Hyperventilation in Postural Hypotension

By John F. Burnum, M.D., John B. Hickam, M.D., and Eugene A. Stead, Jr., M.D.

In normal subjects hyperventilation causes a drop in arterial blood pressure, apparently because of the direct effect of hypocapnia on the peripheral vessels. In persons with disease of the autonomic nervous system, the drop in blood pressure is much greater because it is not effectively opposed by reflex vasoconstriction. Consequently, hyperventilation caused by anxiety may greatly intensify postural hypotension in such patients and contribute significantly to their disability. A pertinent case is reported.

Patients with postural hypotension from disease of the autonomic nervous system present an easy problem in diagnosis, but a difficult problem in therapy. The purpose of this paper is to point out the unusual response to overbreathing seen in these subjects and to call attention to the fact that prevention of the loss of carbon dioxide may be occasionally of therapeutic importance.

Hyperventilation is a frequent cause of disturbances in consciousness in anxious patients when they are lying down. When these patients stand, increased reflex action from standing, the usual postural fall in cerebral arterial pressure, and anxiety, caused by queer feelings from carbon dioxide loss, combine to produce marked overactivity of the autonomic nervous system and the common faint (reflex vasodyspnea syncope). Unless one faints, large and maintained falls in arterial pressure are not usually seen with emotional overbreathing.

In our experience the disturbances in blood pressure and consciousness produced by standing in patients with disease of the autonomic nervous system are not usually accompanied by hyperventilation. Our attention was called to the adverse effect of carbon dioxide loss on maintaining blood pressure by the following patient:

This 39 year old married white woman (A. M.) was seen at Duke Hospital in August 1952 complaining of attacks of unconsciousness on attempting to stand. She was a known hypertensive for 17 years with minimal signs of blood vessel disease. Thirteen months before entering here she began to lose weight, have a “faraway” feeling and attacks of dyspnea with numbness of the hands and face. In retrospect these seem to have been typical hyperventilation attacks. Six months later, a bilateral lumbodorsal sympathectomy was done at another hospital. Since then she had been incapacitated by inability to stand. On examination the blood pressure was found to be quite variable. On standing it began to fall immediately, and in a short time the radial pulse became imperceptible. As soon as the patient stood, she began to overbreath obviously, and this became more and more marked until she lost consciousness. It was easy to demonstrate at the bedside that rebreathing into a paper bag caused a considerable lessening of the fall in blood pressure and improved the ability to stand.

The relationships between position, ventilation volume, arterial carbon dioxide pressure and arterial pH are shown in table 1.

Within 82 seconds after tilting, ventilation volume had increased to 31 liters per minute, arterial carbon dioxide tension had fallen to 24 mm. Hg and blood pressure by the auscultating method was unobtainable. Use of a rebreathing mask which prevented carbon dioxide loss allowed a much longer period in the head-up position. In the supine position, breathing of 5 per cent carbon dioxide or breath-holding caused hypotension.

Two additional patients with the syndrome of orthostatic hypotension, fixed pulse, impotency, and impaired sweating, as described in the classic paper of Bradbury and Eggleston, were studied. The etiology was unknown in one and the other had diabetic neuropathy.
Spontaneous overbreathing was not a problem in either of these patients, but in each patient voluntary forced breathing caused a dramatic fall in arterial pressure (fig. 1). Measurements of the cardiac output by the dye technic showed that the fall in arterial pressure was caused by vasodilation and not by a fall in cardiac output. Forced breathing of 5 per cent carbon dioxide caused no fall.

There was no doubt that in these subjects the postural hypotension resulted from organic disturbances in the sympathetic nervous system. In addition to the findings on physical examination, they all showed the characteristic response of sympathetic paralysis when the Valsalva maneuver was performed. On release of the high intrathoracic pressure, the blood pressure remained at hypotensive levels and did not show the increase in pressure above the resting levels characteristic of a normal autonomic nervous system in the absence of severe heart disease (fig. 2).

**Table 1.** Variations in Arterial Carbon Dioxide Tension and Blood Pressure

<table>
<thead>
<tr>
<th>Ventilation L/min</th>
<th>Blood Pressure mm Hg</th>
<th>Arterial Blood pCO₂ mm Hg</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horizontal......... 6.2</td>
<td>140/112</td>
<td>37.3</td>
<td>7.37</td>
</tr>
<tr>
<td>30° tilt 40 sec.... 12.1</td>
<td>80/?</td>
<td>31.9</td>
<td>7.41</td>
</tr>
<tr>
<td>30° tilt 82 sec..... 31.0</td>
<td>Unobtainable</td>
<td>24.1</td>
<td>7.46</td>
</tr>
<tr>
<td>Horizontal......... 150/100</td>
<td>82/76</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sitting 10 min. with rebreathing mask. 112/86</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Horizontal......... 120/104</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Horizontal after breathing 5% CO₂ for 2 min. 190/140</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Horizontal......... 162/110</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Horizontal after 45 sec. breath-holding. 210/130</td>
<td></td>
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</tbody>
</table>

**Fig. 1.** Response of arterial pressure to hyperventilation. The upper tracing is from a normal subject and the lower three are from the patients described in the text. Hyperventilation begins, in each tracing, at the arrow.
Laplace\textsuperscript{2} noted that in a patient with classic orthostatic hypotension, voluntary hyperventilation while standing precipitated collapse, and that maintenance of arterial carbon dioxide by rebreathing through a piece of tubing prevented hypotension.

Standard textbooks of physiology state that a loss of carbon dioxide from the arterial blood causes (1) vasoconstriction by direct effect on the blood vessels, and (2) vasodilation through effect on the central nervous system; the central effect is dominant and the over-all result is a fall in peripheral resistance. The observation that decreased function of the autonomic nervous system potentiated the vasodilator effect in man led us to doubt that the over-all effect of carbon dioxide deficit on the blood vessels was vasoconstriction. Detailed studies in normal subjects, in patients with most of the sympathetic ganglia removed, and in patients receiving ganglionic blocking agents, showed that the over-all effect of carbon dioxide deficit in the arterial blood was vasodilation, and that this caused a sustained fall in arterial pressure unless it was counteracted by the normal homeostatic neurogenic mechanisms for maintaining constant arterial pressure.\textsuperscript{2} Thus, in normal subjects, the fall in peripheral resistance does not cause a large and sustained fall in arterial pressure.

**Summary and Conclusions**

1. A patient with neurogenic postural hypotension is reported who had her postural adaptation made much worse by reflex over-breathing. Attention to the problem of over-breathing simplified the clinical management.

2. Observations on three patients with neurogenic postural hypotension showed that a loss of carbon dioxide by over-breathing produced a striking and sustained hypotension with the patient in the horizontal position.

3. The observation that the greater the defect in the autonomic nervous system, the greater the fall in arterial pressure from over-breathing led to more detailed study of the over-all effects of hypocapnia on the circulation. These results reported in detail elsewhere demonstrate that hypocapnia caused an over-all vasodilation by its direct effect on blood vessels and not by an indirect effect through the nervous system.

**Sumario Español**

1. Una paciente con hipotensión postural neurogénica se informa en la cual la sobreventilación por reflejo acentuó mucho más su adaptación postural. Atención al problema de sobre-ventilación simplificó el manejo clínico.

2. Observaciones en tres pacientes con hipotensión postural neurogénica mostraron que la pérdida de bióxido de carbono debido a la sobre-ventilación produjo una dramática y sostenida hipotensión con el paciente en la posición horizontal.

3. La observación de que mientras mayor sea el defecto en el sistema nervioso autónomo, mayor la caída en presión arterial debido a sobre-ventilación, condujo a un más detallado estudio de los efectos generales de la hipocapnia en la circulación. Estos resultados informados en detalle en otro lugar demuestran que la hipocapnia produce una vasodilatación generalizada por medio de su efecto directo en los vasos sanguíneos y no por un efecto indirecto en el sistema nervioso.
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