

SPORTING MISCELLANY

The sub-24-minute 10 000 metres, 2040 AD

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It was a special event. The attack on the world 10 000 m record had been publicised worldwide, and the satellite sports channels had unique coverage. Steve Coe, the challenger – grandson of a famous father, whose own world record had lasted for almost as many years as Steve had been alive – had put in three periodised sessions daily for the past 24 months under his coach Harry Anderson, specifically to prepare for this attempt to be the first to run under 24 minutes for the distance.

In carefully dovetailed micro-, meso-, and macrocycles he had put in thousands of multiple daily sessions either on the indoor tracks and courses, or on the computer-programmed Powerpace treadmill in his altitude chamber, complete with forced-draft wind simulation and projection screen with variable scenic or stadia video cassettes. Adinike, his sponsoring company, had developed a skintight hi-flo Lycra racing suit that both decreased wind resistance and increased heat dissipation. It moulded his smoothly shaven head, and it maximised advertising space. Equally, he had undergone many hundreds of precisely targeted isokinetic and biokinetic conditioning sessions, with considerable emphasis on plyometric lower-body work, to greatly enhance elastic energy storage in Achilles and other tendons together with the ligaments of the transverse and horizontal arches of the foot. In this he hoped to increase his Achilles elastic storage from some 37% to nearer 43%, and that of his foot arches from 17% to 20%.

Routine biomechanical and physiological assessments were performed as was felt appropriate, although their usefulness latterly seemed to lie as much in aiding Steve's morale as in any new data which they brought him and Harry, unless there were any training breaks for illness or injury, which fortunately were short and seldom. Over the 24 months, Harry and Steve embarked on a carefully selected racing programme at varying distances.

Adinike had also developed a racing shoe weighing under 25 g, with a sole incorporating the latest Pogothane insert, which not only increased foot-strike rebound by 22% without interfering with the natural connective tissue elasticity of his legs and feet, but also provided adequate plantar sensory feedback to minimise impact injury through the otherwise possibly false subjective estimation of plantar comfort. The shoes also featured the latest micro-spikes. The Ariadne track, a highly advanced version of the old Harvard tuned track, was programmed for Coe's body mass, stride length,

heelstrike force, and running cadence. The moveable roof for the Whizlet stadium would be electronically adjusted for perfect environmental control, and the master control for race pacing would be provided by the programmable light-guide "hare" built into the track kerb, and which his eight pace makers had been given a great deal of money and very strict orders to follow. The pace makers were not expected to stay with the race, but to trot round until the particular sub-4-minute four-lap section was called for.

Steve's weekly blood samples had been submitted to full automated analysis, and his diet supplemented where necessary, together with cervical epidural branched chain amino acid and endorphin injections (to minimise fatigue), routine intravenous glutamine (to increase lymphocyte nutrition), and slow-creatine in enteric tablet form (to enhance muscle energetics). Regular bone scans provided early warning of impending stress fractures, and NMR scans for early inflammatory foci around tendon, ligament or origins or insertions of muscle, were similarly routinely performed.

Frequent and thorough clinical, physiotherapy, osteopathic, and podiatric investigations were performed prophylactically, and twice daily a sports masseur eased out the tired muscles. A nutritional and a micronutrient biochemist, with the assistance of two dieticians, formulated most of Steve's food.

His fluid replacement throughout had been geared to actual daily weight loss correlated with quantified NMR site-specific adipose tissue estimations, and his electrolyte replacement was paralleled to computed losses in sweat. Months of electronic stimulation, at 40 Hz square wave pulses on the respective motor points of carefully selected muscles, had optimised his functional fibre-mix for the coming race; this had been carefully preceded by specific intramuscular injections of insulin-like growth factor (IGF). Appropriate phases of contrasting 10 Hz stimulation had been applied over 96-hour sessions to maximise selected-muscle capillary beds, to increase local blood flow, in order to maximise the removal of both protons and heat.

Then graded infusions of lactate over many months had helped raise his anaerobic threshold much higher than could otherwise have been achieved. Such infusions had been controlled where necessary by similar infusions of sodium hydrogencarbonate, to adjust muscle pH, as monitored by NMR spectroscopy. Gene-specific triggers for enzyme induction of

hepatic and renal Cori cycle enzymes had been injected in target-specific virus vectors, to maximise that particular aspect of lactate removal.

Steve had undergone many phased courses of post-hypnotic suggestion to minimise the subjective elements of fatigue, had practised regular mental rehearsal of incremental lap times and final time with the aid of a virtual reality simulator, and had been subjected to bouts of relaxations and autogenic training together with thought-stopping and biofeedback to counter stress and anxiety.

In the final few days, major attention was paid to the most critical parameter of all, his muscle glycogen, as this was held to be the absolute key to success in the attempt. The optimum fibre mix achieved by nerve doping, with the whole being boosted by site-specific slow release IGF had optimised the fibre type profile together with the enzyme profile per fibre type. This had set the scene for gene programmed increase in sarcolemmal insulin receptors, together with the programmed synthesis of sarcoplasmic glycogen synthetase and glucose transporter proteins, which resulted in a glycogen boost able to be taken to $420 \mu\text{mol}\cdot\text{g}^{-1}$ muscle – over 400% of the normal resting level.

Over $400 \mu\text{mol}\cdot\text{g}^{-1}$ muscle was the lowest value calculated¹ as the minimum for a 24 minute race, but the problem with so much glycolysis would be the resulting overwhelming concentration of pyruvic acid and its conversion to lactic acid. Specific mitochondrial inducing training by the Hoppeler method had been carried out to maximise the lactate shuttle. Intra- and extracellular buffers had been optimised, mainly with the induction of the Newsholme cycle of fast hydrogen-carbonate, by the process discovered by him at the turn of the millenium. So the success of the whole enterprise depended upon whether, at the sarcoplasmic level, Steve could produce enough glycolytic energy, yet still remove the resulting protons fast enough, to sustain the pace of a 57.6 400 m plus 12 consecutive 800 m, each at 1 m 55.2.

Dietary, electrolyte, fluid and psychological preparations were completed over the final 72 hours, with a final intravenous infusion of the fatigue inhibitor antioxidant N-acetylcysteine (to which he was known not to react adversely) immediately before stepping out onto the track. Steve Coe had trained, peaked and tapered to the form of his life, and he was ready

to run 10 000 metres in under 24 minutes before a mega-satellite 27-channel pay-sport audience of just over half the world. This would net him one billion pounds, and a lifetime of ease.

In all this, he had not detectably infringed article 29 of the current Olympic Charter which states, simply, “Doping is forbidden”.

Appendix

The calculations were based on those of Newsholme *et al*, in the reference cited, as follows:

10 000 m in 24 min requires the following concentrations of muscle glycogen:

100 m in 10 s requires $3 \mu\text{mol ATP}\cdot\text{g}^{-1}$ muscle $\cdot\text{s}^{-1}$

10 000 in 24 min (1440 s) = 14.40 s per 100 m
ATP needed is $3 \times 10/14.40 = 2.08 \mu\text{mol ATP}\cdot\text{g}^{-1}$ muscle $\cdot\text{s}^{-1}$

Assume, for this 10 000 m, that 65% ATP is provided aerobically and 35% anaerobically.

(1) *How much glycogen is consumed aerobically for this 10 000 m?*

$2.08 \times 65\% = 1.352 \mu\text{mol ATP}\cdot\text{g}^{-1}$ muscle $\cdot\text{s}^{-1}$
 $= 1.352/37 = 0.0365 \mu\text{mol glycogen}\cdot\text{g}^{-1}$ muscle $\cdot\text{s}^{-1}$ [assuming 37 moles of ATP are produced per mole of glucose (from glycogen) used]

= 52.6 $\mu\text{mol glycogen}\cdot\text{g}^{-1}$ muscle for the event

(2) *How much glycogen is consumed anaerobically for this 10 000 m?*

$2.08 \times 35\% = 0.728 \mu\text{mol ATP}\cdot\text{g}^{-1}$ muscle $\cdot\text{s}^{-1}$
 $= 0.728/3 = 0.243 \mu\text{mol glycogen}\cdot\text{g}^{-1}$ muscle $\cdot\text{s}^{-1}$
= 349.44 $\mu\text{mol glycogen}\cdot\text{g}^{-1}$ muscle for the whole event

The total is $402.04 \mu\text{mol glycogen}\cdot\text{g}^{-1}$ muscle, which is nearly four times the normal concentration of glycogen stored in muscle. (And which compares with a 10 000 m in 27 min 20 s, during which 97% ATP might be considered to be provided aerobically, and 3% anaerobically, requiring, per gram of muscle, concentrations of $79 \mu\text{mol}$ and $30 \mu\text{mol}$ glycogen respectively; a total of $109 \mu\text{mol glycogen}\cdot\text{g}^{-1}$ muscle, which is the total amount of glycogen stored in 1 g of muscle.¹)

¹ Newsholme EA, Bloomstrand E, Eckblom B. Physical and mental fatigue: metabolic mechanisms and importance of plasma amino acids. *Br Med Bull* 1992;48:477–95.