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EFFECT OF VALSALVA MANEUVER ON PULSE WAVE VELOCITY

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Abstract : Pulse wave velocity (PWV) was recorded in different arterial segments in 25 male normal healthy individuals, before, during and after Valsalva maneuver (VM). Heart rate (HR) and blood pressure (BP) showed significantly significant (P<0.001) variation in phase II and IV of VM compared to baseline. PWV in aorta to femoral (AO–Fem) arterial segment revealed progressive increase from baseline to phase IV of VM (P<0.001), while PWV in femoral to dorsalis pedis (Fem-DP) arterial segment showed more increase (P<0.001) in phase II rather than phase IV from baseline with insignificant variation in aorta to radial (AO–Rad) arterial segment, demonstrate different response of arterial segments to VM.

Key words : valsalva maneuver blood pressure pulse wave velocity

INTRODUCTION

The Valsalva maneuver (VM) is widely accepted and reliable stress test to assess cardiovascular and autonomic functions in normal subjects and in cardiac disorders (1–3). Changes in blood pressure (BP) during VM are associated with other haemodynamic alterations including those in heart rate (HR), blood flow, peripheral resistance, pulmonary capillary wedge pressure (4), and left ventricular filling pressure (5). The haemodynamic alterations during VM have been studied by numerous investigators (3–5). However, the relation between compliance of blood vessels and VM has not been paid attention. Since we know that catastrophic rise in BP is known to be a precipitating factor for vascular events (6) and VM is associated with much rise in BP. VM occurs regularly during coughing, defecation and heavy lifting (7). Pulse wave velocity (PWV) is a test for arterial function. So the author aimed to evaluate the effect of VM, on PWV of different arterial segments (central and peripheral of both upper as well as lower limbs) in normal healthy individuals.

MATERIALS AND METHODS

The study was carried out in 25 young healthy male subjects in the age group of 20–40 years. Detailed history was taken and clinical examination was done with particular emphasis on the examination of cardiovascular system to exclude the

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presence of congenital, valvular or organic heart disease. Excluded from the study were subjects having history, examination or investigations suggestive of collagen disorder, diabetes, hypertension, atherosclerosis, and intake of beta blockers or vasodilators and smoking. Subjects selected had normal haemoglobin, blood pressure and electrocardiogram (ECG). Each subject was investigated at about 10 am to avoid effect of circadian variation (8) after being given 20 minutes rest. Procedure of recording was explained to the subjects. Recording of PWV was first taken at rest. A deeper than normal inspiration was followed by a forceful exhalation into mouth piece attached by a tube to sphygmomanometer, so that pressure of 40 mmHg was maintained for about 15 seconds (3). Nose clips prevented air leaks and all subjects had intact tympanic membranes. Care was taken that subject was not merely blowing his cheeks. Recordings were made throughout the period of VM. Each subject performed at least two complete Valsalva maneuvers (VMs). Three phases of VM (3) were analysed to calculate PWV in each subject as mentioned below :

1. The control period before initiation of strain.
2. The mid to late strain period after the transient response and appearance of reflex tachycardia (phase II).
3. The post release period (phase IV).

PWV was recorded from the following arterial segments:

1. From the root of the aorta to the left radial artery at the wrist (AO-Rad) (peripheral arterial segment in upper limbs).
2. From the root of the aorta to the left femoral artery just below the inguinal ligament (AO-Fem) (central arterial segment).
3. From the left femoral artery just below the inguinal ligament to the left dorsalis pedis artery (Fem-DP) (peripheral arterial segment in lower limbs).

Pulse wave velocity (PWV) was recorded as described by Eliakim and associates (9). For this, subject was asked to lie down on couch in supine position. Pulse was felt at femoral artery just below the inguinal ligament, left radial artery at the wrist and dorsalis pedis artery in first metatarsal space on the dorsum of the foot. On these sites, pulse transducers having built in photodetectors and light source were placed and strapped, so that they remain in position. ECG was also recorded. Simultaneous recordings of arterial pulse from above mentioned sites were made on Polyrite (INCO) at a paper speed of 50 mm/sec. For arterial segments AO–Rad and AO–Fem, time delay (T) of pulse waves was measured from the peak of the R wave of ECG to the beginning of onset of pulse wave in the radial and femoral artery simultaneously (Fig. 1). For Fem–DP arterial segment, it was measured between the onset of pulse waves in femoral and dorsalis pedis arteries (Fig. 2) and expressed in milliseconds. Length of arterial segment (L) was measured (mm) on the surface of the body. The position of root of aorta was considered to lie in left third parasternal space. The PWV (m/sec) was then calculated by using formula L/T (9). Only clearly defined tracings were analysed. In each subject five measurements were made and mean of that was calculated. Heart rate (HR) was analysed by ECG. Blood pressure (BP) was recorded

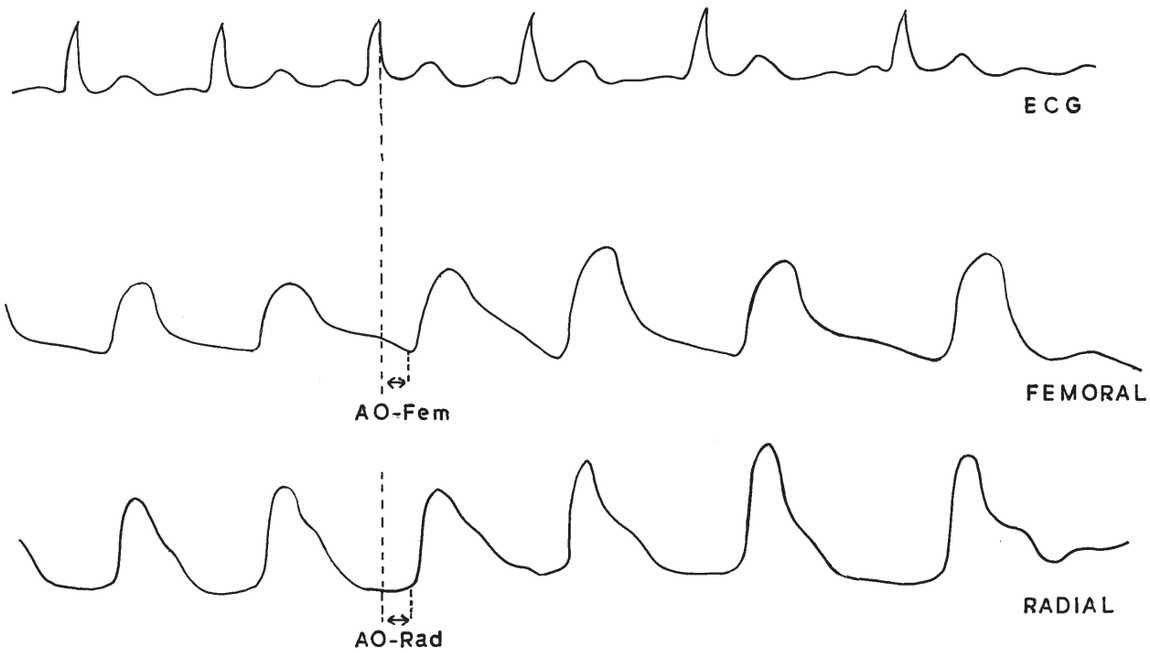

Fig. 1 : Recording of ECG and arterial pulse waves from femoral (AO–Fem arterial segment) and radial (AO–Rad arterial segment) arteries.

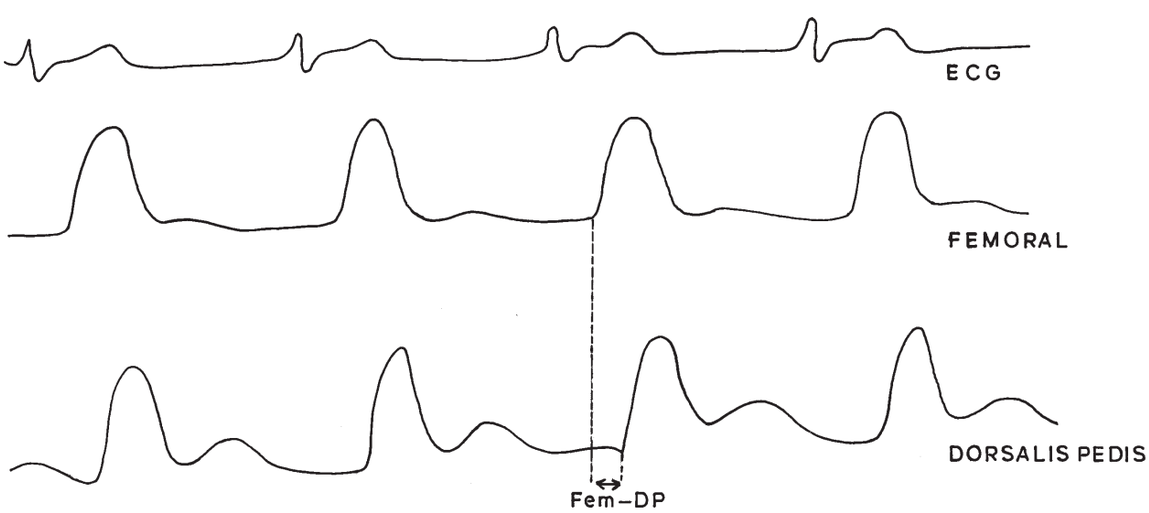

Fig. 2 : Recording of pulse waves from femoral and dorsalis pedis arteries (Fem–DP arterial segment).

by sphygmomanometer. Data were analysed statistically using paired 't' test. Significance of differences was compared between resting values and those of phase II and phase IV of VM.

RESULTS

Baseline demographics of the subjects are given in Table I. HR and BP showed statistically significant (P<0.001) variation in phase II and IV of VM compared to baseline. PWV in central arterial segment (AO–Fem) showed progressive increase from baseline to phase IV of VM (P<0.001), while in peripheral arterial segment (Fem–DP) PWV increased more (P<0.001) in phase II (16.3%) compare to phase IV (12.3%) from baseline and insignificant changes were recorded in AO–Rad arterial segment.

TABLE I : Base line demographics, heart rate, blood pressure and pulse wave velocity (mean±SD) in different arterial segments before and after Valsalva maneuver.

Vareiable	*Before VM*	*During VM*	*After VM*
Age (yrs)	25.62±10.30	–	–
Sex	Male	–	–
Quetelet index	24.2±0.34	–	–
Heart rate (beats/min)	74.62±11.03	89.84±17.50*	63.22±9.7*
Blood pressure:			
Systolic (mm Hg)	116.84±10.02	100.21±9.46*	138.90±9.27*
Diastolic (mm Hg)	76.83±10.47	68.72±8.52*	91.06±10.57*
PWV (m/sec) in arterial segment			
AO–Rad	8.06±2.23	8.39±3.50	8.04±1.85
AO–Fem	4.4±1.08	4.6±1.59	5.02±2.05*
Fem–DP	8.53±1.24	9.6±2.17*	9.35±1.90*

*P<0.001, AO–Rad (aorta to radial artery), AO–Fem (aorta to femoral artery), Fem–DP (femoral to dorsalis pedis artery).

DISCUSSIONS

Clinical criteria provide much information about vascular insufficiency but these are difficult to quantitate. Functional status of blood vessels can be evaluated by PWV. It is found to be increased with age, in coronary artery disease, diabetes and atherosclerosis (9). Changes in HR and BP during VM in current study were consistent with earlier observations (10, 11). PWV recorded in different arterial segments in normal healthy individuals agree fairly well with those reported earlier (12). PWV is changed according to age (7, 9, 13) and sex (14). In order to eliminate these differences, we investigated only in males in age group of 20–40 years. The present study clearly demonstrates that PWV increases both in peripheral and central arterial segments during different phases of VM. There is much variation in HR in phase II and IV of VM but changing pulse rate has no effect on PWV (9). PWV increases with rise in BP (15). Cold pressure test and head up tilt test of autonomic functions causes rise in BP associated with augmentation in PWV (16). Hypertension has been reported to increase PWV (9. 13). Dependence of PWV on BP was also stressed by other workers (17). Increase in PWV can also be explained by increased circulation time (18) and increase in left ventricular ejection time (LVET) during VM (19). Our observations revealed that changes in PWV did not occur quantitatively equally in all segments. Different haemodynamic response to VM may be due to different rate of distension of arterial wall (20). So it is concluded that different arterial segments behave differently to VM.

REFERENCES

1. Baldwa VS, Ewing DS. Heart rate response to Valsalva maneuver: reproducibility in normals and relation to variation in resting heart rate in diabetics. *Br Heart J* 1977; 39: 641–644.
2. Kraaij D, Schuurmans M, Janson R. Use of Valsalva maneuver to identify haemodialysis patients at risk of congestive heart failure. *Nephrol Dial Transplant* 1998; 13(6): 1518–1523.
3. Felker M, Cuculich PS, Gheorghiade M. Valsalva maneuver: a bedside biomarker for heart failure. *Am J Med* 2006; 119: 117–122.
4. Mc Intyre KM, Vita JA, Lambrew CT. A noninvasive method of predicting pulmonary capillary wedge pressure. *N Eng J Med* 1992; 327: 1715–1720.
5. Sharma GVRK, Woods PA, Lambrew CT. Evaluation of a noninvasive system for determining left ventricular filling pressure. *Arch Intern Med* 2002; 162(18): 2084–2088.
6. Chakrabarti P, Chatterjee S. A study of blood pressure changes with variation in posture. JAPI 2001; 49: 61.
7. Ganong WF. Review of Medical Physiology. Cardiovascular regulatory mechanisms. 23rd Ed. Section VI. Chapter 33. Tata Me Graw Hill Education Private Limited, New Delhi. Copyright © 2010; 562.
8. Maruo T, Nakatani S, Kanzaki H, Kakuchi H, Yamagishi M, Kitakaze M, Ohe T, Miyatake K. Carcadian variation of endothelial function in idiopathic dilated cardiomyopathy. *Am J Cardiol* 2006; 97: 699–702.
9. Eliakim M, Sapoznikov D, Weinman J. Pulse wave velocity in healthy subjects and in patients with various disease states. *Am Heart J* 1971; 82(4): 448–457.
10. Levin AB. A simple test of cardiac function based upon heart changes induced by the Valsalva maneuver. *Am J Cardiol* 1966; 18: 90–99.
11. Judson WE, Hatcher JD, Wilkins RW. Blood pressure responses to the Valsalva maneuver in cardiac patients with or without congestive failure. *Circulation* 1955; 11: 889–899.
12. Rossman I. Clinical geriatrics. 2nd Ed. Philadelphia JB Lippincott Company. 1979; 26.
13. Simonsan E, Koff S, Keys A, Minckler J. Contour of toe pulse reactive hyperemia and pulse transmission velocity: group and repeated variability, effect of age, exercise and disease. *Am Heart J* 1955; 50: 260–266.
14. Schimnler W. Correlation between the pulse wave velocity in aortic iliac vessel and age, sex and blood pressure. *Angiology* 1966; 17: 314–317.
15. Learoyd BM, Taylor MG. Alterations with age in viscoelastic properties of human arterial wall. *Circ Res* 1966; 18: 278–292.
16. Brod L, Fenel V, Hejl Z, et al. Haemodynamic changes underlying pressure reactions in man. *Rev Ezeehoslov Med* 1958; 4: 170.
17. Nye ER. The effect of blood pressure alteration on pulse wave velocity. *Brit Heart J* 1964; 26: 261–265.
18. Stucki P, Hatcher JD, Juatson WE, Wilkins RW. Studies of circulation time during Valsalva test in normal subjects and in patients with congestive heart failure. *Circulation* 1955; 11: 900–908.
19. Layton C, Nunzio H, Gent G, Freedman P, Mc Donald A. Rate corrected systolic time intervals and Valsalva maneuver. *Br Heart J* 1973; 35: 236–244.
20. Peterson LH, Jensen RE, Parenell J. Mechanical properties of arteries *in vivo*. *Circulation Res* 1960; 8: 622–639.