Stress-induced hypotension in pure autonomic failure

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Abstract

We report a 47-year-old woman with pure autonomic failure who complained of dizziness during emotional stress. Emotional stimuli have not previously been reported to cause hypotension in patients with autonomic failure. In the patient, ambulatory blood pressure recording revealed severe hypotension (50/30 mm Hg) after a stressful event. During a tilt-table test hyperventilation was shown to cause a significant fall of blood pressure. This suggests that emotional stress can induce hypotension, probably through hyperventilation, in subjects with autonomic failure.
An important feature of autonomic failure is orthostatic hypotension, giving rise to sensations of light-headedness or frank syncope following standing up or during prolonged standing. Post-exercise hypotension is another important feature, giving rise to similar complaints immediately following the cessation of physical exercise. Emotional stress is not known to provoke hypotensive complaints in autonomic failure. Here, we report stress-induced hypotension in a patient with pure autonomic failure (PAF), probably mediated through hyperventilation.

CASE REPORT

A 47–year–old woman complained of light-headedness. Prolonged standing and cessation of exercise, typically after climbing stairs, provoked her complaints. Besides this, light-headedness was triggered by emotional upset, such as confrontations with an adolescent child. She had never lost consciousness during such episodes. The patient has a medical history of Gilles de la Tourette's syndrome. Physical examination revealed severe orthostatic hypotension (160/75 mm Hg supine, 70/45 mm Hg after 3 minutes standing). Neurological examination was without abnormalities. Basic laboratory tests (blood count, electrolytes, glucose, renal and liver function tests) were normal. Magnetic resonance imaging of the brain was without abnormalities. Autonomic function testing revealed normal heart rate variability during rest and deep breathing but a reduced heart rate response to standing (ratio 0.98) and to the Valsalva manoeuvre (ratio 1.28). Further analysis demonstrated decreased plasma cathecholamine concentrations (noradrenaline: 0.41 nmol/l supine and 0.47 nmol/l upright; adrenaline: not detectable supine and 0.07 nmol/l upright; dopamine: 0.10 nmol/l supine vs. 0.12 nmol/l upright). There was a complete absence of $^{123}$I–metaiodobenzylguanidine (MIBG) uptake at cardiac scintigraphy. A diagnosis of PAF was made.

The patient was educated regarding physical counter-manoeuvres such as leg crossing and squatting, to combat hypotension. Despite the beneficial effects of these manoeuvres, our patient required pharmacological treatment, as her complaints progressed over the next 2 years. Midodrine 10 mg tid gave a satisfactory reduction of her hypotensive episodes without serious supine hypertension. However, despite this improvement emotional stimuli continued to trigger her complaints. Ambulatory blood pressure (BP) monitoring confirmed the hypotensive nature of these complaints with one recording of 50/30 mmHg when her symptoms were provoked by a row. In view of the consistent provocation of complaints by emotional stress, a suspicion of hyperventilation-related hypotension was raised. During a tilt-table test the patient was asked to hyperventilate while ECG, BP (Finometer, finger photoplethysmography) and end-tidal CO$_2$ tension (PET,CO$_2$) were continuously monitored (Figure 1). Hyperventilation in a supine position induced hypotension. Tilting induced enough hypotension to cause the typical complaints, but supine hyperventilation did not. However, when blood pressure was first reduced by a moderate degree of tilting, hyperventilation caused an additional fall of BP and light-headedness. The patient did not experience paraesthesiae during hyperventilation.

Discussion

Emotional stress was found to provoke a hypotensive episode in our patient. This is remarkable, as psychological stressors are known to increase sympathetic outflow and
thus raise BP in healthy subjects.[3] To our best knowledge, emotional stimuli have not previously been reported to cause hypotension in patients with autonomic failure. Both tilting and hyperventilation caused a fall of BP in our patient and evoked complaints similar to those in daily life. However, the question remains whether her stress-related complaints can be explained by hyperventilation induced hypotension. Therefore we should consider the following questions: can stress induce hyperventilation, and can hyperventilation induce hypotension?

It is known that emotional stress evokes part of the fight-flight-fright response, associated with a mild degree of hyperventilation. Emotional or mental stressors frequently cause mild hypocapnia.[4] In normal subjects these small fluctuations of PET,CO₂ do not give rise to paraesthesiae or other complaints related to hypocapnia. Hyperventilation causes an increase of heart rate in normal subjects.[4,5] The effects of hyperventilation on BP are not unequivocal, but it can be stated that hyperventilation apparently does not cause frank hypotension in healthy subjects.[5] However, in our patient even mild decreases of PET,CO₂ resulted in a significant fall of BP. Earlier studies have shown that hyperventilation can in fact cause a fall of BP and only a moderate increase of heart rate in subjects with autonomic failure.[6,7] The amount of increase of HR during hyperventilation in autonomic failure depends upon the amount of vagal denervation. In our patient, the heart rate increased markedly during hyperventilation indicating that vagal innervation was relatively spared compared to the marked sympathetic vasoconstrictor failure. In autonomic failure, the fall of BP during hyperventilation is associated with a lowering of peripheral vascular resistance, that outweighs an increase in cardiac output.[6] It is likely that hyperventilation in autonomic failure lowers BP through hypocapnia, as effects could not be reproduced through other features associated with hyperventilation, such as an increase of pH or PaO₂, or by an increase of respiratory movements without hypocapnia.[7] A direct peripheral vasodilator effect of hypocapnia seems probable, as hyperventilation causes hypotension in conditions with insufficient sympathetic innervation, as in autonomic failure or in healthy subjects during ganglionic blockade.[7,8]

Our results suggest that emotional stress can act as a factor inducing hypotension in subjects with autonomic failure, and we recommend an open mind towards this possibility. This may not only help understand events, but may also help the patient: emotional stressors are hard to avoid, and our patient still experiences lightheadedness following such stimuli, even though she is fully aware of the mechanism. However, knowing that the complaints are due to defective BP regulation rather than to an emotional hypersensitivity offers some reassurance.
Legend Figure 1
During the tilt-table test the patient subsequently underwent the following conditions: supine rest (a), 60º tilt (b), supine rest (c), hyperventilation (d), supine rest (e), 30º tilt (f), 45º tilt (g), 45º tilt + hyperventilation (h) and supine rest (i). Both tilting and hyperventilation caused a significant fall of BP. The vasodepressive effect of HV added to the hypotensive effect of tilting (condition h). Abbreviations: BP= finger blood pressure, HR=heart rate, $P_{ET, CO_2}$=end-tidal CO$_2$ tension, HV= hyperventilation.

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References


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