD (Kg)	73.22 ±11.29
Mean height ± SD (cm)	170.73 ± 11.29
Mean Body surface area ± SD	1.83 ± 0.14
Mean Body mass index ± SD (Kg/m2)	24.46 ± 2.10

Figure (1): Figure showing trace of ECG(leadΠ) in which the control value of HR and the changes which take place during different phases of VM and that after 2min of VM. (a): control, (b):phase 1, (c):phase 2L, (d):phase 4, (e):after 2min of VM.

Result:

Hemodynamic changes that occur during Valsalva maneuver (VM) had been studied in seventy subjects, comparison of hemodynamic changes were made between different phases of VM.

Changes in hemodynamic variables during Valsalva maneuver (VM) and after 2min of VM Table (2).

The comparison of hemodynamic variables between the control values and other phases of VM (phase 1, phase 2L, phase 4) and 2min after VM indicate the following changes: Table(2)

Stroke volume (SV) is significantly lower in phase1 and phase 2L than the control value (phase 2L at $p<0.005$) with no significant changes of SV in phase4 and 2min of VM. Figure (2).

Heart rate (HR) is significantly lowered in phase 1, phase 4 and 2min after VM, and significantly higher in phase2L than that of control value (at p value <0.005). Figure (3).

Cardiac output (CO) is significantly lower in phase1, phase 2l, phase4 and 2min after VM than the control value. Figure (4).

Table (2): Comparison between different phases of Valsalva maneuver and after 2min of VM.

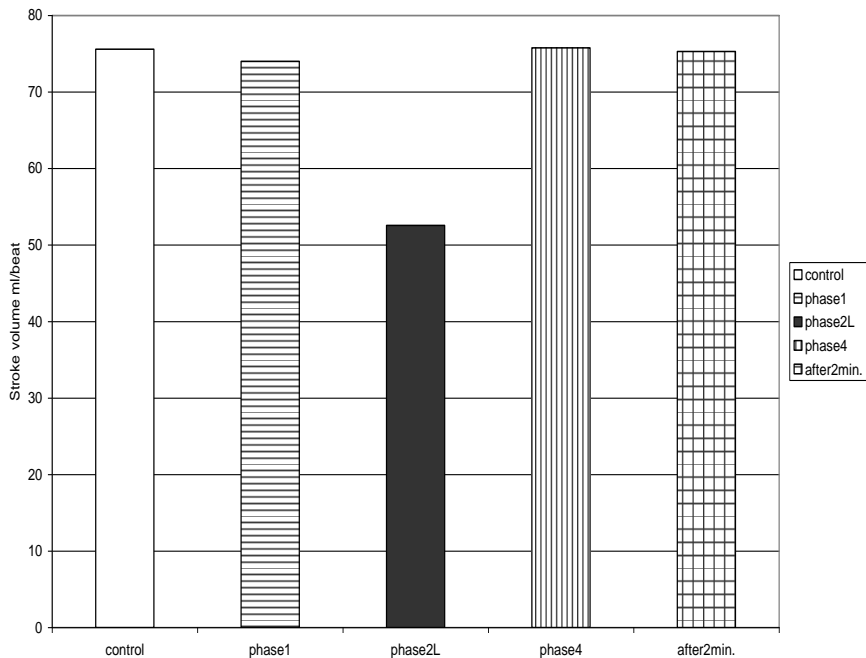| Parameter	Control	Phase1	Phase2L	Phase4	After2min
Stroke volume ml/beat	75.60 ± 9.81	74.00 ± 9.90	52.56 ± 6.23	75.77 ± 9.90	75.30 ± 9.50
Heart rate…… beat/min	80.40 ± 13.80	73.04 ± 12.48	92.02 ± 17.37	65.18 ± 9.46	79.17 ± 11.91
Cardiac output (L/min)	6.06 ±1.06	5.34 ± 0.84	4.77 ± 0.75	4.93 ± 0.69	5.93± 0.82
Systolic blood pressure(mmHg)	127 ± 8.94	149.57 ± 13.20	135.90 ± 10.53	165.60 ± 14.08	127.40 ± 8.90
Diastolic blood pressure(mmHg)	80.68 ± 10.96	98.60 ± 9.39	95.85 ± 10.17	109.57 ± 11.50	81.10 ± 6.67
Mean blood…. Pressure(mmHg)	96.90 ± 6.81	115.62 ± 9.85	109.60 ± 9.64	128.30 ± 11.52	97.16 ± 7.22
Peripheral vascular resistance (Unit)	16.49 ± 2.89	21.99 ± 3.53	23.38 ± 3.85	26.97 ± 5.30	16.25 ± 3.53

Figure (2): Changes in stroke volume before valsalva maneuver (VM) (controle phase), during VM, (in phase 1, phase 2L, and phase 4), and 2 min. after VM of group 1.
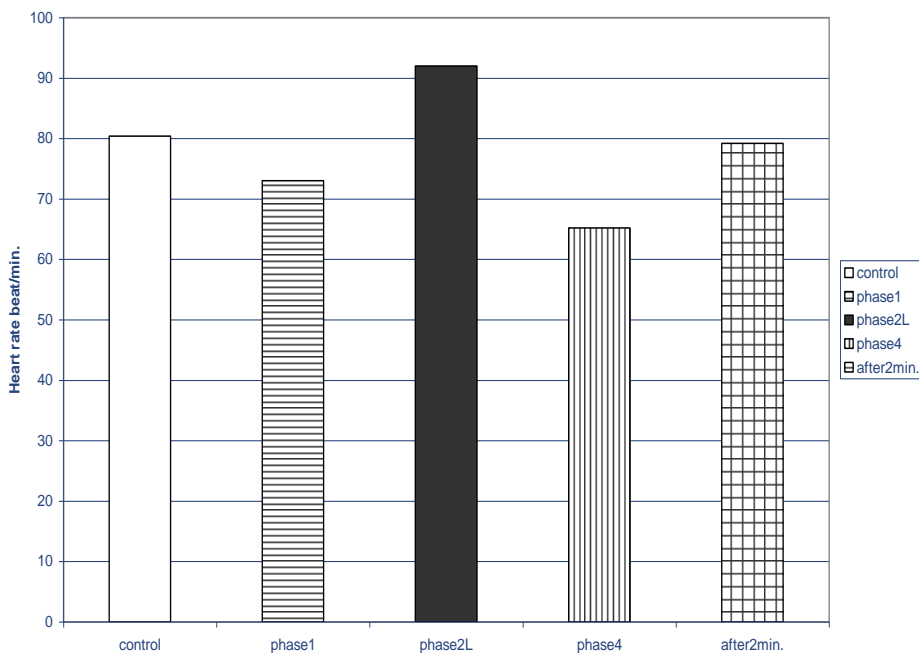
Figure(3):Changes in heart rate group 1 before VM, during VM,
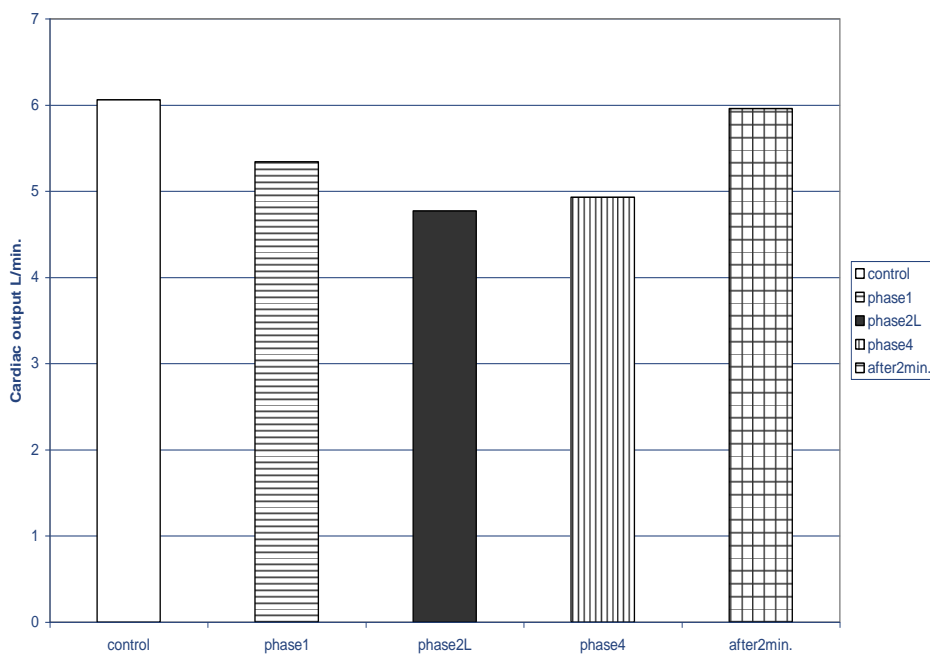(in phase 1, phase 2L, phase 4), and 2 min. after VM.

Figure (4): Changes in cardiacoutput in group 1 before VM, during VM
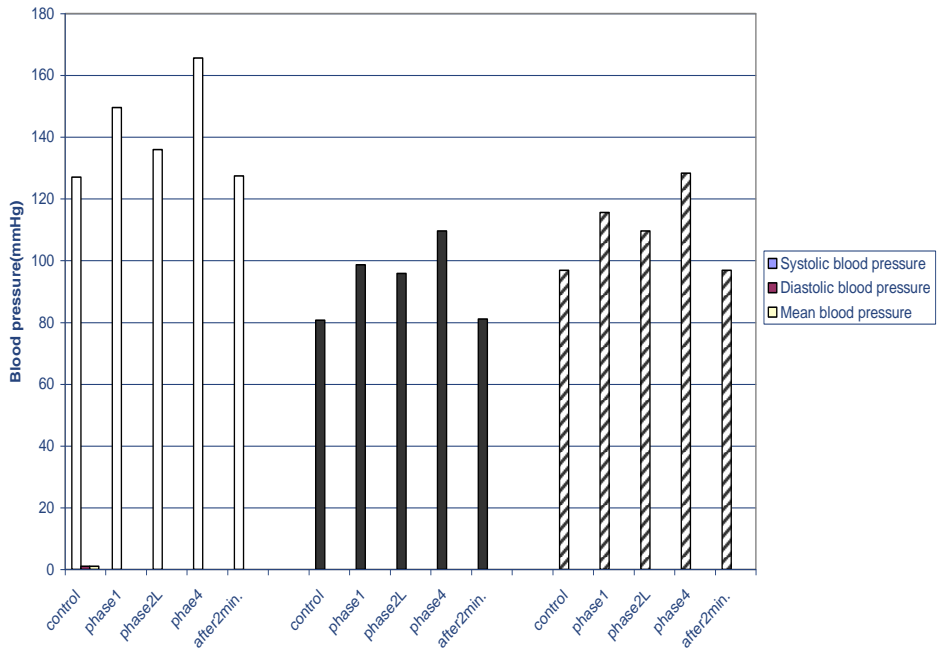, (in phase 1, phase 2L and phase 4),

re (5): Changes in systolic blood pressure and mean blood pressure in group re VM, during VM, (in phase 1, phase 2L, phase 4), and 2 min. after VM.
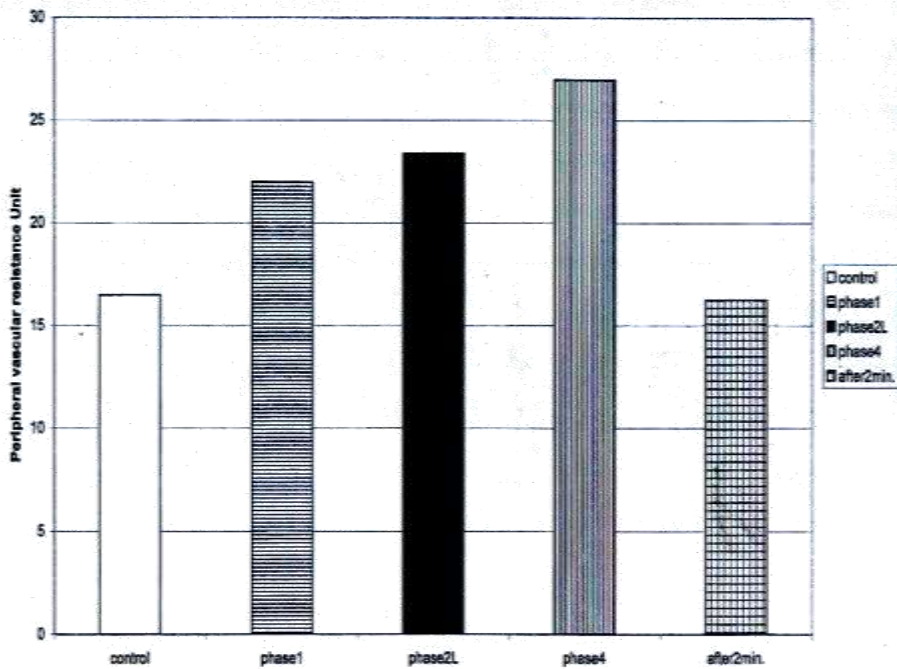

Figure (6): Changes in peripheral vascular resistance in group 1 before VM, during VM, (in phase 1, phase 2L, phase 4), and 2 min. after VM.

Systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean blood pressure (MBP) are significantly higher in phase1, phase2L and phase 4 (phase 4 at $p<0.005$) than the control values with no significant changes after 2min of VM. Figure(5).

Peripheral vascular resistance (PVR) is significantly higher in phase1, phase2L, phase4 (phase4 at $p<0.005$) and 2min after VM than that of control value. Figure (6).

Comparison between phase1 and phase2L indicate that there are significant decrease in SV, CO, SBP, DBP and MBP and significant increase in HR and PVR in phase2L than that of phase1.

Comparison between phase1 and phase4 indicate that SV, SBP, DBP, MBP and PVR are significantly higher and that the HR and CO are significantly lower in phase4 than that of phase1.

Comparison between phase2l and phase4 indicate that SV, SBP, DBP, MBP and PVR are significantly higher and the HR is significantly lower in phase4 than that of phase2l with no significant changes in CO between the two phases.

Discussion:

Hemodynamic changes during different phases of VM of normal healthy subjects with mean age27.31±5.28years:

In this study the stroke volume (SV) is significantly decreased in phase1 and phase2l in comparison with the control value, this is because during straining of VM, the increased intrathoracic pressure narrows veins at the thoracic inlet and obstructs systemic venous return so intracardiac volumes decrease, reducing cardiac preload and hence SV(6,18), and since cardiac output (CO) is measured by the equation (CO = HR × SV) therefore CO is also significantly decreased in phase1 and phase 2L.

The heart rate (HR) is significantly lower in phase1 than that of control value but systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean blood pressure (MBP) are significantly higher than control values because during phase1 of VM there is transient mechanical increase in aortic blood pressure and a decrease in HR immediately after the commencement of straining(14,19), and for this reason the peripheral vascular resistance (PVR) is significantly increased in phase1 than that at control phase according to the equation (PVR = MBP/CO).

The values of HR, SBP, DBP, MBP and PVR are significantly higher in phase 2L than control values because during early period of phase 2 (phase 2E), arterial pressure falls to low point, low pressure cardiac receptors are stimulated, and this leads to sympathetic afferent

stimulation of vasoconstrictor region, baroreceptor impulses originating from the carotid sinus and aortic arch decrease due to lowered mean aortic pressure. The resulting sympathetic out flow increases HR and contractility, and constrict peripheral arteries so increasing vascular resistance and constrict veins so increasing venous pressure outside the chest. As a result arterial pressure stabilize and may even increase.(6)

There is no significant difference between SV during control phase with that of phae4, but HR during phase4 is significantly lower than that during control phase while SBP, DBP, MBP and PVR are significantly higher in phase 4 in comparison with control values, these findings can be explained that during phase 4, which is after release of strain, intracardiac volumes return to premaneuver levels within few seconds as mentioned before, this recruitment of the cardiac length-tension mechanisms coupled with sympathetic cardiac support increases SV and translocates the increased venous return in to the constricted arterial circulation. The sudden transfer of this additional volume raises arterial and pulse pressures above premaneuver levels so blood pressure "over shoot" resulting in baroreflex-mediated bradycardia.(6,15)

There are no significant differences between SV, SBP, DBP and MBP during control phase and these variables after 2min of VM as they return to premaneuver levels, but the HR of some subjects were not return back to premaneuver levels even after 2min of the end of VM since VM is one of the methods of increasing vagal tone so some times could be used as method of treatment of supraventricular tachycardia.(20) Therefore CO after 2min of VM is significantly lower than that during control phase, and that PVR is significantly increased after 2min of VM when compared with the control value according to the equations (CO = HR × SV; PVR = MBP/CO).(21)

The values of SV, CO, SBP, DBP and MBP during phase 2L are significantly lower than these values during phase1 while HR and PVR are significantly higher in phase2L than HR and PVR during phase1, because during phase1 the increase in intrathoracic pressure is mechanically transmitted through the arterial tree increasing blood pressure, with a reflex decrease in HR while in phase2, the reduction in venous return result in a decrease in CO and BP which falls low enough to cause baroreflex response increase in HR and vasoconstriction.(22)

The SV during phase 4 is significantly higher than SV during phase1 as SV during phase4 return to premaneuver level as mentioned before. HR and CO are significantly lower during phase 4 than that during

phase1 but SBP, DBP, MBP and PVR are significantly higher in phase 4 than that in phase1 because of the over shooting of BP with reflex bradycardia during phase 4.(19)

As SV during phase 2L decreased and mediated by reflex tachycardia, on the contrary SV in phase 4 return to premaneuver level but there is bradycardia during this phase, so SV during phase 4 is significantly higher than that during phase 2L but HR in phase4 is significantly lower than that of phase2, with no significant difference of CO of phase 2L and phase 4.

Because of the over shooting of BP during phae4 so SBP, DBP, MBP and PVR are significantly higher in phase 4 than these hemodynamic values during phase 2L.

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