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Periodic breathing induced by hyperventilation

We report a case of inappropriate hyperventilation that caused recurrent episodes of unconsciousness and apnoea. The presentation was sudden and resembled that of a brain stem embolus.

Case report

A 39 year old man with aortic incompetence secondary to endocarditis due to *Lactobacillus plantarum* was admitted to the regional infectious diseases unit. After four weeks of antibiotic treatment, during which he appeared to be doing well, he was transferred urgently to the regional cardiac centre.

On the day of transfer he had awakened at 3 00 am with severe anterior chest pain. Immediate examination showed a pulse rate of 84 beats/min, blood pressure 100/60 mm Hg, and systolic and early diastolic murmurs at the left sternal edge that were unchanged from previously. Chest examination was normal as was an electrocardiograph. Fifteen minutes later he collapsed and became unconscious and apnoeic for about 30 seconds. No change was noted in pulse rate or blood pressure. This occurred again several times, and after several hours he was transferred, when the attacks were occurring every two or three minutes. Examination showed no obvious focal neurological signs, and during the intervals between the episodes of unconsciousness he was lucid but tachypnoeic. His vital signs were unchanged. He was admitted to the intensive care unit.

Investigations showed normal blood glucose, urea, and electrolyte concentrations and normal full blood count. Arterial blood samples were drawn for blood gas analysis at the moment of apnoea and again at the moment when breathing restarted (figure). Profound hypocapnia was accompanied by considerable alkalosis, and inappropriate hyperventilation was diagnosed. Explanation and reassurance accompanied directions to him to breathe less. Diazepam 10 mg was given intravenously over 10 minutes. There were no further episodes of apnoea or unconsciousness, and blood gas analysis after one hour showed normal values. He admitted to a feeling of air hunger, especially in the mornings.

Results of neurological examination and computed tomography were normal, and a psychiatric assessment reported him as mentally normal but with an anxious personality. During his hospital stay he had several more episodes of hyperventilation, which responded to reassurance. He subsequently underwent cardiac catheterisation, which disclosed severe aortic incompetence. Coronary angiograms were normal. He underwent aortic valve replacement without incident.

Comment

Because of his bacterial endocarditis the sudden onset of fluctuating levels of consciousness and of periodic breathing in this man was taken to indicate a brain stem embolus, despite the absence of other focal neurological signs. Hyperventilation may be caused by brain

Changes in oxygen pressure (pO_2), carbon dioxide pressure (pCO_2), and pH with time.

Conversion: SI to traditional units—Oxygen and carbon dioxide pressures: 1 kPa $\approx$ 7.5 mm Hg.

stem disease, but in this patient respiratory alkalosis did not recur after supportive treatment, which argues against an organic hyper-ventilatory state.

Syncope may occur with hyperventilation because the cerebral circulation responds to a decrease in carbon dioxide pressure by vasoconstriction and consequently reduced cerebral perfusion. In this case the severe alkalosis and low carbon dioxide pressure appeared to "switch off" respiration in an attempt to correct the abnormalities. During the apnoeic episode the oxygen pressure fell sharply, and probably this was the stimulus to restart breathing.

Hypocapnic alkalosis has direct circulatory effects. Blood pressure falls owing to a reduction in peripheral resistance resulting from local histamine release.¹ Direct effects on the heart include increased myocardial contractility,² increased stroke volume and cardiac output,³ but also increased coronary vascular resistance with diminished coronary flow.⁴ Angina threshold (defined as the product of the heart rate and blood pressure at which angina occurs) decreases with hyperventilation.⁴ This has been postulated as contributing to stress induced angina.

Our patient initially developed chest pain, which suggested myocardial ischaemia. Possibly he became caught in a vicious cycle of hyperventilation leading to angina, leading to anxiety, leading to more angina and hyperventilation. Hyperventilation may cause unilateral somatic symptoms⁵ and should be considered as a possible cause of collapse and loss of consciousness in patients with cardiovascular disease, many of whom are extremely anxious.

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