Valsalva and modified Valsalva maneuver

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Abstract

Valsalva maneuver is a diagnostic and adjunctive method in non-medical treatment of supraventricular tachycardia, in physical examination, differential diagnosis and imaging-guided evaluation of congenital heart defects and valvular diseases and also to show the presence of autonomic neuropathy caused by diabetes, chronic renal failure, certain cardiomyopathies and neurological disorders. The modified Valsalva maneuver is more effective and less complicated variant of the standard Valsalva maneuver.

Key words: Valsalva, modified Valsalva, cardiovascular reflex

The Valsalva maneuver (VM) was first described by Antonio Maria Valsalva [1]. Valsalva maneuver is performed in the form of forced and sudden expiration with the glottis closed (Figure 1). The maneuver should be performed on a specially prepared mercury manometer with an average pressure of 40 mmHg and end for 10-15 seconds. The expiration should be done abruptly and terminated abruptly [1]. Patients should be allowed to rest in a sitting position for 15 minutes before the test and prevent deep inspiration before expiration. Valsalva maneuver is used for the detection of changes in heart rate and blood pressure values due to autonomic neuropathy, as well as for the diagnosis of heart valve disease, congenital heart disease and evaluation of left ventricular function [2]. Valsalva maneuver is also a treatment method used in the termination of SVT [3].

Figure 1. Valsalva maneuver is performed in the form of forced and sudden expiration with the glottis closed.
In the Valsalva maneuver, there are many changes in heart rate and blood pressure through autonomic reflexes [4]. The Valsalva maneuver acts through the baroreceptor reflex network, which is responsible for cardiovascular hemostasis. Baroreceptors (BR) are receptors on the wall of the sinus caroticum and arcus aorta. The excitations from these baroreceptors reach the nervus glossopharyngeus directly through the nervus vagus or via the Hering nerve and from there to the medullary vasomotor center. The response is transmitted to the vascular wall and nodes via the nervus vagus and sympathetic nerves. When BP increases, the number of stimuli transmitted from the BRs to the medullary vasomotor center increases and the vasoconstriction center is inhibited in parallel with this increase while the nervus vagus is stimulated. When blood pressure decreases, the number of stimuli delivered to the medullary vasomotor center decreases and blood pressure increases with peripheral vasoconstriction, while node stimulation increases and heart rate increases [5]. This mechanism through baroreceptors is a response to sudden hemodynamic changes and has no effect on long-term changes.

In the first stage of Valsalva maneuver, intrathoracic pressure increases with forced and sudden expiration and this pressure reflects on the heart and aorta, and blood pressure rises suddenly. As the intrathoracic pressure continues, venous circulation is prevented, and the blood flow to the heart decreases, resulting in the lower left and right heart volumes, cardiac output volume, cardiac output, blood and pulse pressure. Due to the decrease in blood pressure, baroreceptor activity will decrease, and the number of stimuli delivered to the medullary vasomotor center will decrease and as a result, tachycardia will develop. Tachycardia is a reflex response to a drop in blood pressure. Peripheral vascular resistance develops after approximately 7-8 seconds [6]. Intrathoracic pressure returns to normal and venous return increases with sudden termination of expiration. With the termination of VM, intrathoracic pressure decreases and blood pressure rises above baseline values with the effect of ongoing peripheral vasoconstriction as well as increased return. The increase in BP is followed by an increase in BR reflex response within 3-5 seconds, resulting in vagal bradycardia [7]. This bradycardia is followed by peripheral vasodilatation, which leads to normalization of BP. At this stage, stroke volume increases with the return of peripheral blood to the heart (Table 1) [8,9].

### Table 1: Cardiovascular changes occurring during Valsalva maneuver

<table>
<thead>
<tr>
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<th>stage-1</th>
<th>stage-2</th>
<th>stage-3</th>
<th>stage-4</th>
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</thead>
<tbody>
<tr>
<td>Intrathoracic pressure</td>
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<td>⇑⇑</td>
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<tr>
<td>Blood pressure</td>
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<td>Venous Return</td>
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<tr>
<td>Heart rate</td>
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<tr>
<td>Peripheral Vascular Resistance</td>
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</table>

⇑ : Increases ⇑⇑: Increases significantly _____: Does not change ⇑: Decreases ⇑⇑ : Decreases significantly

The response to the Valsalva maneuver varies depending on the posture during the maneuver in healthy individuals. It was observed that the observed tachycardic response was seen more in the sitting position than the forward-curved position. On the other hand, it was observed that the stroke volume decreased more in the standing and sitting position than in the reclining and tilted position. Blood pressure changes are less observed in the supine position. The posture does not affect the final parts of the Valsalva maneuver. It is better to perform the maneuver in a sitting position where the change in parameters is most prominent, except for the treatment purpose [10]. Although gender difference has no effect on Valsalva, different responses are obtained according to ethnic origin [11].

Valsalva maneuver deteriorates with or without heart disease due to sympathetic vasoregulation and desensitization of BR reflex mechanism in older ages. The increase in vagal bradycardia and BP is uncertain with age, whereas the decrease in BP becomes more pronounced. In the elderly, VM does not differ in patients without cardiac involvement and in those with chronic renal disease undergoing hemodialysis, liver cirrhosis and primary hyperaldosteronism [23,24]. Although the presence of parasympathetic denervation in patients with dilated cardiomyopathy due to Chagas disease can be demonstrated by VM and VR, it has been reported that the test response does not differ in patients without cardiac involvement and in those who develop Chagas myocarditis [25]. Response to VM may be affected by drugs such as alpha-1 and beta blockers angiotensin-converting enzyme inhibitors, diuretics, certain tranquilizing, and hypopotasemia [26-28]. Myocardial infarction also causes autonomic neuropathy in some patients and disrupts the response to VM. In cases of heart failure and evident left-to-right shunting, there is no response to Valsalva.
VM may aid in the differential diagnosis of cardiac murmurs. In hypertrophic cardiomyopathy and mitral valve prolapse, the murmur increases in the early stage of VM and decreases at the end of the maneuver, while in aortic stenosis, mitral stenosis, pulmonary stenosis, tricuspid stenosis, aortic insufficiency, mitral insufficiency, tricuspid insufficiency, pulmonary insufficiency and in ventricular septal defect, the murmur severity decreases during VM and increases after VM. Since the venous return returns to the left heart after pulmonary circulation, the murmurs of the right heart appear earlier than the murmurs of the left heart (Table 2).

| Table 2 | Changes in cardiac murmurs during Valsalva maneuver |
|-----------------|-----------------|-----------------|
| **Systolic Murmurs** | **During VM (Stage-2)** | **VM (Stage-4)** |
| Aortic Stenosis | ⇑⇓⇑⇑ | ⇑⇑⇓⇑ |
| IHSS | ⇑⇑⇓⇓ | ⇑⇑⇓⇓ |
| Pulmonary Stenosis | ⇑⇓ | ⇑⇓ |
| Ventricular Septal Defect | ⇑ | ⇑ |
| Mitral Failure | ⇑ | ⇑ |
| Mitral Valve Prolapse | ⇑ and early click | Late click |
| **Diastolic Murmurs** | | |
| Aortic Failure | ⇑ | ⇑ |
| Mitral Stenosis | ⇑ | ⇑ |
| Pulmonary Insufficiency | ⇑ | ⇑ |
| Austin Flint Murmur | ⇑ | ⇑ |

⇑: Increases  ⇑⇑: Increases significantly  ⇓: Decreases  ⇑⇓: Decreases significantly

It should be known that repeated Valsalva maneuver can cause a number of complications including pulmonary embolism [29]. Ocular complications such as large central preretinal and diffuse hemorrhages, vitreous hemorrhage and purtscher retinopathy may occur in addition to small premacular hemorrhages in the eye [30]. The most feared complication is cardiac arrest, which can occur during changes in hemodynamics.

Modified Valsalva

In the modified Valsalva maneuver, 45 degrees elevation is applied to the patient's feet immediately after the standard Valsalva maneuver is performed (Figure 2). Thus, venous return and vagal stimulation are increased during the relaxation phase. This increases the effectiveness of Valsalva in SVT. In the REVERT study examining the efficacy of the

Figure 1. Modified Valsalva maneuver is performed by applying a 45 degree height to the patient's feet immediately after the standard Valsalva maneuver is performed.
network. As a result of the study, it was observed that modified Valsalva maneuver increases tolerance to standard maneuver in patients with vasovagal syncope and reduces the risk of syncope formation [34].

**Conclusion**

Valsalva and modified Valsalva maneuvers are still essential, despite rapid advances in diagnosis and treatment. Valsalva and modified Valsalva maneuver are of great value, especially in the early treatment of SVT. It is also a good adjunctive diagnostic tool for the assessment of cardiac autonomic neuropathy caused by diabetes and neurological diseases. It provides important clues to the clinician in the evaluation of congenital heart diseases and heart valve diseases in cases where imaging methods are insufficient.

Nevertheless, these maneuvers have little diagnostic value. These maneuvers should not be used in coronary artery disease, decompensated heart failure, hemodynamic disorders and elderly patients. The clinician should be sensitive to patient selection. Modified Valsalva maneuver is preferable to Valsalva maneuver because of its higher efficacy and fewer side effects in the treatment of SVT.

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**References**


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