'Boosting' performance in disability sport

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There is no less contentious issue in elite sport than doping to enhance performance. Athletes are always looking to find the edge over opponents by fair means, or by foul in some cases. Potentially hazardous risks to health through doping are taken in the pursuit of sporting excellence. The increasing profile of disability sport and its potential rewards combined with the frailty of human nature has lead some athletes with disabilities to seek improved performance through the administration of banned substances. There is however, a doping method unique to disability sport, which is termed 'boosting'. Boosting is the intentional induction of autonomic dysreflexia to enhance performance and was deemed a banned method by the International Paralympic Committee (IPC) in 1994. But how and why did this practice come about?

The tetraplegic athlete has limited potential for improvements in cardiac output and maximal oxygen uptake for several reasons. Firstly the loss of sympathetic cardiac innervation results in a maximum heart rate of between 110-130 beats per minute determined by intrinsic sino-atrial activity [1]. The restricted heart rate reserve and reduced stroke volume are further compounded by a loss of catecholamine response to exercise and by the absence of the muscular venous pump in the lower limbs. These are limiting factors on performance that some tetraplegic athletes believe can be partly compensated for by the induction of the dysreflexic state.

Autonomic dysreflexia is a clinical phenomenon unique to patients with spinal cord injury above the major sympathetic outflow tract [2]. The lesion is most commonly at or above the T-6 level and therefore occurs in high paraplegic and more commonly in tetraplegic subjects. A nociceptive stimulus below the level of the spinal cord injury may initiate reflex sympathetic activity. Absence of higher control over reflex sympathetic activity due to cord injury is the main problem but additional factors such as supersensitivity of receptors have also been implicated [3]. Systemic hypertension occurs which is not controlled by feedback of the parasympathetic system but flushing above the level of the lesion, vascular headache and nasal congestion occur. Autonomic dysreflexia has been regarded as a medical emergency because of the severe rises in blood pressure that can occur [4]. Complications include seizures [5], cerebral haemorrhage [6], cardiac arrhythmia [7] and death [6]. Perhaps not surprisingly then the IPC Medical Committee deemed boosting to be a banned method in an attempt to avoid a fatality in competitive sport.

But why should athletes intentionally induce such a potentially life-threatening state? During training and competition some tetraplegic athletes had noticed that the dysreflexic state actually reduced the rating of perceived exertion for pushing and faster top speeds were achieved. Although initially the phenomenon was occurring

spontaneously, it was found that the condition could be induced by practices including clamping of the urinary catheter to produce bladder distension, excessive tightening of the leg straps, twisting and/or sitting on the scrotum. Athletes felt that in this way they could control the boosted state to command. A study of eight athletes using this technique during maximal treadmill tests and during simulated races confirmed significant performance enhancement with the most striking change being a 9.7% improvement in race performance time [8]. This would be approximately equivalent to reducing the able-bodied marathon record by twelve minutes! In the boosted state at rest there was a lower heart rate and during exercise subjects were able to achieve levels in excess of the normal maximum. Significant rises in noradrenaline levels were seen (7.1 nmol/l boosted v. 2.35 nmol/l unboosted.) but no change in adrenaline levels occurred. Athletes felt that they were only getting access to a catecholamine response and heart rate reserve that they could normally achieve in exercise if uninjured. However significant rises in blood pressure were observed during the study and the reported ability to control the response was found to be fallacious. There are no reported incidents of adverse events occurring during induced autonomic dysreflexia during exercise. Possibly the cardiovascular fitness of the athlete has a protective effect when compared to the deconditioned patient in a spinal injury unit but the numbers taking part in elite disability sport with this level of lesion are relatively small.

The IPC having deemed boosting to be a banned method of doping have a real practical problem with enforcement. Although the concern is for the safety of athletes there are comparisons with growth hormone abuse – unless you catch the athlete in the act, how do you detect it. Firstly, autonomic dysreflexia occurs spontaneously in tetraplegics and so to enforce this ban there has to be a method of not only detecting an athlete in a dysreflexic state but also proving that the state was intentionally induced. The first is not easy during a race. You cannot 'pit stop' athletes for blood pressure checks. Blood pressure measurements were made in the call-up room at the Atlanta Paralympics and the potential threat was to withdraw athletes with abnormally high readings. The 'normal value' of blood pressure in a call-up room before a Paralympic final is difficult to predict but I suspect the IPC might be open to litigation if they withdrew an athlete on these grounds. The importance of education of athletes and dialogue with athletes is surely the way forward in preventing a potential disaster and this task has yet to be addressed by the IPC. For the sports physician working with athletes with this disability it is important to be aware of this condition, whether intentionally induced or not. The immediate management is to remove the nociceptive stimulus where possible and to administer sublingual nifedipine to reduce the blood pressure.

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