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THE VALSALVA MANOEUVRE A CRITICAL REVIEW

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Abstract

The Valsalva manoeuvre is commonly used in diving to equalise middle ear pressures during descent. A forceful expiration with the nostrils and mouth held shut results in an increased nasopharyngeal pressure and opening of the Eustachian tubes. The correctly performed manoeuvre is easily taught, effective and usually without complications.

When performed incorrectly, prolonged periods of raised intrathoracic pressure may lead to decreased venous return, decreased arterial pressure and increased pressures within the superior and inferior vena cavae. An intact autonomic nervous system will initiate compensatory cardiovascular reflexes. The manoeuvre has clinical and research uses which rely on induced physiological changes and the initiation of reflex responses. The physiology and clinical uses of the manoeuvre are discussed.

The inappropriate use of the manoeuvre has been associated with significant morbidity. This includes pulmonary and aural barotrauma, hyper- and hypotension,

cardiac arrhythmias, arterial and venous haemorrhage, gastric reflux and stress incontinence. The complications of the manoeuvre are discussed.

Introduction

The Valsalva manoeuvre was first described in 1704 by the Italian physician Anton Maria Valsalva (1666-1723) as forced expiration against a closed glottis. For divers it is the process of making a forceful attempt at expiration while holding the nostrils closed and keeping the mouth shut for the purpose of adjusting middle ear pressure.

For many divers the manoeuvre is their only means of equalising middle ear pressures upon descent. However, it is not without its dangers and the inappropriate use of the manoeuvre has been associated with significant morbidity and even mortality. The purpose of this review is to summarise the physiological changes which occur during a forceful Valsalva manoeuvre and to describe some of the documented dangers and complications.

To put this into its proper perspective, the vast majority of manoeuvres are performed correctly and without the generation of intrathoracic or intravascular pressures likely to lead to complications.

Physiology

A standardised Valsalva manoeuvre has been described and is divided into four phases.¹ The patient is requested to exhale against a resistance of 40 mmHg for 20 seconds while heart rate and blood pressure are monitored. During Phase 1 of the manoeuvre, the increase in intrathoracic and intra-abdominal pressure will cause aortic compression and an increase in peripheral resistance resulting in a transient increase in blood pressure.

The increase in intrathoracic pressure during Phase 2 (maintenance or strain phase) hinders venous return to the heart and pressures in the superior and inferior vena cavae are increased.¹⁻³ The decrease in venous return leads to a decrease in ventricular end-diastolic volume, cardiac output and consequently systolic arterial pressure.^{1,3-7} This fall in arterial pressure is detected by baroreceptors in the carotid artery sinuses and results in a decrease in afferent nervous discharge from the sinuses to the brain stem via Herring's and then the glossopharyngeal nerves. The glossopharyngeal nerves relay in the nucleus tractus solitarius and the decrease in their rate of discharge has an inhibitory effect upon the vagus nerve centre (parasympathetic) and an excitatory effect upon the vasomotor centre (sympathetic). This results in a reflex tachycardia and peripheral vasoconstriction after about seven seconds of strain.^{1,3} The increased pressures within the vena cavae are transmitted in a retrograde fashion along

the venous blood column. This results in raised venous pressures especially within the head and neck, genitalia and the limbs.

Phase 3 (release phase) begins with the cessation of forced expiration and leads to a further decrease in blood pressure as the pressure surrounding the aorta decreases. During Phase 4 (overshoot phase), the accumulated venous blood is pumped by the heart into the constricted vascular bed causing an "overshoot" of arterial pressure above the normal level.^{1,3,5,6} This is detected by the carotid sinuses and results in a reflex bradycardia, mediated by the pathway described above. Ultimately, with the return of normal respiration and intrathoracic and intra-abdominal pressures, arterial and venous pressures and pulse rate return to baseline levels.

Clinical uses

The Valsalva manoeuvre is familiar to all those who have undertaken scuba diving where it is commonly used in the equalisation of middle ear pressures during descent. The manoeuvre increases air pressure in the nasopharynx which separates the Eustachian tube cushions and forces air into the middle ear. The pressures required to do this vary from 20 to 100 cmH₂O.⁸ As the glottis must be open to allow transmission of the increased intrathoracic pressure to the nasopharynx, the mouth and nares need to be held shut in order to prevent the escape of air pressure. The manoeuvre is frequently used in other situations where the ambient atmospheric pressure changes rapidly e.g. descent when flying.

It is generally accepted that the Valsalva manoeuvre should be used in the examination of Eustachian tube function as part of the pre-diving medical examination. Indeed, Moser and Wolf described the Valsalva manoeuvre, with otoscopic visualisation of the excursion of the eardrum, as the most reliable test of tubal function.⁹ However, while this manoeuvre is simple and accessible to the examining physician, it can prove frustrating if eardrum excursion is not demonstrated, particularly if the history from the examinee suggests no difficulty in the equalisation of middle ear pressures during ambient pressure change. In this situation the physician has not been able to demonstrate Eustachian tube function objectively and may elect to try other manoeuvres, re-examine at a latter date or refer the examinee for specialist ear, nose and throat examination. Some physicians will rely solely upon the history in the assessment of Eustachian tube function. While this approach may be considered unacceptable by some, others consider it as a practical necessity and draw attention to the work of McNicholl who concluded that immobility of the tympanic membrane on performance of the Valsalva manoeuvre does not necessarily denote the presence of Eustachian tube dysfunction.¹⁰

Among the most common uses of the Valsalva manoeuvre in clinical medicine is in the management of paroxysmal supraventricular tachycardia. Upon termination of a prolonged and forceful manoeuvre, the vagally-induced reflex bradycardia seen during phase 4 is accompanied by decreased conductance through the atrio-ventricular node which often is sufficient to interrupt and terminate the circus movement responsible for the arrhythmia.^{11,12}

The Valsalva manoeuvre can be used to test both the sympathetic and parasympathetic divisions of the autonomic nervous system. With sympathetic dysfunction, the fall in blood pressure occurring during phase 2 may not be followed by reflex tachycardia or vasoconstriction.¹ Consequently, the blood pressure increase at the start of phase 4 will be attenuated. With parasympathetic dysfunction, the baroreceptor-mediated reflex bradycardia response to the elevated blood pressure in phase 4 will not occur.¹ Accordingly, the manoeuvre is useful in the investigation and quantification of generalised autonomic failure, autonomic neuropathies, distal small-fibre neuropathy and adrenergic function.¹³⁻¹⁶ It is of particular importance in the assessment of cardiovascular and anaesthetic risk in diabetic patients.^{17,18}

Other uses for the manoeuvre in clinical practice rely on venous and intracardiac pressure changes. They include the evaluation of cardiac murmurs and left ventricular function,¹⁶ diagnosis of congestive cardiac failure,¹⁹ facilitation of insertion of central venous catheters,²⁰ enhancement of lower limb colour Doppler flow imaging²¹ and the ultrasonographic detection of biliary obstruction.²² Furthermore, the manoeuvre is being used clinically in the investigation of atrial septal defects (ASD) and patent foramen ovale (PFO) where the intrathoracic pressure changes lead to a transient inversion (right atrial pressure higher than left atrial pressure) of the inter atrial pressure gradient. This leads to flow of blood from the right atrium (venous blood) directly into the left atrium and into the arterial circulation thereby bypassing the lungs. This arterialed venous blood can be detected using contrast transthoracic and transoesophageal echocardiography,²³⁻²⁵ oxygen saturation step-up analysis²⁵ and transcranial Doppler sonography after galactose or saline microbubble injection.^{26,27}

A modified Valsalva manoeuvre is often used to induce changes in intraocular pressure and amplitude of tonography pulse pressure which aid in the diagnosis of assorted ocular disorders.²⁸

Complications

Complications associated with the Valsalva manoeuvre are usually only seen when it is performed either too forcefully or for too long a period. These

complications are a consequence of the exaggerated physiological changes which occur, in particular the rises in intravascular, intrathoracic (pulmonary) and intra-abdominal pressures and the vascular reflexes.

Not surprisingly, the exaggerated cardiovascular reflexes induced by a forceful and prolonged manoeuvre have been associated with significant morbidity and even mortality. Most of the observed complications are consequent upon the induced hypertensive reflex. The hypertension induced by resistance exercise has been shown to be exaggerated by the accompanying Valsalva manoeuvre²⁹ and even symptomatic severe paroxysmal hypertension has been attributed to subconscious Valsalva-like manoeuvres.³⁰ Not all symptoms arise from induced hypertension and significant hypotension can be induced during the strain phase of the manoeuvre (phase 2) as a consequence of greatly diminished venous return.³¹

In subjects with fixed stenoses of the coronary vasculature, where myocardial blood flow is compromised, it is conceivable that myocardial ischaemia may be precipitated by either hypotension during the Valsalva strain phase or an increase in the myocardial oxygen demand/supply ratio caused by the hypertension induced upon release. However, it is unlikely that these symptoms would be seen without either repeated and prolonged manoeuvres or associated physical activity. Kern et al., in a study of subjects with normal coronary arteries, showed that despite a marked reduction in the mean arterial pressure during the Valsalva strain phase, the reduction of coronary blood flow velocity was not significant.⁴

Interest has recently been paid to the dangers of an ASD or PFO in scuba diving. These lesions may result in paradoxical gas embolization and serious morbidity.³² This situation may occur in a diver suffering decompression sickness where venous bubbles pass into the arterial circulation during the transient reversal of blood flow through an ASD or PFO following a Valsalva manoeuvre. Wilmshurst et al., in a study of divers with a large PFO, have suggested that the foramen may be associated with clinically significant arterial desaturation and unusual responses of heart rate and blood pressure after the performance of a Valsalva manoeuvre.³³

The Valsalva manoeuvre has been associated with cardiac arrhythmias and rarely even sudden death.³⁴⁻³⁶ Piha et al. assessed the possible dysrhythmogenic effect of cardiovascular autonomic function tests, including the Valsalva manoeuvre, and documented the precipitation of ventricular extrasystoles and non-sustained tachycardia, conduction block and atrial fibrillation.³⁵ While the precipitation of some arrhythmias is likely to be associated with significant alterations in vagal tone, Taggart et al. have suggested that Valsalva-induced changes in ventricular loading influence cardiac repolarisation.³⁶ They found that when ventricular wall motion was abnormal the

effects on regional endocardial repolarisation were often opposite in direction to those when motion was normal. Thus, regional differences in wall motion could generate local electrophysiological inhomogeneity which may be relevant to the association of arrhythmia with impaired left ventricular function.

Most neurological symptoms associated with the Valsalva manoeuvre are the result of the drop in mean arterial blood pressure during the strain phase. Cough and defaecation syncope are well-described syndromes secondary to Valsalva-like manoeuvres.^{37,38} Significant decreases in cerebral perfusion pressures have been demonstrated clinically during the strain phase^{6,7} and, while a modest but significant decrease in vascular resistance has been demonstrated during this phase, it is not rapid enough or of sufficient magnitude to maintain constant cerebral perfusion.⁶ Significant blood pressure rises following the release phase of the Valsalva manoeuvre can produce rapid shifts in cerebrovascular blood flow which have been implicated in cerebral aneurysm rupture and rebleeding.^{39,40} Valsalva-induced fluctuations in cerebrospinal fluid pressure around an aneurysm may also play a part in its rupture.⁴⁰ The Valsalva-like manoeuvre of nose blowing is thought to be associated with some cases of spontaneous rhinorrhoea. The presence of congenital dehiscences and the formation of small meningoceles which rupture during nose blowing is thought to be the aetiological basis.⁴¹

While the increase in intrathoracic pressure during the strain phase of the Valsalva manoeuvre has been shown to decrease pulmonary venous flow,⁴² airways mucosal blood flow⁴³ and the size of the upper airways,⁴⁴ perhaps the most important respiratory complications result from barotrauma. There have been many reports describing Valsalva-induced alveolar rupture with consequent pneumothorax, pneumomediastinum, pneumopericardium and subcutaneous emphysema.⁴⁵⁻⁵⁰ Curiously, pulmonary barotrauma has been reported following the smoking of marijuana and cocaine and it is considered that the Valsalva manoeuvre, often performed in an attempt to increase the effects of these drugs, has led to the rupture of marginal alveoli. The concurrent use of tobacco with these drugs is thought to accelerate lung disease and the likelihood of pulmonary barotrauma.^{49,50}

Damage to the middle and inner ear following a forceful Valsalva manoeuvre during a diving descent has been well documented.^{8,51} There are three postulated mechanisms for labyrinthine round window fistulae during diving. Firstly, sudden descent without volume equalisation forces the eardrum in and the force is transmitted through the ossicles to the stapes which raises the perilymph pressure sufficiently to rupture the round window. Secondly, the sudden clearance of a blocked Eustachian tube allows the transmission of the high nasopharyngeal pressures into the middle ear chamber, driving the

tympanic membrane outwards and jerking the stapes outwards. As the inner ear is effectively a closed system, if the movement is large enough the round window is subjected to an inwards pull beyond the elastic limits of its membrane leading to a tear. This is described as implosive rupture of the round window in the ENT literature. The third possibility is a rupture (described as explosive in the ENT literature) caused by the transmission of a pressure wave in the cerebrospinal fluid through a widely patent cochlea aqueduct during a forceful manoeuvre.^{8,52} Round window fistulae, oval window fistulae, probably due to damage from the stapes footplate, cochlear and vestibular haemorrhage and internal inner ear membrane ruptures have all been reported after forceful Valsalva manoeuvres.⁸

The term "Valsalva haemorrhagic retinopathy" refers to haemorrhage in and around the macula in response to a sudden rise in intrathoracic or intra-abdominal pressure during the Valsalva strain phase. The increased intravenous pressure is felt to be transmitted to the retinal circulation with resulting retinal capillary rupture.⁵³ Numerous reports of ocular haemorrhage have been made including retinal artery macro-aneurysms, suprachoroidal, preretinal, retinal and vitreous haemorrhages, cilio-choroidal detachments and haematomata of the lids.⁵³⁻⁵⁷

Complications of the Valsalva manoeuvre involving the gastrointestinal and genito-urinary tracts are uncommon and are the result of the increased intra-abdominal pressures which are generated. The manoeuvre has long been associated with gastroesophageal reflux and stress incontinence of urine and more recently it has been reported that reversal of scrotal vein blood flow may occur leading to the development of scrotal varicoceles or even their rupture.⁵⁸⁻⁶⁰

Summary

It is appropriate that diving physicians understand the physiology and pathophysiology of the Valsalva manoeuvre as this knowledge is relevant to their clinical practice. Diving instructors must also be cognizant of the dangers of the manoeuvre and must instruct their students in its correct and appropriate use.

Whilst the Valsalva manoeuvre will remain as the most commonly used means of equalising middle ear pressures, consideration should be given to safe alternatives. The "Frenzel manoeuvre" is a technique which involves closing the mouth and nose, both externally and internally (this is achieved by closing of the glottis) and then contracting the muscles of the floor of the mouth and the pharyngeal constrictors. Thus, the nose, mouth and glottis are closed and the elevated tongue can be used as a piston to compress the air trapped in the nasopharynx and

force it into the Eustachian tube. Pressure of less than 10 cm H₂O are required to achieve this manoeuvre.⁸ This technique has the advantage of avoiding large changes in intrathoracic and intra-abdominal pressures and the consequent physiological changes and reflexes. It also avoids rapid increases in middle ear pressures with the sudden opening of one or both Eustachian tubes. While this technique is more difficult to teach, it is often performed subconsciously by the more experienced diver or learned by novice divers as a modification of a "deliberate swallow", itself a safe and often effective technique used to equalise middle ear pressures. Yet another technique is to hold one's nose, shut one's mouth and then blow gently and swallow while blowing.

It has been shown that a prolonged or forceful Valsalva manoeuvre is accompanied by complicated and significant physiological change and that many complications of the manoeuvre have been documented. However, it needs to be emphasised that complications of the manoeuvre are rare and that the correct use of the manoeuvre by the sports diver is both safe and effective. It is unlikely that any diver would suffer from a complication of the manoeuvre if it were performed for very short durations and if forceful attempts were avoided.

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THE WORLD AS IT IS

ACTION DOWN UNDER

Richard Moon

While most scholarly medical societies are organised by physicians. I recently attended a meeting of a society in which nurses and hyperbaric technicians have

taken the lead. The September 1995 meeting of the Hyperbaric Technicians and Nurses Association (HTNA), with Dave King as President, superbly hosted by the Alfred Healthcare Group Hyperbaric Service in Melbourne, Australia, was a winner.

While the setting for the gathering was unsurpassed, it was more than matched by the content. Among many