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Valsalva Maneuver and Strain-Related ECG Changes

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Background: During the four steps of the Valsalva maneuver (VM), opposite changes can be observed in some physiologic parameters. Manifestations of the VM in the electrocardiogram (ECG) have been studied in detail, but there have been few reports comparing VM-related biochemical and hemodynamic changes with a focus on the strain phase of the VM.
Objectives: We studied strain-related ECG changes during the VM.
Patients and Methods: This self-control descriptive study was conducted in 20 healthy male college students aged 20.12 ± 2.23 years. They were well trained to perform the standard VM: maintenance of a 15-second expiratory pressure at 40 mmHg with open glottis. An ECG was continuously recorded in the supine position at (a) rest (before the VM) and at (b) the start and (c) end of strain maintenance in the second phase of the VM. The averages of four successive beats were computed for each set of (a), (b), and (c). Means ± standard errors of the mean were used for comparison.
Results: Following the VM, the RR and PR intervals, corrected QT interval (QTc), P wave duration and amplitude, T wave amplitude, and the ratio of T/R amplitudes showed significant changes. The QRS duration and R wave amplitude did not show significant changes.
Conclusions: The VM caused time and voltage changes in some ECG waves during the strain phase. Alterations in heart or lung volume, electrode-related alterations, and autonomic tone may cause these changes.

Keywords: Valsalva Maneuver; Straining; Electrocardiogram

1. Background

The Valsalva maneuver (VM) often is defined as 15 seconds of expiration with an open glottis against 40 mmHg pressure. The strain maneuver has a very old history and is documented as being used for the diagnosis of skull fracture in 1497. Moreover, the first report about the VM and the cardiovascular system was reported 162 years ago, and the effect of the VM on blood pressure, heart rate, baroreflexes, and its applications for treating some tachyarrhythmias is now well known (1, 2). The VM has four different phases: the first phase is the start of straining that is accompanied by an intrathoracic pressure increase, vasoconstriction, an increase in arterial blood pressure, and transient compensatory bradycardia. The second phase or the continuation of strain decreases venous return to the heart with a subsequent decrease in cardiac output, systolic arterial blood pressure, and carotid sinus stimulation. This is followed by vagus inhibition and sympathetic stimulation. The results are tachycardia and an increase of systemic vascular resistance. In the third phase (stop or termination of straining), venous return and the capacity of pulmonary vessels increases, and the accumulation of blood in the lung causes a further decrease in arterial blood pressure. In the fourth phase (e.g., rest step), left-side venous return from the lung is pumped through high resistance arteries by the left ventricle, causing a severe increase in arterial blood pressure and subsequent reflex bradycardia (3). Although there is general agreement about the effect of the VM on a reduction of left ventricle dimensions, the results concerning its effects on the ECG have been inconsistent (4-6). In nearly all previous studies, ECG changes during the VM were compared with the basal resting state with a focus on mechanical mechanisms, and ECG changes were attributed to the effect of the VM on volume and position of the heart.

2. Objectives

To detect any strain-induced ECG alterations in isolation, we performed this study to compare the effect of straining on time and voltage of ECG waves.

3. Patients and Methods

Subjects were 20 volunteer male college students who were eligible for the study. Mean age was 20.12 ± 2.23 years (range, 18 - 24 years). All subjects provided informed consent. Subjects did not have any history of cigarette smoking, medications, diabetes mellitus, or cardiovascular or

any other diseases. All participants were trained to perform the standard VM. In each set, only one participant was investigated. Tests were scheduled at least 2 hours after meals and between 10 - 11:30 AM. The ECG was recorded in the supine position and in a comfortable place with appropriate ventilation. After a 5-minute adaptation period, data collection was started. A sampling frequency of 1 kHz was used for analog-to-digital data conversion. Lead II was used for analysis of all ECG parameters.

At least four consecutive beats were used for data in all cases. The corrected QT interval (QTc) was computed according to Bazett's formula.

(1) $$QTc = \frac{QT}{\sqrt{RR}}$$

Each participant performed the VM for 15 seconds by expiring through a plastic tube that was connected to a mercury column. The target expiratory pressure with open glottis was set at 40 mmHg. The tube had a small hole to prevent pressure from easily increasing at the mouth without any intrathoracic hemodynamic effects. Recording of data continued for 5 seconds after finishing the maneuver.

The means of four consecutive beats were calculated in three steps as follows: (a) basal condition (before the VM), (b) immediately after the start of the second phase of the VM (strain phase), and (c) at the end of second phase and before the start of third phase. All data were compared using mean and ± standard deviation. PowerLab, the respiratory belt, and LabChart 5 software (all from AD Instruments, Australia) were used for recording the ECG, respiratory movements, and data computing, respectively. Comparisons between different times were performed by repeated measure ANOVA model, followed by Bonferroni post-hoc test for pair wised comparisons. we applies SPSS 16 for Windows (SPSS Inc., Chicago, Illinois) for statistical analysis. Statistical significance was accepted at a level of $P < 0.05$. The work was approved by the regional research ethics committee.

4. Results

The mean heart rate was 78.7 ± 1.9 bpm in the basal condition, (a) increasing to 86.12 ± 2.3 bpm and 94.8 ± 4.46 bpm during the primary (b) and terminal (c) 5 seconds of the strain phase of the VM, respectively. The mean time of the P wave showed a significant difference just at the beginning of the second phase of the VM (0.0840 ± 0.00254 seconds) in comparison to the basal resting condition (0.0893 ± 0.00316 sec). The P wave amplitude significantly increased in (b) and (c) in comparison to (a). Mean amplitudes of P, Q, R, S, and T waves are shown in Table 1.

Following the VM, the RR interval and PR time during (b) and (c) were significantly lower than these times during (a). The QT interval was not different, but the QTc was increased in (b) and (c) relative to (a). The time of the PR, RR, and QT intervals and QTc in (a), (b), and (c) are shown in Table 2.

The QRS duration showed no significant changes. The R wave amplitude had no significant changes during strain (e.g., b and c) in comparison with (a), but was lowest in (c). The T wave amplitude decreased in (b) and (c), but was significantly less in (c) than (b). The ratio of the T/R amplitude in (b) and (c) was less than. The time of data selection for all three steps of (a), (b), and (c) was equal and is represented in Figure 1. A representative ECG over four heart beats at the beginning of the VM and some form factor values are shown in Figures 2 and 3, respectively.

Table 1. Mean Amplitude of P, Q, R, S, and T Waves in the Basal Condition and at the Onset and End of the Second Phase of the Valsalva Maneuver [a,b]

Wave	Basal Condition[c]	Establishment of Second Phase (First 5 Seconds)[c]	End of Second Phase (Last 5 Seconds)[c]
P	0.1245 ± 0.00783	0.1590 ± 0.01187	0.1553 ± 0.01292
Q	-0.0370 ± 0.01168	-0.0495 ± 0.01329	-0.0825 ± 0.01308
R	0.9245 ± 0.05014	0.9465 ± 0.05220	0.8875 ± 0.06106
S	-0.0625 ± 0.03484	-0.0615 ± 0.03761	-0.1250 ± 0.04112
T	0.3105 ± 0.03304	0.2700 ± 0.02342	0.2132 ± 0.02812

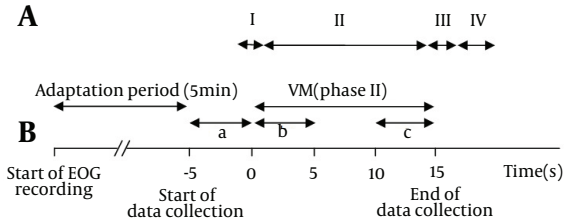
[a] Abbreviations: mV: millivolt, and VM: Valsalva maneuver.
[b] Mean and standard error of amplitude of waves (mV) for four consecutive beats during the VM (n = 20).
[c] All the pair wise comparisons between the parameters are significant (p>0.05)

Table 2. Mean Time of PR, RR, QT, and Corrected QT Intervals in the Basal Condition and at the Onset and End of the Second Phase of the Valsalva Maneuver [a,b]

Interval	Basal Condition[c]	Establishment of Second Phase (First 5 Seconds)[c]	End of Second Phase (Last 5 Seconds)[c]
PR	0.1530 ± 0.00539	0.1450 ± 0.00531	0.1347 ± 0.00461
RR	0.7715 ± 0.01974	0.7025 ± 0.01915	0.6430 ± 0.02873
QT	0.3245 ± 0.00462	0.3345 ± 0.00613	0.3216 ± 0.00781
QTc	0.3705 ± 0.00407	0.4015 ± 0.00726	0.4063 ± 0.00974

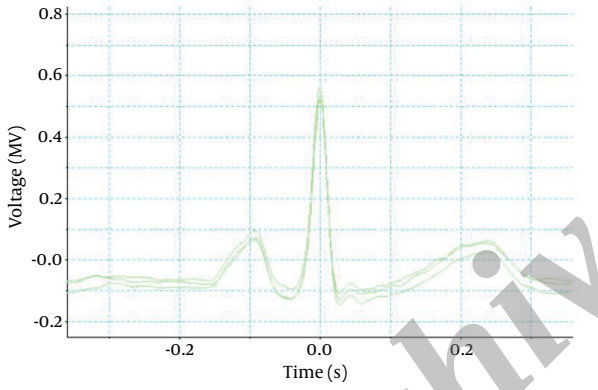
[a] Abbreviations: VM: Valsalva maneuver, msec: millisecond, QTc: corrected QT interval.
[b] Mean and standard error of time of waves (msec) for four consecutive beats during the VM (n = 20).
[c] All the pair wise comparisons between the parameters are significant (p>0.05)

Figure 1. Four Phases of the Valsalva Maneuver and Scheduling of Data Collection



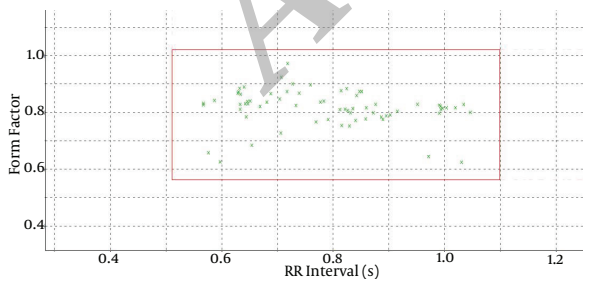
A, Four phases of the VM: I, first phase: from the start of expiration until achievement of 40 mmHg pressure; II, second phase: strain maintenance, continuance of expiration and maintenance of 40 mmHg pressure for at least 15 seconds; III, third phase: end of expiration; IV, fourth phase: recovery. B, Scheduling of data collection, a) control: ECG recording under resting condition; b) ECG recording during the first 5 seconds of phase II of the VM; c) ECG recording during the last 5 seconds of phase II of the VM. VM: Valsalva maneuver, ECG: electrocardiogram.

Figure 2. ECG Recordings Over Four Heart Beats at the Beginning of the Valsalva Maneuver



Time and voltage measurements are different from standard electrocardiography.

Figure 3. Some Form Factor Values of Recorded Signals



Form factor values give an indication of how much the detected beats conform to the shape and time course of the QRS complex of ECG signals and indicates the 'complexity' of the waveform. Form factor values were calculated from data points in an interval of QRS width (Wqrs) centered on the beat marker using the standard deviation of the second derivative, divided by the standard deviation of the signal itself. Form factor values are strongly dependent on the shape and time course of the QRS complex, but are independent of amplitude.

5. Discussion

Results showed that the second phase of the VM causes alterations in amplitude and time of some waves and intervals of the ECG. The Brody effect is one of theories in this setting and proposes that cardiac blood volume and high electrical conductance of heart are the main causes of the increase in T and R wave amplitudes whenever an increase in the ventricular volume load occurs. This condition occurs after cessation of straining in the third phase of the VM, but has not generally been confirmed by others (6). The VM has no effect on QT dispersion (difference of maximum and minimum QT interval) and area under the curve of the T wave (7). Moreover, an increase in P wave amplitude (8) and no alteration or a decrease in R wave amplitude during the VM has been reported (4-6). In addition, a decrease in T wave amplitude has also been reported. However, these changes are not detectable in all leads (6). The VM had no effect on a superficial potential map of the body in 120 leads recording from the body surface (9). Differences in methods and conditions of VM performance is one of the main causes of the inconsistency among results. Therefore, despite many reports regarding the VM, only literature over the past decade has emphasized the effect of the methodology regarding VM performance on the results as well as the necessity of maintaining a standard condition. In the standard condition, results of the VM and its stimulatory effect on the autonomic nervous system and electrical and mechanical activity of heart are valid, accurate, and appreciable (3). The present results also showed an increase in the heart rate in the second phase of the maneuver. This increase continued in the strain period, and heart rate increased until the end of this phase before returning to baseline values under the basic condition. This RR interval shortening was consistent with the reduction in the PR interval at the onset and at the end of straining. An increase in sympathetic stimulation and a decrease in vagus tone will produce PR interval shortening (7). Although the time of waves (excepting the P wave) did not have specific changes, it seems that, in the second phase of the VM, the increase in P wave amplitude and the decrease in T and R wave amplitudes and their ratio are more related to changes in autonomic balance as well as to the effect of lung volume on electrical resistance of the heart (9). In other words, it seems that the main changes in the volume of cardiac chambers in comparison with basal condition occur during the second phase of VM. Until the end of this phase, the sizes of the atria and ventricles seem to show no specific changes. The probability of temporal delay in the incidence of an effect of VM on these changes may be proposed for changes in the P, R, and T waves. Indeed, considering that about 15 cardiac cycles are expected to occur during the strain phase, the first four beats may not be sufficient to represent changes compared to the basic condition. Some limitations of this study include the effect of increased heart rate on P wave amplitude in inferior leads and the use of only lead II for data analysis. The aim of present study was to investigate the effect of the VM on duration and amplitude of normal

ECG waves. To identify the mechanisms of changes it would be necessary to simultaneously record and investigate the volume of cardiac chambers using echocardiography. Moreover, electrode positions and distance from the heart, three-dimensional conditions of the heart chambers, and other complementary studies are important issues.

There are changes in time and amplitude of some waves and intervals compared to a normal ECG after the first phase of the VM and during the strain phase. Alterations in heart or lung volume, electrode-related alterations, and autonomic tone may cause these changes.

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Authors' Contributions

seyed Mehran Hosseini: study design, supervision of the work, and data analysis. Mohsen Jamshir: performance of the work and preparation of the manuscript.

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