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Respiratory compromise: a rare complication of transcutaneous electrical nerve stimulation for angina pectoris

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Abstract

Electrical stimulation of any muscle group may produce tetany. If the intercostal muscles are involved this may lead to respiratory embarrassment. A case is presented in which transcutaneous electrical nerve stimulation (TENS) treatment for angina pectoris resulted in respiratory arrest.

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Key terms: transcutaneous electrical nerve stimulation; respiratory arrest.

Electrical neurostimulation as an analgesic technique has been used successfully for many years particularly in obstetrics. More recently it has been used in patients with severe unstable angina refractory to pharmacological therapy.¹⁻³ One potential hazard is highlighted by this case.

Case report

A 70 year old man with a long history of ischaemic heart disease suffered an attack of angina pectoris during ambulance transport to a routine cardiology outpatient appointment. One year earlier a transcutaneous nerve stimulator system (TENS) had been fitted, with stimulating electrodes placed anteroposteriorly across the chest wall to control the pain of his unstable angina. The episode of chest pain in the ambulance had been very severe and he had increased the TENS current to maximum (50 mA). The ambulance crew brought him directly to the accident and emergency department.

On examination in the resuscitation room the patient was conscious but unable to speak. The respiratory rate was 25/min with very shallow breaths. There was central cyanosis. On auscultation breath sounds were normal

although air entry was minimal. The pulse was 65/min of good volume and regular. Blood pressure was 140/85 mm Hg.

High flow oxygen was given from a reservoir system with no clinical benefit. It was not possible to record an electrocardiograph as discharges from the TENS machine produced considerable electrical interference. The patient deteriorated and chest wall movement virtually ceased. The respiratory pattern was that of tetanic paralysis with incoordinated sustained muscular contraction.

The TENS electrodes were removed. Immediately the patient exhaled and was able to respire with much greater movement of the chest wall. Within a few minutes the respiratory pattern had returned to normal. On questioning he was pain-free. The dyspnoea subsequently resolved completely. An ECG showed no changes from previous electrocardiographs and after attending the outpatient clinic the patient was discharged home.

Discussion

The lack of familiarity with neurostimulators for the treatment of angina pectoris meant that the medical staff treating this patient were at a disadvantage in determining the cause of the acute deterioration. There are no previous reports of similar problems with TENS systems, although subjective symptoms of difficulty in breathing are not uncommon (Mannheimer C, personal communication). This case shows the potential for more serious complications with neurostimulators.

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