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Letter to the Editor

Thermoregulation in tetraplegic patients

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The problem of temperature regulation in patients with tetraplegia has attracted the interest of doctors involved in their care over the last 100 years.[1–3]

In normal subjects, heat loss is regulated by sweating. A patient with a cervical cord transection is unable to sweat and it has been suggested that a supplementary heat loss mechanism is an amplification of the ventilatory (panting) response. Wilsmore *et al*[4] have returned to the problem using sophisticated techniques and commented that previous studies suffered from methodological limitations related to controlling the thermal load, quantification of thermal strain or experimental power.

I was joint author of the study with Sir Ludwig Guttmann and Wyndham nearly 50 years ago, which they were kind enough to quote. As they have commented on our techniques, it is appropriate to amplify and explain the problems we encountered.

Guttmann had always maintained, no doubt under the influence of Foerster, that panting was an important auxiliary method of heat regulation. We, Guttmann *et al*[3] found that the patients were unable to regulate their body temperature against heat and cold as well as normal individuals. We varied the temperature, using the heat chamber as described by Guttmann,[5] and cooling was studied by using the ambient temperature in the UK.

The cervical lesion cases were quite unable to sweat at 35–37°C and skin temperature rose. The breathing was measured on a spirometer. We found that the respiratory rate rose from 15 to 25 breaths a minute and the tidal volume fell from 500 to 350 ml. The rate of rise of rectal temperature did not appear to be reduced by panting. This could be taken to mean that panting is not an effective channel of heat loss in man. We thought that the panting was a direct effect of heat on the temperature-regulating centre.

It occurred to me that, as the ventilation increased, there was a problem. It might well be that the oxygen consumption of the respiratory muscles would increase and as the patient did not have a full complement of respiratory muscles, having to breath with the diaphragm and auxiliary muscles alone, this could be very inefficient and might in turn be increasing the generation of heat. I therefore addressed this problem by carrying out an assessment of the oxygen cost of breathing in a tetraplegic patient.[6]

I studied 11 tetraplegic patients and drove their breathing by increasing the dead space using the method of Campbell *et al.*[7] This estimated the oxygen uptake by recording the disappearance of oxygen from a closed-circuit spirometer. All the subjects behaved in the same way. It was an average of 1.6 ml of oxygen per litre of excess ventilation with a range of 0.85–2.7 ml in the 11 patients. This was higher than the findings of Cournand and Richards[8] of 1 ml and that of Campbell *et al*[7] of 0.25 ml in normal subjects, but considerably lower than findings of 23 and 7 ml, respectively, in patients with cardiopulmonary disease at the same levels of ventilation.

It would appear that the oxygen consumption per litre of excess ventilation is a little higher than that in normal subjects, and this may be because the patients are unable to use all their respiratory muscles, the intercostals and abdominals being paralysed.

The conclusion was that the increased ventilation was not an effective way of losing heat but the increased ventilation did not generate excessive heat.

I trust that this clarifies the issue of our methodology.

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