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CASE REPORT

Correction of orthostatic hypotension by respiratory effort

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Study design: Case report.
Objective: To describe a role for breathing in maintenance for blood pressure in a tetraplegic man.
Setting: Veterans Administration Hospital, USA.
Methods/Results: A 60-year-old man, tetraplegic for 14 years, was successfully treated for orthostatic hypotension (OH) by raising the head of his bed in the mornings before transfer to his wheelchair. To test the role of breathing in the compensation for OH, we monitored the nasal airflow with a thermistor clipped onto a naris and pulse pressure was measured with a transducer held against a supraorbital artery with an elastic band. On raising the head of the bed to 30° the pulse pressure fell and breathing effort increased. Within 1 min, however, pulse pressure rose to baseline levels whereas increased breathing effort continued. On transfer to his wheelchair OH was avoided and medication was unnecessary.
Conclusion: For the tetraplegic subject the partial raising of the head of the bed for a period of time to be determined individually will recruit increased breathing effort and venous return to the chest, preventing OH on transfer to an upright position in a wheelchair.
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Keywords: blood pressure; breathing; spinal cord injury; tetraplegia

Introduction

The neurosympathetic deficit and the hormonal compensation in tetraplegic subjects with orthostatic hypotension (OH) have been described.[1] An additional deficit—that of the pulmonary pump, which describes the enhanced return of venous blood to the chest and heart by the negative pressure generated by breathing—is also important.[2] A tetraplegic man is presented to show the role of the impaired pulmonary pump in the defense against OH.

Case report

A 60-year-old man, spinal cord injured for 14 years, was hospitalized for respite care. Neurologically, his level was C4, motor and sensory complete with a zone of partial preservation to T6. He revealed the typical shallow chest and protuberant abdomen of tetraplegia. Because of a history of OH the head of his bed was routinely raised in the morning for a few minutes before transfer into his wheelchair and the resumption of activities of daily living. The head-raising maneuver being successful, no sympathomimetic medication was prescribed. The time for accommodation to orthostatis being a matter of minutes and seemingly too short to be attributable to hormonal responses, this subject was monitored for the respiratory response to hypotension.

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Methods

With the subject lying flat in bed, an airflow-sensitive thermistor (Astro-Med, Warwick, RI, USA) was clipped to a naris and a piezoelectric pulse pressure transducer (UFI, Morro Bay, CA, USA) was held against a supraorbital artery with an elastic band. Beat-to-beat respiratory and arterial pulses were collected, amplified and printed, using a polygraph with a strip chart recorder (Model 79E; Grass Instruments Co, Quincy, MA, USA). The subject was instructed to breathe only through his nose during the test, keeping his mouth closed. The head of the bed was raised to 30° over a period of 10 s.

Results

With head and trunk elevation pulse pressure and pulse rate diminished. The depth of breathing did not change; but the breathing rate increased. Within a minute at the same position, however, pulse pressure and pulse rate abruptly recovered to baseline levels. The enhanced breathing rate remained unchanged during this recovery (Figure 1; Table 1).

Discussion

The support of blood pressure against gravity depends on (1) reflex vasoconstriction with orthostasis, a rapid response impaired by the transection of sympathetic tracts of the spinal cord;[1] (2) blood volume, a baseline support dependent on hormonal control of salt and water retention but eroded by the vascular leakage of albumen and renal salt wasting of

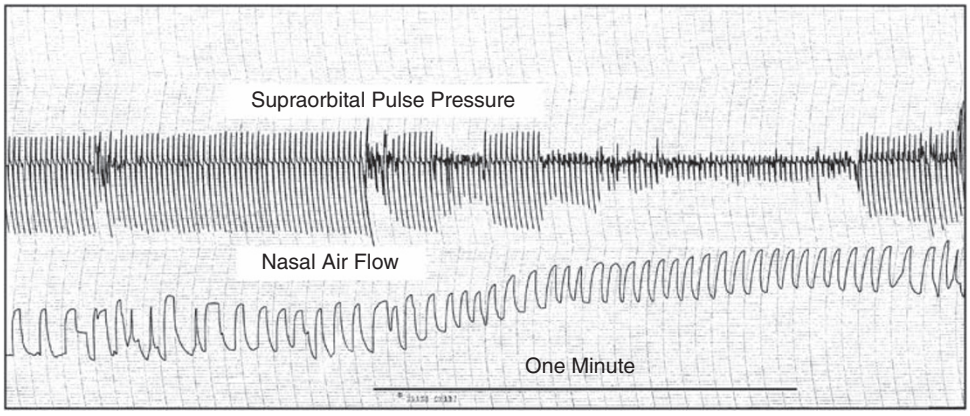

Figure 1 Breathing rate increases and arterial pulse pressure decreases with elevation of the subject's head and chest in bed to 30°. The timing of the head-up position is indicated by the start of the horizontal line. Notice that the increased breathing rate is sustained after the arterial pulse pressure recovers.

Table 1 Effect of head and trunk elevation on parameters of circulation and breathing

Duration of 30° elevation (min)	0	<1	1
Arterial pulse pressure (units)	30	7	27
Arterial pulse rate (beats per min)	90	81	90
Depth of breathing (units)	14	13	14
Respiratory rate (breaths per min)	24	30	30

All rates and units were read by eye. Units are arbitrary, not calibrated to mm Hg for arterial pulse pressure or to the volume of air for depth of breathing. Changes with time for each parameter are relative.

tetraplegia[1,3] and (3) venous return, a constant support dependent on the pulmonary pump, which is impaired by tetraplegia.[2] Despite an inability to breath deeply, the patient presented was able to compensate for OH and to maintain this correction by an increased rate of breathing, enhancing the action of the pulmonary pump.

Although the neural control of breathing is intact in the tetraplegic subject (The baroreceptors, through the spared glossopharyngeal and vagus nerves (the vagal stimulus of the hypotension was manifest in the slowing of the heart rate in this subject and the reversal of that slowing with correction of the pulse pressure), transmit the stimulus of hypotension to the breathing center of the medulla.) the depth of breathing is much impaired. The abdominal musculature of the tetraplegic subject is flaccid; the fulcrum for the diaphragm is removed, particularly in the upright position; and the chest cannot be lifted and expanded, causing a reduced vital capacity, an inability to breath deeply[2] and diminished negative intrathoracic pressure, compromising the pulmonary pump. Compounding this disability, deconditioning of the diaphragm likely occurs for lack of this abdominal resistance.[4] The tetraplegic patient breaths rapidly to compensate for the inability to breath deeply. This phenomenon has been shown by monitoring tetraplegic and neurologically intact subjects in the sitting position, the former breathing rapidly with shallow chest movements, the latter breathing slowly with deeper chest movements.[5]

The case discussed is relevant clinically in that it shows the effectiveness of the head-raising in bed before transferring to a wheelchair and the fully upright position. The duration of effective head-raising may vary from a few minutes to approximately 30 min. Sometimes, if the head elevation is too great, OH in bed will develop and the head may have to be lowered. This practice recruits breathing effort and stimulates, in addition, the renin–angiotensin–aldosterone system. Supplementation of salt intake to provide adequate substrate for this hormonal recruitment will correct volume depletion and further ameliorate OH.[3] These simple nonpharmacological measures can improve quality of life for the OH patients.

Conflict of interest

The author declares no conflict of interest.

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