

# Transient Epidermal Flushing in a Head Injured Patient: A Case Report

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## ABSTRACT

*An unusual case of transient epidermal flushing after median nerve stimulation and endotracheal suctioning in a patient with head injury is described. The mechanisms of transient erythematous skin reactions in a head injured patient are discussed. (Critical Care and Resuscitation 2001; 3: 170-172)*

**Key words:** Head injury, epidermal flush

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Clinical features of increased epidermal vascular reactivity include erythematous flushing and urticaria. Vasogenic flushing is usually controlled by autonomic nerves. However, autonomic functions are complex and there may be many neurotransmitter substances that are released into the circulation with central nervous stimulation. We report an unusual case of transient epidermal flushing in a patient with severe head injury and discuss the mechanisms that may have been involved.

## CASE REPORT

A previously healthy forty-two year old man sustained a head injury in a motor vehicle accident and had a Glasgow Coma Score of 6/15 at the scene of the accident. A computerised tomography scan of his head on arrival at Waikato Hospital showed a right parietal lobe intracerebral haemorrhage with surrounding local oedema. There was blood in the left occipital horn of the lateral ventricle and right posterior ethmoid and spheroid sinuses. There was no obvious evidence of increased intracranial pressure. A right-sided basal skull fracture in the middle cranial fossa was noted. There were no other injuries.

An intracranial pressure monitoring device (Camino fiberoptic probe, Neurocare, Pleasant Prairie, WI, USA) was inserted and the patient was managed in the intensive care unit. He was sedated with a morphine and midazolam infusion and mechanically ventilated. The

cerebral perfusion pressure was maintained at greater than 70 mmHg.

Sedation was withdrawn on day two. He remained unconscious with only a flexor response to pain. Four milligrams of vecuronium were given in order to perform sensory evoked potentials.

During median nerve stimulation, a transient epidermal flush developed on the trunk (Figure 1). His heart rate increased from 60 to 150 per minute and pulse oximetry recorded a decrease in oxygen saturation. The flush disappeared within a few minutes after the nerve stimulation was stopped (Figure 2). The same reaction was reproduced thirty minutes later when nerve stimulation was repeated without vecuronium. The flushing also appeared when the endotracheal tube was suctioned.

The patient had no previous history of skin disorders and serum antinuclear factor, smooth muscle antibodies, anti-mitochondrial antibodies, adrenal antibodies, antinuclear cytoplasmic antibodies, and C-activation tests were tested and were negative. His C-reactive protein was 96mg/L (normal limits 0.8 - 8.0 mg/L). His serum tryptase, C<sub>3</sub>, C<sub>4</sub>, total complement levels, and rheumatoid factor were within normal limits.

The patient was discharged from the intensive care unit after five days. After a week on the general surgical ward he was discharged to his home-town hospital for further rehabilitation. There were no further episodes of the epidermal flushing.

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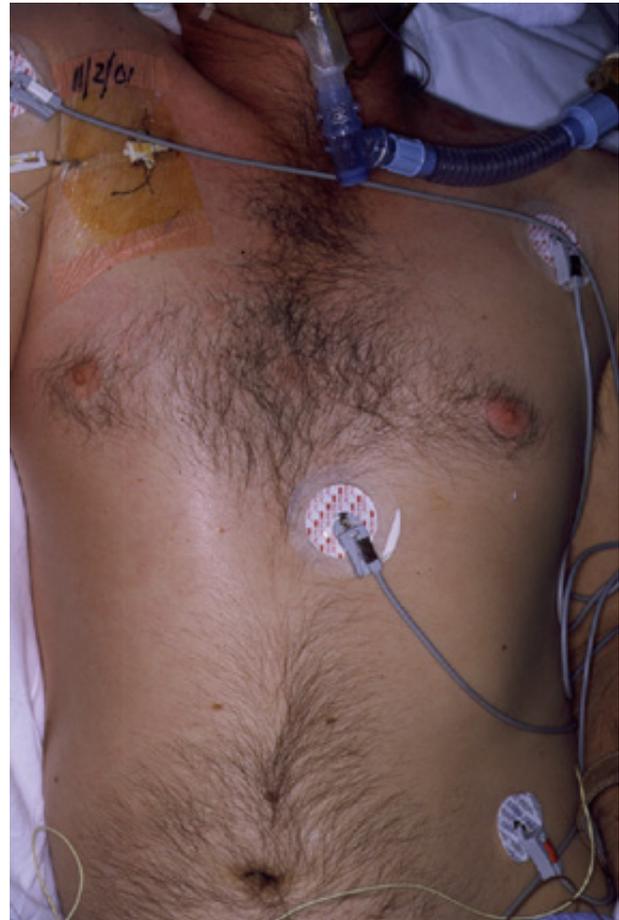
**Figure 1.** Areas of confluent and patchy erythema that developed on the trunk during median nerve stimulation.

#### DISCUSSION

Our patient demonstrated acute reversible epidermal flushing in direct response to mildly stressful stimuli (e.g. median nerve stimulation or endotracheal suction). This response appeared to be neurogenic rather than immunogenic, as the onset and offset were rapid and closely linked to the stimulus. The flushing was also distant to the epidermal stimulus and there was a lack of detectable evidence of systemic mast cell degranulation or complement activation. It would seem likely that the reaction that we observed is part of the spectrum of dysautonomic responses commonly found in severely brain injured patients.

The mechanism of the epidermal vascular reaction in this patient is unclear. While an erythematous rash is a common finding in the intensive care patient and is often due to drug allergy, these are epidermal eruptions that are usually slightly elevated and last for more than 10 - 15 minutes. Urticarias are caused by an increase in vascular permeability and may be associated with an

erythematous skin reaction. They generally present with an oedematous area that may be localised or confluent and also tend to last more than 10 - 15 minutes. Moreover, if an urticaria is induced by a physical stimulus (e.g. cold, heat, pressure, vibration, light, water, exercise and galvanic devices) it is often (but not always) within the area stimulated.<sup>1</sup>



**Figure 2.** The rash clearing from the trunk a few minutes after the median nerve stimulation was discontinued.

Urticarias are thought to be secondary to mast cell activation and inflammatory mediator release. The duration of the lesion is thought to be related to the pathogenesis.<sup>3</sup> In an acute urticaria there is vasodilatation and increased vascular permeability, but no tissue infiltration, in contrast to the perivascular accumulation of mononuclear and mast cells in chronic cases. This is due to the absence of a defined antigen, a brief encounter with the stimulus and rapid removal of vasoactive substances and chemotactic factors.<sup>3</sup> Nearly all physically-induced urticarias tend to disappear within two hours.

Neurogenic vascular reactions may be autonomic-

ally induced or induced by an inflammatory reaction. Neurogenic inflammation occurs when neuropeptides such as substance P and calcitonin gene-related peptide produce an inflammatory response following their release from sensory neurons. Chemical, thermal, and electrical stimulation have also been used to elicit neurogenic inflammation. Stimulation of nociceptive nerve endings in the skin generates an action potential, which is eventually processed to give a sensation of pain or itch. At the same time, the action potential travels retrogradely to branches of the primary afferent neuron to produce vasodilatation and protein extra-vasation in the stimulated area. Mast cells do not appear to be activated in neurogenic inflammation in normal human skin (as opposed to rodent and pig skin) and it has been suggested that the cutaneous vascular response is directly induced by neuropeptides.<sup>4</sup> This may explain the lack of raised tryptase or complement activation in our patient. However, neurogenic inflammation does not explain why the reaction was distant to the dermatome of skin stimulated.

A vascular flush has been previously reported as an unusual clinical sign of a possible rise in intracranial pressure in paediatric patients.<sup>5,6</sup> The erythematous reaction is sudden and severe and lasts for 5 - 15 minutes with the postulated mechanism being a centrally mediated vascular dilatation.<sup>5</sup> The flushing may or may not be associated with increased sweating. A similar

reaction occurred in our patient and a centrally-mediated neurogenic mechanism is also postulated.

This case illustrates the possibility that physical stimuli should be considered in the differential diagnosis of vascular reactivity in intensive care patients. Prolonged stimulation may potentially lead to generalised increase in vascular permeability and if severe it may cause hypotension.

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