

## The Duff, No Duff Casualty

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**SUMMARY:** A patient who presented with “hyperventilation syndrome” was initially mis-treated as severe crush injury, illustrating the need for thorough assessment of all casualties whilst on exercise prior to arranging casualty treatment and evacuation.

### Case Report

During a recent exercise in Germany, a radio message was received by a field ambulance collecting section that there was a “no duff” casualty who had been crushed. This message was confirmed by the field ambulance signaller because the unit from which the message had been received was involved in casualty play at that time. A section was urgently sent and a helicopter evacuation was made ready.

The 31-year-old male casualty was lying under a wooden pallet. On examination he was anxious and distracted, mildly dehydrated with cool peripheries and was too tachypnoeic to give any verbal response. Heart rate was 126 beats per minute, arterial pressure 110/70mmHg. Intravenous access was achieved in the right antecubital fossa, an intravenous infusion was commenced and the casualty was evacuated to the nearby dressing station.

On arrival, his ventilatory frequency exceeded 50 b.p.m., and both wrists and fingers were flexed suggesting carpopedal spasm. A provisional diagnosis of hyperventilation syndrome was made. Attempts to slow the patient’s rate and depth of respiration using a rebreathing bag and a quiet atmosphere were successful. A full secondary survey was entirely normal.

Enquiry later revealed that the patient was initially “acting” but became “carried away” by the simulation exercise. He denied any previous similar episodes or any recent drug or ethanol ingestion.

### Discussion

The term “hyperventilation syndrome” was first used in 1938 to describe patients with hypocapnia and anxiety. It remains an ill-defined term and there is still no consensus on its definition (1,2), but there is an association with depression, hysteria and other psychiatric disorders (2,3).

Hyperventilation and hypocapnia cause symptoms which may be relatively unique, such as tetany, or more generalised, such as dizziness, confusion, syncope, chest pain and palpitations (4,5). Rafferty, Saisch and Gardner (6) have shown that the onset of symptoms occur when end-tidal Pco<sub>2</sub> decreases to 2.6 kPa and is unrelated to rate of change of Pco<sub>2</sub>. Alkalosis and lowered calcium concentrations increase neuronal excitability accounting for tetany, while altered conscious level occurs because of cerebral vasoconstriction (7) induced by hypocapnia. Reduction in peripheral and coronary

circulation also contributes to symptoms (2,4,5).

Respiratory causes of hyperventilation include chronic lung diseases such as asthma, chronic obstructive pulmonary disease, spontaneous pneumothorax, acute laryngeal obstruction and fibrosing alveolitis. Non-respiratory metabolic causes include acidosis secondary to diabetes, uraemia and certain drug overdoses (e.g. Aspirin). Other non-respiratory causes include pulmonary embolus, left ventricular failure, acute and chronic pain, angio-neurotic oedema and major system failure (3).

The use of opioid analgesia to reduce the pain that the casualty was initially thought to be suffering from (a crush injury), would have reduced his level of consciousness and could have precipitated a respiratory arrest (8,9).

This report emphasises the need for full assessment of all casualties and clear and precise communication to avoid intensifying confusion.

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