Middle cerebral artery blood velocity during a Valsalva maneuver in the standing position

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Middle cerebral artery blood velocity during a Valsalva maneuver in the standing position. J Appl Physiol 88: 1545–1550, 2000.—Occasionally, lifting of a heavy weight leads to dizziness and even to fainting, suggesting that, especially during standing, the hemodynamic changes associated with straining against a closed glottis would compromise blood supply to the brain. We hypothesized that, especially during standing, the hemodynamic changes associated with straining against a closed glottis would compromise blood supply to the brain. We evaluated the effects of a supine and an upright Valsalva maneuver on MCA Vmean and determined CVP to assess the significance of the Valsalva-induced elevation in intrathoracic pressure on the cerebral perfusion pressure.

METHODS

Subjects and experimental protocol. Ten subjects [4 women and 6 men; age 25 (21–38) yr (median with range), weight 79 (50–90) kg, and height 183 (162–196) cm] participated in the study after giving informed consent to the protocol, as approved by the Ethics Committee of Copenhagen (KF 01–120/96). A mouthpiece was connected to a manometer, and the subjects were instructed to maintain an expiratory pressure of 40 mmHg for 15 s (28). A small leak in the tubing prevented the subjects from maintaining the pressure by closing the glottis, and care was taken to prevent deep breathing before and after the release of the strain. After instrumentation, the subjects were allowed to rest in the supine position for at least 30 min. After a test run, subjects rested for 5 min and performed three Valsalva maneuvers, each followed by 3 min of recovery. Subjects were then asked to stand up in a relaxed position, and, after 5 min, they performed three Valsalva maneuvers, each followed by 3 min of recovery.

Transcranial Doppler. The proximal segment of the right MCA was insonated (Multidop X, DWL, Sipplingen, Germany) through the posterior temporal “window.” Once the optimal signal-to-noise ratio was obtained, the probe was covered with adhesive ultrasonic gel (Tensive, Parker Laboratories, Orange, NJ) that served for fixation to the skin. For further stabilization, a rubber band (DWL) was strapped around the head and the transducer. If necessary, tape was used to further fix the probe to the skin. Signal quality was evaluated during vigorous movement of the head, including bending forward and backward as well as shaking the head. By visual judgment of the Doppler signal, it was accepted when no interruptions or obvious artifacts of the spectral outline occurred. Vmean was computed as the integral of

LIFTING OF A HEAVY WEIGHT may lead to a “blackout” (5), coughing may induce a syncope (15), and wind instrument players too may experience occasional fainting (3). These experiences suggest that, especially during standing, the expiratory strain of a Valsalva-like maneuver may critically reduce blood flow to the brain. A forced expiration against a closed glottis (a Valsalva maneuver) leads to a proportional elevation in the intrathoracic and central venous pressures (CVP), a marked reduction in cardiac output (CO), and characteristic changes in mean arterial pressure (MAP). Such hemodynamic alterations have consequences for the perfusion of the brain, as, even during a Valsalva maneuver performed in the supine position, the transcranial Doppler-determined middle cerebral artery (MCA) mean blood velocity (Vmean) is reduced by ~35% (29).

In healthy subjects, passive head-up tilt reduces MCA Vmean (4, 12) and cerebral O2 saturation (14). Presyncopal symptoms appear when MCA Vmean is reduced by ~50% (4, 12). These results indicate that, during standing, the downward shift in the distribution of the blood volume and the resulting decrease in CO may compromise cerebral perfusion. We hypothesized that, especially during standing, the hemodynamic changes associated with straining against a closed glottis would compromise blood supply to the brain. We evaluated the effects of a supine and an upright Valsalva maneuver on MCA Vmean and determined CVP to assess the significance of the Valsalva-induced elevation in intrathoracic pressure on the cerebral perfusion pressure.

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maximal frequency shifts over one beat divided by the corresponding beat interval. Cardiovascular monitoring. Finger arterial pressure was measured with a Finapres model 5 (Netherlands Organization for Applied Scientific Research, Biomedical Instrumentation; TNO-BMI). The cuff was applied to the midphalanx of the middle finger of the dominant hand and placed at heart level. Beat-to-beat systolic and diastolic pressures, as well as MAP, were computed after analog-to-digital conversion at a sampling rate of 100 Hz. MAP was obtained as the integral of pressure over one beat divided by the corresponding beat interval. Heart rate (HR) was the inverse of the interbeat interval. A catheter (1.7 mm ID, 16 gauge) was placed in the superior caval vein through the basilic vein for CVP and interval. A catheter (1.7 mm ID, 16 gauge) was placed in the superior caval vein through the basilic vein for CVP and central venous O2 saturation monitoring (SVo2). CVP was recorded from a transducer (Bentley, Uden, The Netherlands) fastened to the subject in the midaxillary line at the level of the right atrium and connected to a monitor (8041, Simonsen & Wed, Copenhagen, Denmark). Stroke volume (SV) was obtained from the arterial pressure pulse wave by model flow analysis (FAST-m/-CZ system, TNO, Amsterdam, The Netherlands). This method computes an aortic flow waveform by simulating a nonlinear, time-varying model of the aortic input impedance. The resulting CO correlates to a determination based on thermodilution (9). CO was the product of SV and HR and was expressed relative to control. The central blood volume was monitored by thoracic electrical impedance (TI) measured between skin electrodes employing 10 mA at 100 kHz (model 304 B, Minnesota Impedance Cardiograph, Sorcom, Minneapolis, MN). Two electrodes were positioned 5 cm above each other behind the right sternocleidomastoid muscle, and two other electrodes were placed at a similar distance in the left midaxillary line at the level of the xiphoid process. The outer electrodes served for current, whereas the inner pair recorded TI. An inverse correlation between TI and the central blood volume has been established during head-up tilt (20). Furthermore, during head-up tilt, TI is sensitive to changes in the distribution of technetium-labeled red cells (17) and also to the release of atrial natriuretic peptide from the right side of the heart (16, 21). We limited the use of TI as a marker of central blood volume to supine and standing baseline measurements. During the transition from normal breathing to strenuous expiratory straining, impedance would be expected to change independently of the central blood volume, as both an increase in electrode distance and the increased volume of air contained in the thorax would elevate TI.

Blood gases. A 1.0-mm ID (19 gauge) catheter was placed in the brachial artery of the nondominant arm for arterial carbon dioxide tension (PaCO2) and O2 saturation. For one Valsalva maneuver under each condition, blood samples were taken anaerobically in heparinized syringes (QS50, Radiometer, Copenhagen, Denmark) at rest, during the last seconds of the strain, and after ~10 s of recovery. Samples were analyzed immediately by spectroscopy (OSM-3,ABL, Radiometer).

Data analysis. Tracings of the variables were checked for artifacts and transformed to equidistantly resampled data at 2 Hz by polynomial interpolation, and for each subject the three runs were averaged as triggered by the onset of the increase in CVP. The circulatory response to the Valsalva maneuvers was divided into four phases (8, 25). A transient increase in MAP at the onset of the strain reflects transmission of the elevated intrathoracic pressure to the arterial tree (phase I) followed by a decrease in MAP, pulse pressure, and SV due to the reduced atrial filling (phase IIa) with partial recovery of MAP and HR toward the end of the strain (phase IIb). After release of the strain, MAP drops for 1–2 s as blood pools in the distended pulmonary vascular bed (phase IIII) followed by a pressure overshoot (phase IV) as an elevated CO is expelled against a constricted vascular bed. Values are presented as means with standard error. The Friedman test was used to determine whether significant changes occurred with time or between circumstances, and such changes were located with Wilcoxon’s matched-pair signed-rank test. To evaluate the significance of the Valsalva elevation in CVP for perfusion pressure to the brain, linear regression analysis was applied between MCA Vmean and MAP, and between MCA Vmean and the MAP-to-CVP difference (MAP-CVP). A P value of <0.05 was considered significant.

RESULTS

We observed a stable transcranial Doppler signal quality during the supine-to-standing transition, during standing, and also during the Valsalva maneuvers. Supine Valsalva maneuver. Table 1 lists the hemodynamic variables corresponding to each phase as defined from the changes in MAP. During straining, CVP

| Table 1. Systemic and cerebral hemodynamic variables corresponding to 5 phases of the Valsalva maneuver |
|-------------------------------------------------|-------------------|-------------------|-------------------|-------------------|-------------------|
| Position | Baseline | I | IIa | IIb | III | IV |
| MAP, mmHg | Supine | 89 ± 2 | 112 ± 4* | 87 ± 3 | 111 ± 6* | 95 ± 5 | 108 ± 5* |
| Standing | 78 ± 3| 93 ± 6† | 65 ± 6† | 85 ± 7† | 65 ± 6* | 90 ± 4* |
| CVP, mmHg | Supine | 2 ± 1 | 38 ± 4* | 43 ± 3* | 45 ± 4* | 3 ± 1 | 2 ± 1 |
| Standing | 2 ± 2 | 41 ± 3* | 42 ± 3* | 46 ± 4* | 2 ± 2 | -2 ± 2† |
| HR, beats/min | Supine | 74 ± 2 | 78 ± 4* | 95 ± 4* | 116 ± 5* | 118 ± 6* | 92 ± 8* |
| Standing | 95 ± 3 | 90 ± 3† | 109 ± 3† | 131 ± 3† | 133 ± 3† | 96 ± 6 |
| ΔSV, % | Supine | 100 | 97 ± 7 | 53 ± 5* | 37 ± 3* | 44 ± 5* | 87 ± 4* |
| Standing | 64 ± 3| 65 ± 6| 25 ± 2* | 23 ± 2* | 28 ± 2* | 84 ± 9 |
| ΔCO, % | Supine | 100 | 109 ± 10 | 65 ± 5* | 57 ± 4* | 69 ± 9* | 103 ± 7* |
| Standing | 84 ± 4| 83 ± 9| 37 ± 3† | 42 ± 5† | 48 ± 5† | 107 ± 9* |
| Vmean, cm/s | Supine | 73 ± 4 | 80 ± 5* | 47 ± 4* | 73 ± 5 | 70 ± 5* | 101 ± 7* |
| Standing | 62 ± 5| 76 ± 6* | 39 ± 5† | 62 ± 5* | 53 ± 4† | 98 ± 7* |

Values are means ± SE. MAP, mean arterial blood pressure at middle cerebral artery (MCA) level; CVP, central venous pressure; HR, heart rate; ΔSV and ΔCO,%changes in model flow stroke volume and cardiac output, respectively; Vmean, MCA mean blood velocity. Values presented correspond to the changes in MAP and reflect peak changes in Vmean. *Significantly different from baseline (P < 0.05). †Significantly different from the supine position (P < 0.05).
increased immediately to 38 ± 4 mmHg and continued to increase toward the end of the strain (45 ± 4 mmHg). MAP demonstrated the characteristic changes with an increase in phase I (23 ± 4 mmHg) followed by a reduction close to baseline (phase IIa) and a subsequent recovery in phase IIb (22 ± 6 mmHg above baseline; Fig. 1). In phase III, MAP dropped to the baseline level, which was followed by an overshoot in phase IV (19 ± 5 mmHg).

During the maneuver, CO decreased to 57 ± 4% of the baseline value until just before the release of the strain (Table 1, Fig. 1). SvO₂ was not influenced significantly, whereas arterial O₂ saturation became slightly elevated at the end of the maneuver and PaCO₂ was reduced (Table 2).

Compared with MAP, MCA Vmean reached the distinct phases of the Valsalva maneuver ~0.5 s earlier. Peak changes in MCA Vmean were a slight increase in phase I (+10 ± 2% Table 1), a drop in phase II (~35 ± 4%), and an overshoot after the release of the strain (+40 ± 8%). Phase III could not be detected in MCA Vmean.
Table 2. Arterial and central venous blood-gas variables during a Valsalva maneuver in the supine and standing position

<table>
<thead>
<tr>
<th>Position</th>
<th>Baseline</th>
<th>Valsalva Maneuver</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>SaO₂, %</td>
<td>Supine</td>
<td>97.6 ± 0.2</td>
<td>98.7 ± 0.2*</td>
</tr>
<tr>
<td></td>
<td>Standing</td>
<td>97.9 ± 0.2</td>
<td>98.6 ± 0.2*</td>
</tr>
<tr>
<td>SvO₂, %</td>
<td>Supine</td>
<td>80.0 ± 1.2</td>
<td>79.2 ± 1.5</td>
</tr>
<tr>
<td></td>
<td>Standing</td>
<td>64.4 ± 1.3</td>
<td>69.3 ± 2.1*</td>
</tr>
<tr>
<td>PaCO₂, Torr</td>
<td>Supine</td>
<td>39.8 ± 0.9</td>
<td>34.5 ± 1.1*</td>
</tr>
<tr>
<td></td>
<td>Standing</td>
<td>37.5 ± 0.9</td>
<td>31.5 ± 0.8*</td>
</tr>
</tbody>
</table>

Values are means ± SE. SaO₂, arterial oxygen saturation; SvO₂, venous oxygen saturation; PaCO₂, arterial carbon dioxide tension. Samples were taken 1 min before the maneuver, during the last seconds of the strain, and after 10 s of the recovery. *Significantly different from baseline (P < 0.05). †Significantly different from Valsalva maneuver (P < 0.05). ‡Significantly different from the supine position (P < 0.05).

Standing. In the standing position, TI increased from 40 ± 3 to 50 ± 3 ms. CVP decreased by (4 ± 2 mmHg), whereas MAP at heart level increased by 9 ± 2 mmHg, corresponding to a reduction at the level of the MCA by ~10 mmHg (Fig. 1). SvO₂ was lower (Table 2), and CO decreased by 16 ± 4% (Fig. 1), reflecting a lowered SV (~36 ± 3%) not fully compensated for by an increase in HR (by 21 ± 3 beats/min). Compared with supine rest, MCA Vmean was reduced during standing (by 15 ± 5%). PaCO₂ was only slightly reduced during standing (Table 2).

Upright Valsalva maneuver. During straining, CVP increased immediately to 41 ± 3 mmHg and was not different from the CVP developed during the Valsalva maneuver in the supine position. Compared with the supine Valsalva maneuver, MAP changed more in phase IIa (~13 ± 4 mmHg below baseline), phase IIb (7 ± 5 mmHg above baseline), and phase III (~13 ± 4 mmHg below baseline), whereas increments were similar in phases I and IV. CO also decreased further during the standing Valsalva maneuver (to 37 ± 3% of the supine baseline value; Fig 1) and reached the lowest value during phase IIa, reflecting a plateau in SV. Compared with the supine position, MCA Vmean increased more in phase I (24 ± 4%) and dropped markedly in phase IIa (~47% from supine baseline). In contrast to the supine position, MCA Vmean dropped in phase III (12 ± 4%), and the overshoot after the release of the strain (64 ± 11%) was higher. Peak changes in MCA Vmean were not significantly different between the male and female subjects (Mann-Whitney test). None of the subjects fainted or became dizzy.

MCA Vmean and perfusion pressure. The changes in MAP-CVP are shown in Fig. 1. For the supine Valsalva maneuver, the coefficient of determination (R²) was larger for MCA Vmean vs. MAP-CVP than for MCA Vmean vs. MAP (Table 3). During standing, in the individual subjects but not for the pooled data, R² tended to be higher for MCA Vmean vs. MAP.

DISCUSSION

This study evaluated the influence of an upright Valsalva maneuver on the MCA Vmean. In the standing position, MCA Vmean was reduced ~15%, and this reduction was exaggerated by a Valsalva maneuver to ~50% of the value obtained during supine rest, whereas during a supine Valsalva maneuver the reduction in MCA Vmean was by only ~35%. These results indicate that, particularly in the upright posture, expiratory straining may critically compromise cerebral perfusion.

During standing, a drop in MCA Vmean by ~15% (2) may be in consequence of a decline in blood pressure at the level of the brain by ~9 mmHg, but also the reduction in CO (~16%) and PaCO₂ (~2 Torr) is likely to contribute. During orthostasis, the "CO₂ reactivity" of brain circulation is not known, but it is similar for the supine and seated individual (2.9 vs. 2.6%/mmHg; Ref. 18), indicating that, during standing, PaCO₂ accounts for ~5% of the reduction in MCA Vmean. Even at brain level, MAP was within the range normally associated with cerebral autoregulation, yet the lowered MAP may contribute to reduce cerebral perfusion (10).

The importance of CO for the MCA Vmean is demonstrated by an attenuated increase in MCA Vmean during dynamic exercise after β-adrenergic blockade (a 12 vs. 22% increase) as the increase in CO is restricted ~15% (11).

During the Valsalva maneuver, orthostasis affected MAP by causing a more pronounced drop below baseline in phase IIa and a blunted recovery in phase IIb. In the upright position, the smaller intrathoracic blood volume causes CO to become dependent on venous return and more so during the strain (27, 28). During the standing Valsalva maneuver, CO was as low as ~40% of the value obtained during supine rest, reflecting a reduction in SV by ~75%. Compared with the respective baseline values, an overshoot in SV and CO was pronounced only after the standing maneuver. Also, the increase in SvO₂ during the upright Valsalva maneuver supports that CO did not adequately perfuse all parts of the body.

Table 3. Individual coefficients of determination (R²) for relation of MCA Vmean vs. MAP and MAP-CVP, respectively, during a standing and supine Valsalva maneuver

<table>
<thead>
<tr>
<th>Subject</th>
<th>Vmean vs. MAP</th>
<th>Vmean vs. MAP-CVP</th>
<th>Vmean vs. MAP</th>
<th>Vmean vs. MAP-CVP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.68</td>
<td>0.73</td>
<td>0.82</td>
<td>0.69</td>
</tr>
<tr>
<td>2</td>
<td>0.64</td>
<td>0.85</td>
<td>0.74</td>
<td>0.72</td>
</tr>
<tr>
<td>3</td>
<td>0.68</td>
<td>0.76</td>
<td>0.84</td>
<td>0.71</td>
</tr>
<tr>
<td>4</td>
<td>0.34</td>
<td>0.44</td>
<td>0.84</td>
<td>0.55</td>
</tr>
<tr>
<td>5</td>
<td>0.58</td>
<td>0.87</td>
<td>0.42</td>
<td>0.67</td>
</tr>
<tr>
<td>6</td>
<td>0.51</td>
<td>0.38</td>
<td>0.88</td>
<td>0.88</td>
</tr>
<tr>
<td>7</td>
<td>0.33</td>
<td>0.40</td>
<td>0.97</td>
<td>0.72</td>
</tr>
<tr>
<td>8</td>
<td>0.86</td>
<td>0.79</td>
<td>0.55</td>
<td>0.48</td>
</tr>
<tr>
<td>9</td>
<td>0.39</td>
<td>0.91</td>
<td>0.22</td>
<td>0.29</td>
</tr>
<tr>
<td>10</td>
<td>0.70</td>
<td>0.94</td>
<td>0.85</td>
<td>0.70</td>
</tr>
<tr>
<td>Average</td>
<td>0.57</td>
<td>0.71*</td>
<td>0.71</td>
<td>0.64</td>
</tr>
<tr>
<td>Pooled data</td>
<td>0.48</td>
<td>0.57</td>
<td>0.60</td>
<td>0.64</td>
</tr>
</tbody>
</table>

Regression on data sampled at 2 Hz during 15 s of Valsalva straining. MAP-CVP, difference between MAP and CVP. *Significantly different compared with Vmean vs. MAP, P < 0.05.
During the supine Valsalva maneuver, MCA $V_{\text{mean}}$ resembled the established pattern, i.e., it decreased with the onset of straining, recovered toward the end of the maneuver, and then demonstrated an overshoot \((29)\). The distinct patterns of the arterial pressure, with its marked initial increase in phase I and sharp drop in phase III, were not reflected in MCA $V_{\text{mean}}$, suggesting that CVP dominates the cerebral outflow pressure and, in turn, the perfusion pressure. In the supine position, we found a close relationship between MCA $V_{\text{mean}}$ and MAP-CVP (Fig. 1, Table 3). During standing, we found a close relationship between MCA $V_{\text{mean}}$ and MAP-CVP (Fig. 1, Table 3). However, during standing, MCA $V_{\text{mean}}$ increased in phase I and dropped in phase III, more so than expected from the change in MAP-CVP. Furthermore, during standing, this initial increase in MCA $V_{\text{mean}}$ was larger than in the supine position, although the accompanying increase in MAP and CVP was similar. In an animal preparation, Kongstad and Grände \((13)\) demonstrated that an increase in venous pressure has no influence on the tissue pressure for as long as the venous pressure is below the tissue pressure. Only when the venous pressure equals the tissue pressure does collapse of the outflow vein disappear and the two pressures increase in parallel. In the supine position, intracranial tissue pressure approximates CVP, and the Valsalva elevation in CVP would induce a parallel increase in the cerebral outflow pressure. In the standing position, the smaller influence of a 40-mmHg elevation in CVP for cerebral outflow pressure could reflect collapsed outflow veins \((1)\). Such small deviation in the counterpressure is likely to become less important with further elevation of CVP.

$P_{\text{aCO}_2}$ decreased especially during the standing Valsalva maneuver. In contrast to breath holding without straining, a Valsalva maneuver is associated with a pronounced reduction in CO and, in turn, a reduced washout of $CO_2$ from the tissue \((19)\). The time constant for the MCA $V_{\text{mean}}$ response to a step decrease in end-tidal $CO_2$ is \(\approx 6\) s, whereas the response to a step increase in $CO_2$ takes \(\approx 14\) s \((24)\). Thus during the Valsalva maneuver, $P_{aCO_2}$ may account for 10–15% of the reduction in MCA $V_{\text{mean}}$, whereas it is unlikely to account for the overshoot after the maneuver.

The Valsalva maneuver also serves as a test for cerebral autoregulation, as demonstrated in patients with unilateral carotid stenosis and impaired cerebral autoregulation \((29)\). In comparison with the healthy side, the rate of MCA $V_{\text{mean}}$ recovery in phase II vs. that of MAP is reduced, and the phase IV overshoot of MCA $V_{\text{mean}}$ is attenuated. During both the supine and the standing Valsalva maneuver, MCA $V_{\text{mean}}$ reached peak values earlier than MAP, and, in phase II, recovery of MCA $V_{\text{mean}}$ appeared to be similar. These findings could be taken to indicate maintained cerebral autoregulation during dynamic changes in MAP. On the other hand, MCA $V_{\text{mean}}$ was markedly reduced during standing, suggesting that, in orthostasis, concomitant changes in SV, CO, and also in $P_{aCO_2}$ render the Valsalva maneuver inappropriate for quantification of cerebral autoregulation.

Transcranial Doppler monitors blood velocity rather than volume flow, and changes in the diameter of the insonated vessel could modulate velocity independent of flow \((7, 30)\). During craniootomy, Gillier et al. \((7)\) found the diameter of the large cerebral vessels unchanged with large changes in arterial pressure. Furthermore, as determined with magnetic resonance imaging in healthy individuals during hypocapnia, the MCA diameter remains stable, suggesting that the MCA is not involved in the regulation of cerebral vascular resistance \((30)\).

Sympathetic activation is of potential importance for velocity in cerebral arteries because a reduction in diameter of the insonated artery would elevate velocity at an unchanged, or even reduced, volume flow. Evidence for MCA vasoconstriction was demonstrated during direct stimulation of the sympathetic trunk \((31)\) and during maximal exercise, eliciting a 16-fold increase in plasma catecholamines \((22)\) but not with the moderate increase in sympathetic nerve activity during, e.g., postexercise muscle ischemia \((23)\). The Valsalva maneuver elicits large bursts of muscle sympathetic nerve activity during phase II of straining when the MCA $V_{\text{mean}}$ is lowest, and sympathetic nerve activity drops to below baseline after the strain when MCA $V_{\text{mean}}$ is maximal \((26)\), arguing against sympathetically mediated modulation of the MCA diameter.

Syncope is reported for weight-lifting exercise \((5)\) when intrathoracic pressure increases up to 160 mmHg. The concomitant elevation in CVP protects the brain by counteracting the large increase in MAP, and the weight-lifters' blackout is ascribed to a critical reduction in cerebral perfusion due to preexercise hyperventilation \((5)\). The occasional syncope that is observed during playing of wind instruments when mouth pressure may rise to \(>150\) mmHg \((6)\) is linked to cardiac arrhythmia, as frequently present during playing of the French horn \((3)\). We suggest that, associated with intense expiratory strain, occasional fainting is related to the rise in CVP and, in turn, to a critical reduction in cerebral perfusion.

In conclusion, in healthy individuals, orthostasis induces a reduction in MCA $V_{\text{mean}}$ that is exaggerated by the performance of a Valsalva maneuver to a level that may induce syncope. In contrast to the supine position during an upright Valsalva maneuver, cerebral perfusion pressure is dominated by the reduction in arterial inflow pressure and the contribution of CVP as outflow pressure is reduced.

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