

# The Valsalva manoeuvre—cardiovascular effects and performance technique: a critical review

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## Abstract

Variations in the technique of the Valsalva manoeuvre (VM) have been shown to greatly influence the pattern of cardiovascular response (CVR) to the test. Intra-strain tachycardia, post-strain bradycardia, Valsalva ratio, and baroreflex sensitivity decrease in proportion to an increase in lung volume and a decrease in strain pressure at VM. In conditions of completely expanded lungs and low strain pressure many subjects reveal an intra-strain bradycardic response to VM instead of the usual tachycardic one. Intra-strain arterial hypotension and post-strain hypertension decrease with decrease in strain pressure. The changes in heart rate and blood pressure during an expiratory VM are greater than the responses observed during completion of an inspiratory VM. The rate of the deep inspiration prior to strain has an impact particularly on phase I of the VM. The magnitude of the CVR correlates with the strain duration, particularly at high levels of strain pressure, and depends on the baseline level of the cardiovascular parameters and their variations.

The paper discusses the possible mechanisms of different CVRs to variations in the technique of the VM. Some practical recommendations are suggested.

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## 1. Introduction

In 1851, Ernst Heinrich Weber reported a considerable deceleration or even a transitory arrest of the heart in response to the Valsalva manoeuvre (VM). Some years later, [Donders \(1854\)](#) reached the opposite con-

clusion finding a significant tachycardia as a typical response to the VM. The reason for this apparent discrepancy has remained unclear. One might assume, however, that some differences in experimental conditions and/or in the technique of the VM were involved. The next 150 years witnessed thousands of studies on cardiovascular effects of the VM in normal and diseased subjects. The studies took into consideration a number of experimental conditions and factors affecting the

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cardiovascular response (CVR) to the VM. Thus, it was suggested that the VM tests should be performed in the morning at least 2 h after a light breakfast. Subjects were requested to abstain from coffee and cigarettes on the previous evening. The room temperature and the subject's age were to be taken into account (Wieling, 1993).

Surprisingly, much less attention has been paid to the effects of the variations in the performance technique such as volume and rate of the prestrain breath, extent and rate of the strain pressure increase, changes in lung volume and strain pressure, duration of the strain period, depth and rate of the poststrain breathing. All these technical conditions have been shown to affect the patterns of CVR to the VM (Looga, 1970).

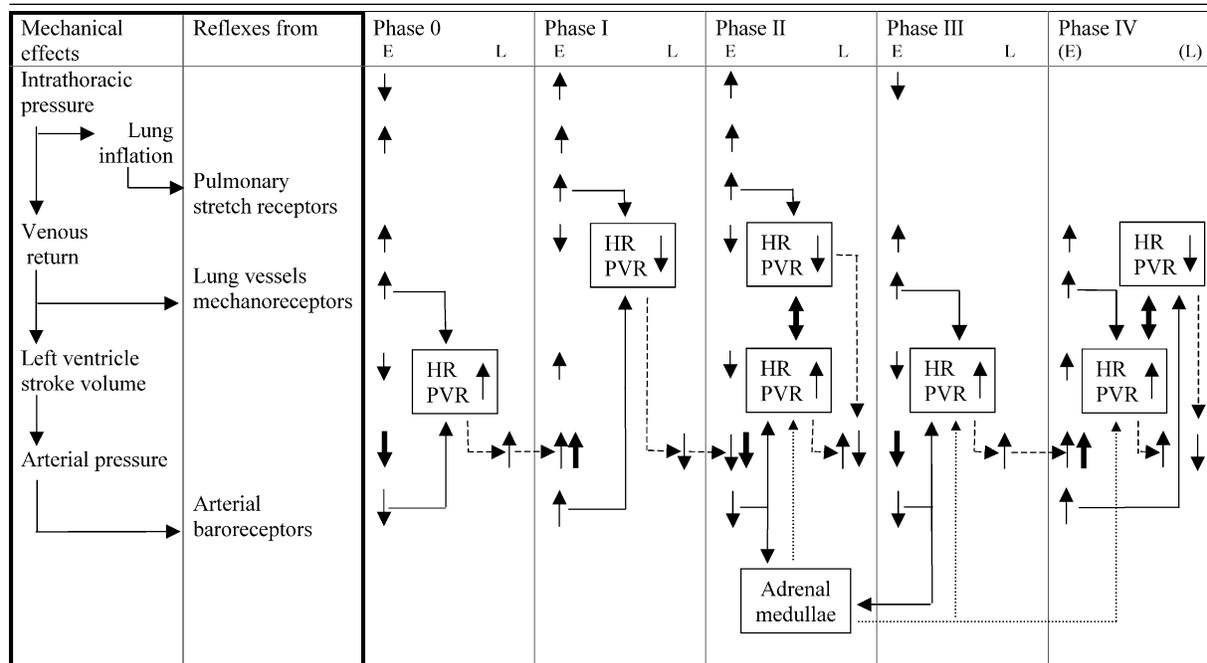
The purpose of the present paper is to review the cardiovascular effects and their mechanisms of some common but mostly neglected, variations in the performance technique of the VM.

## 2. Fundamental mechanisms of the cardiovascular response to the Valsalva manoeuvre

Table 1 presents a schematic overview on the fundamental mechanisms of the CVR to the Valsalva manoeuvre. According to Hamilton et al. (1936), this response is divided into four basic phases. If a final deep breath precedes the VM, as is usually the case, the concomitant CVR is treated as phase 0.

In all phases, the CVR may be divided further into an earlier subphase (E) and a subsequent later subphase (L). As a rule, the subphase E is related to the primary mechanical disturbance of arterial blood pressure by respiratory changes in intrathoracic pressure, and the subsequent secondary subphase L, to the reflex compensatory response to such disturbance. Input from the arterial baroreceptors plays the main role in this compensatory response (Sharpey-Schafer, 1965; Eckberg, 1980; De Burgh Daly, 1986). However, some

Table 1  
Fundamental mechanisms of cardiovascular response to the inspiratory Valsalva manoeuvre



E: mechanical impact on arterial blood pressure, and L: its subsequent reflex compensation; HR: heart rate; PVR: peripheral vascular resistance; bold, regular, interrupted, and dotted arrow: mechanical, neuroafferent, neuroefferent and hormonal impact on arterial pressure respectively; bold double arrow: competitive counteracting reflex impacts on arterial pressure.

other reflex influences, such as a depressor reflex from the slowly adapting pulmonary stretch receptors and a pressor reflex from lung blood vessel mechanoreceptors (Looga, 1997) may essentially modify the baroreceptor CVR to the VM.

The fall in arterial pressure in phase  $0_E$  seems to be the direct mechanical effect of decreasing intrathoracic pressure during deep inspiration. The following rise in the heart rate (HR) and peripheral vascular resistance (PVR) present a reflex-compensatory response by the arterial baroreceptors and mechanoreceptors of the lung vessels to the decrease in blood pressure and an increase in venous return. As a result, blood pressure rises (phase  $0_L$ ).

The phase  $0_L$  effect links up to the rise of blood pressure in phase  $I_E$ . The latter is induced by propelling a quantity of blood into peripheral arteries from heart and intrathoracic blood vessels under the direct mechanical action of the increased intrathoracic pressure (Eckberg, 1980; De Burgh Daly, 1986). In the following compensatory phase,  $I_L$ , the blood pressure and the heart rate start to fall under the stimulation of arterial baroreceptors and pulmonary stretch receptors.

Phase  $I_L$  links up to the mechanical decrease of the blood pressure in phase  $II_E$ . The latter is induced by a considerable decrease in the stroke volume of the left ventricle resulting from obstruction of the venous return by increased intrathoracic pressure (Fox et al., 1966; Smith et al., 1987). Usually phase  $II_E$  is evident due to its relatively long duration. Mechanical hypotension in phase  $II_E$  evokes a powerful compensatory reflex from the arterial baroreceptors: the HR and PVR start to rise, restoring gradually the baseline level of blood pressure. Under increased sympathetic nerve activity (Smith et al., 1996) the adrenal medulla is activated and large quantities of epinephrine and norepinephrine are released into the circulating blood (Sandroni et al., 2000). However, in the case of a deep inspiration prior to VM, the pulmonary stretch receptors are stimulated as well, and a significant depressor impact may restrict or suppress the pressor effect from the baroreceptors. Then a bradycardic response appears in place of tachycardia although the peripheral vasoconstriction still continues. Such situations have been observed by weak stimulation of baroreceptors (due to low strain pressure) with the concomitant vagotonic state of

autonomic activity (Looga, 2001). Thus, in phase II one can observe antagonistic influences from two sets of receptors.

The cessation of the VM strain evokes a sudden fall in the intrathoracic pressure with a simultaneous mechanical fall in arterial blood pressure (Eckberg, 1980; De Burgh Daly, 1986), phase  $III_E$ . The inhibited venous return is released again and blood flow rushes into lung vessels, the volume of which increases abruptly due to the first post-strain deep inspiration. A reflex compensatory response follows from arterial baroreceptors and from the mechanoreceptors of the pulmonary blood vessels, whereby the existing tachycardia and peripheral vasoconstriction from phase  $II_L$  are augmented still further (phase  $III_L$ ).

Next, there is an increase in diastolic filling and stroke volume of the heart. As the total peripheral resistance remains elevated, the ejection of the increased stroke volume into the constricted arterial system produces a sharp increase in arterial blood pressure, “overshoot” (Sarnoff et al., 1948), phase  $IV_E$ . The following reflex compensatory mechanism originates from arterial baroreceptors, phase  $IV_L$ . However, its impact presents itself mainly in the form of the bradycardia. Although the blood pressure falls significantly from the level of the overshoot, it still remains elevated for a considerable time. During this period the sympathetic nerve activity is low (Smith et al., 1996), and the post-strain hypertensive state is explained by continuing action of the adrenal medulla hormones (Sandroni et al., 2000).

Due to deep breathing and continuation of the increased venous return in phase IV (Fox et al., 1966; Smith et al., 1987) one might assume a pressor action from the mechanoreceptors of the lung blood vessels, which could counteract the baroreceptor effects. On the other hand, the latter might be supported by the vasodilator reflex from the thoracic aorta resulting from the increased pulse pressure in phase IV (Gruhzeit et al., 1954). Furthermore, the CVR mechanisms in phase IV may be further modified by changes in blood gases during the VM (Smith and Hatch, 1959; Meyer et al., 1966; Mateika et al., 2002).

In *summary*, the mechanisms of CVR to the VM are related to the neurohormonal response of the heart and blood vessels to the mechanical alterations in arterial pressure by changes in intrathoracic pressure before, during, and after the strain.

There are a number of *procedural technical factors* that may considerably modify the pattern and magnitude of CVR to the VM, provided the autonomic effector function is normal.

### 3. Prestrain final breath

Straining by VM may be initiated after a deep inspiration (inspiratory VM), at the end of expiration (expiratory VM), or at some intermediate level of the preceding inspiration (intermediate VM). Different researchers have employed one of these variants. However, most studies ignored the role of the depth and rate of the preceding breath in the pattern of CVR to VM. The completion of this breath has been commonly left to the discretion of the experimental subjects.

However, the depth of the preceding inspiration determines the volume of the lungs and intrapulmonary blood at the onset and during the VM. Also, it affects the level of blood gases and the straining force. These factors may induce various interactions between the inputs from different receptor fields, influencing the pattern of CVR to VM by the mechanisms of competition for the final common path (Looga, 1997). Relatively few authors have discussed the significance of the preceding breath for the CVR of the VM (Rushmer, 1947; Matthes, 1951; Looga, 1970; Mateika et al., 2002).

#### 3.1. Inspiratory VM

A single slowly performed (6–12 s) deep inspiration, particularly in subjects with a marked sinus arrhythmia, evokes mainly a two-stage CVR: a primary short (3–5 s) pressor reaction (steep rise in arterial pressure and heart rate) followed by a prolonged (5–15 s) depressor stage (fall in blood pressure and heart rate). Usually this response is preceded by a brief mechanical hypotension during 1–2 heart beats.

The pressor stage has been explained by baroreceptor and cardiopulmonary reflex mechanisms, including a pressor reflex from intrapulmonary vessels resulting from the increased blood flow into the lungs during the deep inspiration, and the depressor stage, by the compensatory baroreflex response to the first stage hypertension (Looga, 1997). When a deep inspiration precedes the VM, either the first or both stages may appear

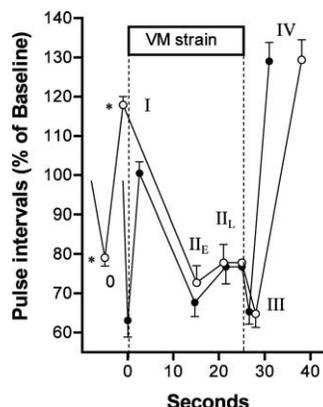


Fig. 1. The response of the heart rate (in term of its reciprocal, pulse interval) to the inspiratory Valsalva manoeuvre (VM) with slowly (open circles) or rapidly (filled circles) performed preceding deep inspiration; strain pressure 40 mmHg. The mean values of the sample points (the lowest pulse interval (PI) and blood pressure (BP) values in phases 0, II<sub>E</sub>, III, and highest PI and BP values in phases I, II<sub>L</sub>, IV) are plotted against time ( $n = 12$ ). Bars indicate  $\pm$ S.E.M. Asterisks (\*) mark values significantly different ( $P < 0.05$ ) from respective values of rapid deep inspiration (from Looga, 1970).

before the onset of the strain, depending on the duration of this breath.

Fig. 1 shows the heart rate response to slow and fast deep inspirations prior to VM. The tachycardic response component of the slow deep inspiration is less pronounced but lasts longer than of the fast deep inspiration. The second stage heart deceleration of the slow deep inspiration is considerable. It ceases with the onset of VM or even 1–2 s earlier. It seems to correlate with the bradycardic effect of phase I. Due to the short duration of the fast deep inspiration the second stage heart deceleration and the phase I bradycardia are small and often the latter does not appear at all. The arterial blood pressure during fast deep inspiration increases in a similar manner as in the first stage of the slow deep inspiration. However, in a number of subjects the blood pressure drops during the fast deep inspiration. This hypotensive effect may be explained by the direct mechanical impact of the brief but abrupt decrease in intrathoracic pressure on heart and great vessels. Due to the short duration of the inspiration, the increase in venous return is too small for triggering a cardiopulmonary pressor reflex. The concomitant tachycardic response may originate from arterial baroreceptors.

It is worth mentioning that in the case of normal tidal breathing the character of the blood pressure alterations

during the respiratory phases essentially depends on the breathing rate (De Burgh Daly, 1986).

The different speed of the deep inspiration prior to the strain seems to have a conspicuous effect on the CVR to the VM mainly in phase I. In the following phases of the VM (II–IV) its effect is insignificant, and a usual heart response occurs: intra-strain tachycardia and post-strain bradycardia. However, at low strain pressures (10–20 mmHg) many subjects (in our study 40 out of 75) reveal an obvious intra-strain bradycardia, although the classical four-phase course in blood pressure remains. The conditions favouring the intra-strain bradycardia include completely expanded lungs, a low expiratory strain pressure, and a vagotonic state. It has been suggested that the bradycardia is induced by a vagal reflex originating from the slowly adapting pulmonary stretch receptors (Looga, 2001).

### 3.2. Expiratory VM

In the conditions of the expiratory VM one can identify the usual four-phase course in blood pressure and heart rate. However, the changes are more pronounced than with the inspiratory VM (Rushmer, 1947; Matthes, 1951; Looga, 1970; Mateika et al., 2002).

In all phases of the expiratory VM the blood pressure is higher than in the corresponding phases of the inspiratory VM. Similarly the intra-strain tachycardia and the post-strain bradycardia of the expiratory VM is more pronounced than after inspiratory VM (Fig. 2). Often, if the expiratory VM lasts more than 10–15 s, the typical intra-strain tachycardia is replaced by an abrupt bradycardia and a steep rise in blood pressure, and the post-strain bradycardia is prominent and prolonged (Fig. 3).

The differences in CVRs to the inspiratory and expiratory VM have been explained in various ways: the generation of a gradient in pressure from the abdomen towards the thorax, which provides a more adequate venous return during the expiratory VM than during the inspiratory VM (Rushmer, 1947), the cessation of the input from pulmonary stretch receptors due to the low lung volume in expiratory VM (Looga, 1970, 2001) or changes in blood gases during the expiratory VM (Mateika et al., 2002). An integrated influence of all these mechanisms seems to be likely. Probably the changes in blood gases are

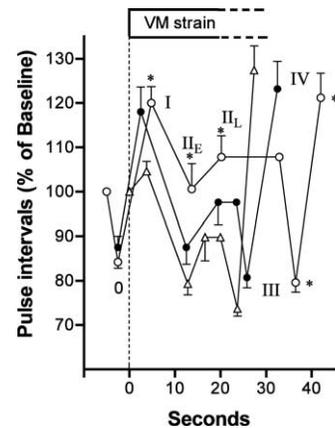


Fig. 2. The heart rate response to the inspiratory (open circles), intermediate (filled circles; 60% of the vital capacity), and expiratory (open triangles) Valsalva manoeuvre (VM); strain pressure 20 mmHg; duration of the strain was not controlled. The mean values of the sample points (see Fig. 1) are plotted against time ( $n = 13$ ). Bars indicate  $\pm$ S.E.M. Asterisks (\*) mark values significantly different ( $P < 0.05$ ) from respective values of expiratory VM (from Looga, 1970).

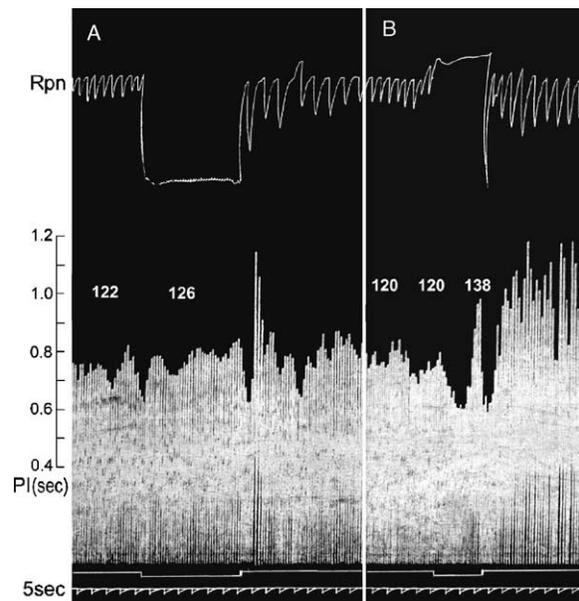


Fig. 3. The heart rate response to the inspiratory (A) and expiratory (B) Valsalva manoeuvre; strain pressure 20 mmHg. Tracings from above downward: breathing (Rpn, pneumograph; inspiration = downwards); pulse intervals (PI), equal to the reciprocal of heart rate; test signal; time marking with zero line of the PIs. The numbers above the PI tracing designate of blood pressure in mmHg (from Looga, 1970).

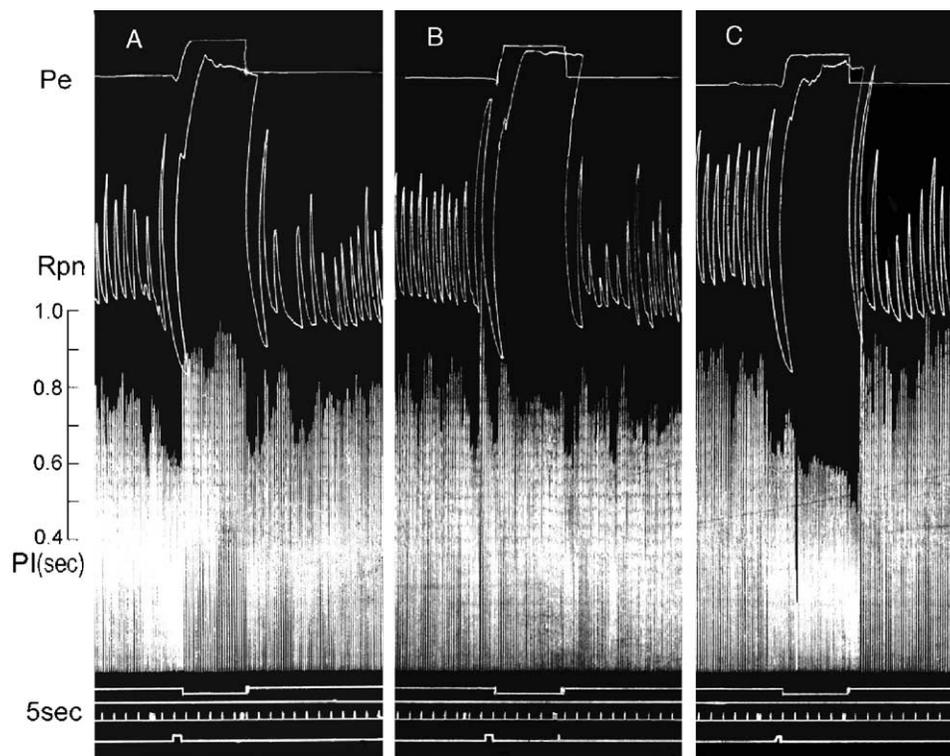


Fig. 4. The heart rate response to the inspiratory (A) and intermediate (60% (B) and 40% (C) of the vital capacity) Valsalva manoeuvre; strain pressure 30 mmHg. Tracings from above to downward: strain pressure (Pe); breathing (Rpn, pneumograph; inspiration = upward); pulse intervals (PI); test signal; zero line of PI; time marking and the signals for the start and end of the manoeuvre; inspiration from spirometer (from Looga, 1970).

mainly responsible for the conspicuous intra- and post-strain bradycardic responses.

### 3.3. Intermediate VM

The intermediate VM presumes control of the lung volume. It may be produced directly, in which case a subject expires maximally before the test and then inhales the required volume from the spirometer (Agostoni and Rahn, 1960), or indirectly by various techniques recording the respiratory movements and volume.

The pattern and magnitude of CVR to the intermediate VM are characterized by a tendency to increase the intra-strain and decrease the post-strain heart rate in proportion with the decrease of intra-strain lung volumes (Figs. 2 and 4). This effect may be explained by the same mechanisms with different intensity as in

the case of the expiratory VM. Some individual peculiarities may be observed in the intermediate VM. In some subjects the effect of the intermediate VM is revealed by a decrease in the lung volume by 5–10%, in others, only by a decrease of 50–60%. The effect is also more conspicuous in subjects with an intra-strain bradycardic response to the inspiratory VM (Fig. 4). The blood pressure response to the intermediate VM shows the usual four-phase course, but it does not show a definite correlation with the lung volume (Looga, 2001).

## 4. Strain pressure

The strain pressure indirectly determines the force of the straining action by VM. Usually it is measured by the elevation of the gas pressure in airways after a forced exhalation into a manometer to a predetermined

level and maintaining it there for a scheduled period. During this procedure the glottis must be open in order to prevent an unequal rise of pressures in the mouth and in the lungs. However, the glottis in subjects performing the VM may readily tend to be closed since it is easier to keep the predetermined pressure level by elevated intra-oral pressure alone without any simultaneous effort of the expiratory muscles. Moreover, the previous experience from everyday life may complicate the proper performance of VM since many physical activities are associated with straining with closed glottis (e.g. lifting of heavy weights).

In order to prevent the closure of the glottis during the VM some special methods have been developed. The most popular one is to provide the manometer system with a small air leakage (e.g. by inserting a small bore needle into the tubing of the manometer). Thus, during the VM a continuous air flow from alveoli is produced, preventing the closure of the glottis. Even a special device with an adjustable exhaust port has been constructed to permit the escape of air from the alveoli in a steady stream (Valentinuzzi et al., 1974).

However, even a moderate decrease in the lung volume accompanying this air-escape method may influence the pattern of CVR to VM (Looga, 2001). This shortcoming has been overcome in various ways: completing the VM the subject hold a tube, connected to manometer, deep (7.5–10 cm) in his/her mouth (Manzotti, 1958), or with a mouthpiece 20–30 mm in diameter, held by the subject between his/her teeth (Looga, 2001). Some authors believe that the glottis is open during the VM when the subject's face becomes flushed, neck veins distended, and a characteristic 'gasp' takes place on the release of strain (Baldwin and Ewig, 1977).

However, these methods can only help the cooperating subject to hold his/her glottis open during the VM and do not exclude the possibility for the subject to cheat, maintaining the manometer level high only by mouth pressure.

Obviously, the best way to control the VM strain pressure is a direct recording of the intrathoracic pressure (Hamilton et al., 1936; Sharpey-Schafer, 1965). However, the method is invasive and not free from hazards.

The usual level of the strain pressure, as employed in many VM studies, is 40 mmHg. The usual CVR to it consists of an intra-strain hypotension and tachycardia,

and a post-strain hypertension and bradycardia. However, sometimes at the inspiratory VM a remarkable intra-strain bradycardia has been observed instead of tachycardia (Bürger and Michel, 1957; Piha and Seppänen, 1991).

When the strain pressure is more than 40 mmHg, the frequency of the occurrence of this exceptional heart response shows a tendency to increase (Looga, 1970). An experimental study interprets the bradycardic response to a very high airway pressure as resulting from active suppression of the sympathetic reflex from baroreceptors and vagal activity. The site of the vagal reflex remains unknown (Hayashi, 1969). One might presume that the results of the classic study by Weber (1851) were elicited by the VMs conducted probably at a maximal strain pressure.

Systematic studies of the VM effects have shown that generally phase II hypotension and tachycardia, and phase IV hypertension and bradycardia increase with increasing strain pressure (Bürger and Michel, 1957; Korner et al., 1976; Benarroch et al., 1991; Smith et al., 1996). Therefore, the ratio between the values of the post-strain bradycardia and intra-strain tachycardia ("Valsalva ratio") increase with increasing strain pressure (Levin, 1966; Looga, 1970).

Fig. 5 shows the heart rate alterations in the phases of the inspiratory VM (I–IV) at different strain pressures. The magnitude and duration of phase II<sub>E</sub> tachycardia increases, and phase II<sub>L</sub> slowdown decreases in proportion to the rise in strain pressure. Phase III tachycardia and phase IV bradycardia increase, but phase I bradycardia decreases with increasing expiratory pressure.

Under the influence of low strain pressures (10–20 mmHg) a number of persons may reveal a uniform bradycardia during the entire effort of the inspiratory VM. On the other hand, in some subjects under the action of high strain pressures (30–50 mmHg) phase II<sub>L</sub> deceleration does not develop. A uniform tachycardia appears after the end of phase II<sub>E</sub> or the initial tachycardia increases gradually until the termination of the VM strain. During the post-strain period some subjects, particularly by performing a low-strain VM, do not have phase IV bradycardia. Instead a mild heart acceleration is observed, or there is no clear-cut response at all (Looga, 2001).

The rate of rise of the strain pressure at the start and rate of fall at the end of the VM may have an essential impact on the pattern of the heart rate response (Fig. 6).

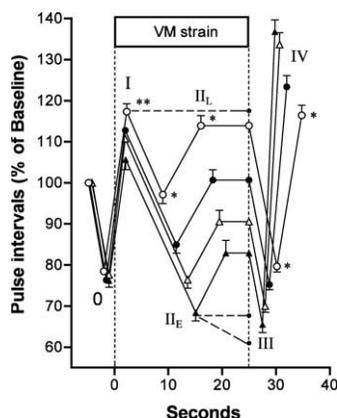


Fig. 5. The heart rate response to the inspiratory Valsalva manoeuvre (VM) at strain pressure 10 (open circles), 20 mmHg (filled circles), 30 mmHg (open triangles) and 40 mmHg (filled triangles). The mean values of the sample points (see Fig. 1) are plotted against time ( $n=75$ ). Bars indicate  $\pm$ S.E.M. Asterisks (\*) mark values significantly different ( $P<0.05$ ) from respective values of inspiratory VM at strain pressure 20, 30 and 40 mmHg (\*\* $P\leq 0.005$ )—from respective values at 40 mmHg. The course of special responses without phase II<sub>L</sub> are depicted arbitrarily by the broken lines (from Looga, 2001).

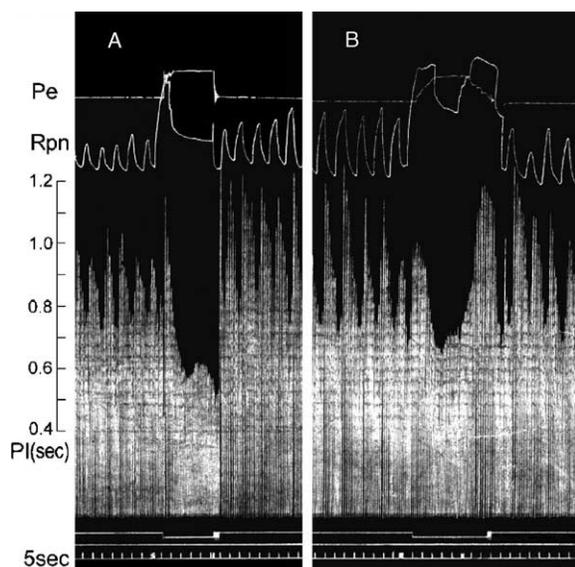


Fig. 6. The heart rate response to the inspiratory Valsalva manoeuvre with either fast (A) or slow (B) increase and decrease at a strain pressure of 40 mmHg. The succession of the records is as in Fig. 4 (from Looga, 1970).

The same is true of the effect of a sudden change in strain pressure during the VM.

## 5. Strain duration

In most VM studies the duration of the straining has been 15 s. During this period all intra-strain phases of the CVR to VM are usually distinguishable. If the duration is shorter, phases I and II<sub>E</sub>, or only phase I may be observed, and when longer, phase II<sub>L</sub>, and less often phase II<sub>E</sub> will be lengthened (depending mainly on the level of strain pressure; Fig. 7). If the duration of the VM is not fixed and the subjects are asked to perform the VM as long as they feel comfortable, the duration of inspiratory VM is much longer than that of the expiratory or intermediate VM (Looga, 1970; Fig. 2).

The strain duration has an essential influence on the magnitude of the CVR to VM in its basic phases (II and IV). Phase II tachycardia and arterial hypotension, and phase IV bradycardia and hypertension correlate with the duration of the straining, especially at high levels of strain pressure. The maximal increase in the heart rate during phase II correlates with the duration of the VM too, but only if performed at a high strain pressure

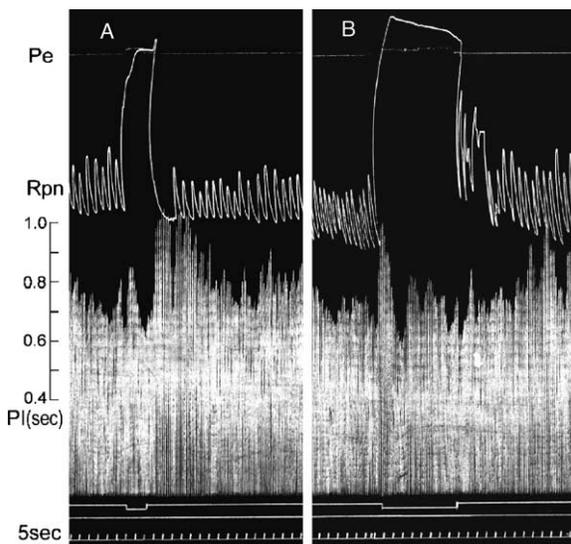


Fig. 7. The heart rate response to the inspiratory Valsalva manoeuvre lasting 10 s (A) and 40 s (B); strain pressure 20 mmHg. For symbols, see Fig. 4 (from Looga, 1970).

(Benarroch et al., 1991). The maximal decrease of the heart rate during phase IV is independent of the VM duration at both low (20 mmHg) and high (50 mmHg) expiratory pressures. On the other hand, the maximal changes in the mean arterial pressure in phase II<sub>L</sub> and phase IV significantly correlate with the duration of the VM at both low and high expiratory pressures. The same is true of the correlation between the recovery time of the responses to phase IV heart rate and blood pressure and the duration of straining (Benarroch et al., 1991). The Valsalva ratio gradually increases with an increase in the strain duration from 8 to 14 s (Levin, 1966). The time course of the CVR in separate VM phases varies with the degree of the strain pressure as well (Fig. 5).

The absence of correlations between the maximal heart rate response and the duration of the VM at low strain pressures can be explained by a significant individual variation in the patterns of the heart rate response during phases II and IV. During phase II this correlation may be concealed by the interactions of the mechanisms of the reflex tachycardia from baroreceptors and the reflex bradycardia from pulmonary stretch receptors (see Section 3). Not always is the onset of phase IV bradycardia as abrupt as it is usually depicted (Piha and Seppänen, 1991). When the VM is short (5–15 s), an abrupt onset of phase IV bradycardia is often observed. However, when the duration is longer (20–40 s), there is a gradual rather than an abrupt deceleration of the heart rate during phase IV (Fig. 7). The first effect may be explained by a vigorous baroreflex cardiac response to the suddenly elevated arterial pressure resulting from the injection of an increased left ventricular stroke volume into the arterial bed constricted during phase II. After the release of a prolonged VM as a result of intrastrain breathholding a period of immediate hyperventilation appears, which may considerably modify the baroreflex effect on the heart rate.

## 6. Baseline level

The magnitude and pattern of the CVR to the VM are usually estimated in reference to the baseline mean level. However, the baseline cardiovascular indices are not stable, showing perpetual regular and irregular fluctuations under the influence of various external and internal stimuli. Therefore, in all Valsalva studies efforts

have been made to exclude or diminish the influences of various erratic stimuli on the autonomic control.

According to one of the methods the subject rests in a quiet separate room at a table with a strain measuring manometer placed on it. The investigator with the recording device is located in the adjacent room. He observes the readings of the subject. When the baseline parameters are stabilized, he signals (for instance by an electrical buzzer) to the subject to start the VM (Looga, 1970). On the other hand, any such signal acts as a psychic stimulus which may disturb the stabilized baseline shortly before the VM (anticipatory effect). Therefore, some researchers have left the moment of the initiation of the test voluntarily to the subject him/herself without any command from the researcher (Nobrega et al., 1994). In order to avoid occasional changes in the breathing rhythm during the resting period prior to the VM a method of controlled respiration has been introduced. The subject breathes with the agreed depth and rhythm which he/she can control by an oscilloscope positioned in front of him/her and by beeping sounds (Smith et al., 1996; Guo et al., 1999). The method of controlled respiration is particularly suitable if the subject is located in the same room with the investigator and the equipment.

Nevertheless, the baseline values, determined by the above-mentioned method may show remarkable variations. In some subjects these values may differ markedly on different days or even during the day and affect the VM response. For instance, the higher the baseline heart rate, the lower the heart rate response in phase II and the higher in phase IV (Elisberg, 1963; Kalbfleisch et al., 1978; Piha, 1991). The intrastrain bradycardic responses are stronger if the preceding baseline heart rate is high and vice versa (Looga, 1970). Some attempts have been made to work out special formulae for the interrelations between the baseline values and test responses (Manzotti, 1958; Kalbfleisch et al., 1978).

## 7. Methodological recommendations

In order to stabilize the CVR to VM, one should control and record the breathing movements before, during, and after the test, including the depth and rate of the final inspiration prior to the onset of the strain. Also, the subjects are recommended to observe the course

of the baseline parameters and initiate the VM voluntarily by themselves without any command from the researcher. It is important to repeat the VM only after a full restoration of the baseline heart rate values from the influence of the previous test. Since the changes in blood gases during the VM may have an essential impact on the nature of the CVR, it is desirable to measure the respiratory and blood gases together with heart rate and blood pressure.

For an all-round study of the pattern and mechanisms of CVR to the VM it is reasonable to employ a battery of VM tests: at random, inspiratory and expiratory VM with high (30–40 mmHg) and low (10–20 mmHg) strain pressures. The inspiratory VMs should reveal the interactions between the two antagonistic autonomic reflexes, from the arterial baroreceptors and from slowly adapting pulmonary stretch receptors, and indirectly the functional state of the sympathetic and parasympathetic cardiovascular innervation. The role of the changes in blood gases and in venous return during the strain may be studied in expiratory VMs. The heart rate responses are considered to provide more valuable information than blood pressure responses.

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