

Case study

An elite runner with cerebral palsy: cost of running determines athletic performance

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Background: Running performance is widely understood in terms of the Joyner model (VO_2max , $\% \text{VO}_2\text{max}$ at ventilatory threshold (VT), running economy (often measured as cost of running (CR) as VO_2 in $\text{ml}\cdot\text{kg}^{-1}\cdot\text{km}^{-1}$).

Objective: To test the Joyner model by evaluating a runner in whom one element of the Joyner model is systematically abnormal.

Methods: The case of a two-time Paralympian with cerebral palsy (CP), 2nd place in the Sydney 2000 Paralympic 1500m (T37) is reported. Incremental and steady state treadmill runs as well as simulated competitions were completed. Incremental and steady state (50% PPO) cycling with two legs (2L), the non-affected leg (NL), and the affected leg (AL) were also completed.

Results: His silver medal (2000 Sydney OG) performance for 1500m was 269 s (4:29) (77.2% of velocity in contemporary able-bodied world record (WR). At the time of study, his VO_2max was $64.2 \text{ ml}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$. His cost of running (CR) (1% grade) was higher, at 257 vs 228, 211 and $188 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{km}^{-1}$ (for ACSM norms, elite Europeans, elite East Africans). During cycling, his VO_2max with 2L, NL and AL was 3.74, 3.78 and $3.71 \text{ l}\cdot\text{min}^{-1}$, and his gross efficiency (GE) was 18.4, 12.2 and 9.3%, respectively.

Conclusions: In a former elite runner with CP, there is little evidence of a central oxygen transport limitation. The higher CR (plausibly reflected by the reduced GE of his AL) appears to account for much of the difference in performance compared to able-bodied runners. The results provide both insight into the physiological limitations of runners with CP and support for the Joyner model of competitive running performance.

Keywords: biomechanics, athletic training, exercise performance, exercise physiology

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Running performance is well described by the Joyner model which explains performance as a function of VO_2max , the $\% \text{VO}_2\text{max}$ at lactate/ventilatory threshold and running economy, often expressed at the cost of running (CR) or VO_2 ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{km}^{-1}$).^[1]

There has been general support for this model, over a generation of sports scientists. A novel way of further testing this model is to evaluate runners where one element of the model is systematically different. This paper reports a case study of an elite runner with cerebral palsy (CP). CP is “a disorder of the development of movement and posture, causing activity limitations attributed to non-progressive disturbances in the developing fetal or infant brain”.^[2] CP is classified from the nature of the movement disorder

and the anatomical distribution of motor abnormalities or severity. Our subject had spastic hemiplegia on the right side of his body, with unilateral involvement or spasms of the muscles including both arm and leg. He began running in his youth, and competed in different distances (800 m, 1500 m, and 5000 m) at two Paralympic Games (Athens 1996, Sydney 2000). His best performance was the silver medal in the 1500 m (T37 class) at the Sydney 2000 Paralympic games. His personal bests (compared to contemporary WR's in able-bodied runners) were: (800 m=132 s (2:13) vs 102.3 s (1:43)=77.5%WR; 1500 m=269 s 4:29) vs 207.7 s (3:27.7)=77.2%WR; 5000 m=1060 s (17:42) vs 769.6 s (12:49.6)=72.5%WR).

The purpose of this study is to provide insight into two aspects of performance in athletes with CP. First, what are the characteristics of running performance of a runner with CP compared to able-bodied runners? Second, how do persons with CP adapt to pathological conditions to achieve optimal function. These authors believe that this is the first study to report on CR and physical adaptation in an elite runner with CP.

Methods

At the time of the study (2011), the subject was 31 years old. He had participated in the 1996 and 2000 Paralympics, and had trained competitively until 2004. Although not competitive at the time of the physiological studies, he was still running three times per week for 30-40 min and cycling for transportation, mostly at intensities below the ventilatory threshold (VT). The study was approved by the university human subjects committee, according to principles of the Declaration of Helsinki, and the subject provided written informed consent.

The tests consisted of three parts: treadmill running, cycle ergometry and race simulations. All tests were performed at the same time of day (18h00-20h00) during summer. He undertook no intense training for 48 hrs and no training (except cycling for transportation purposes) for 24 hrs before each test. Laboratory tests and races were separated by at least 48 hours.

The subject performed incremental treadmill running until exhaustion ($2.68 \text{ m}\cdot\text{s}^{-1}$ ($9.64 \text{ km}\cdot\text{h}^{-1}$) $+0.22 \text{ m}\cdot\text{s}^{-1}$ ($0.8 \text{ km}\cdot\text{h}^{-1}$) each minute). He also performed steady state treadmill running (5 minute stages with 1 min walking recovery between stages). Performed on a different day, the steady state runs began at $2.23 \text{ m}\cdot\text{s}^{-1}$ ($8.0 \text{ km}\cdot\text{h}^{-1}$) and increased by $0.22 \text{ m}\cdot\text{s}^{-1}$ ($0.8 \text{ km}\cdot\text{h}^{-1}$) until the velocity at blood lactate = $4 \text{ mmol}\cdot\text{l}^{-1}$ was exceeded. All treadmill runs were performed with the treadmill belt at 1% elevation to mimic wind resistance. Respiratory gas exchange was measured using open-circuit spirometry, with a mixing chamber based metabolic system (AEI Inc, Bastrop, TX), and standard calibration procedures. VO_2max was accepted as the highest continuous 30 s VO_2 observed during the test. The cost of running (CR) was calculated from the interpolated VO_2 -velocity relationship at a $\text{VO}_2=90\%$ of that at VT. This was done to assure that the CR represented steady state conditions, and to allow comparison to published norms.^[3] Blood lactate was measured in capillary blood obtained from a fingertip using dry chemistry (Lactate Plus).

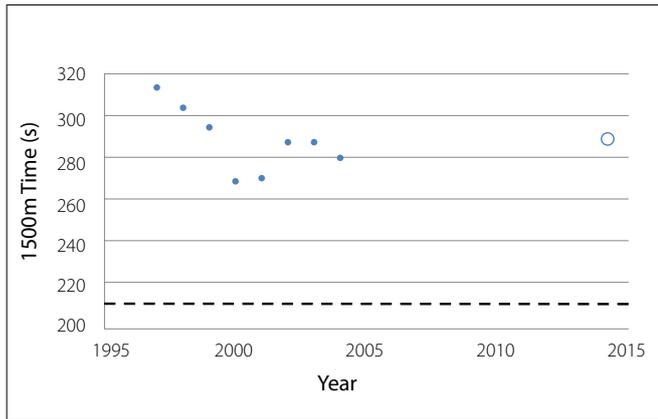


Fig. 1A. Seasonal best 1500m performances (closed circles), with the contemporary WR for reference (dashed line). Note that despite officially retiring from competition, the time for the 2011 time trials (open circle) was still in the general range of his silver medal winning Paralympic performance.

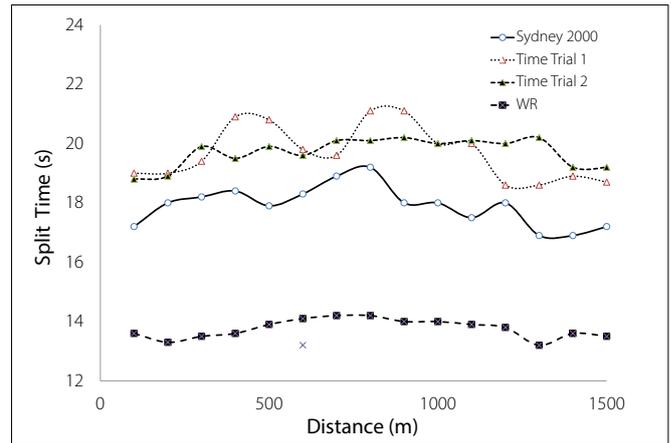


Fig. 1B. Split 100m times during 1500m time trials (2011) (296 and 297 s), during the Paralympic silver medal race (2000) (269 s), and for the contemporary WR (208 s) for able-bodied runners. Of note is that all races had essentially the same pacing pattern (fast-slow-fast).

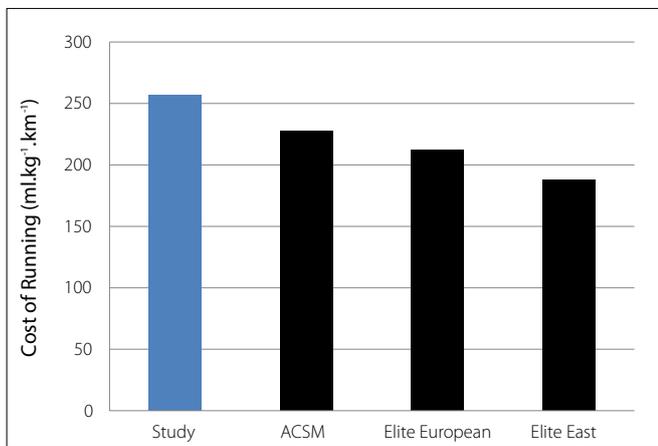


Fig. 1C. Cost of running, expressed in ml.kg⁻¹.km⁻¹, for the runner with CP in this study (light), in reference to the ACSM norms, and norms for elite European runners and elite East African runners (dark)^[3]

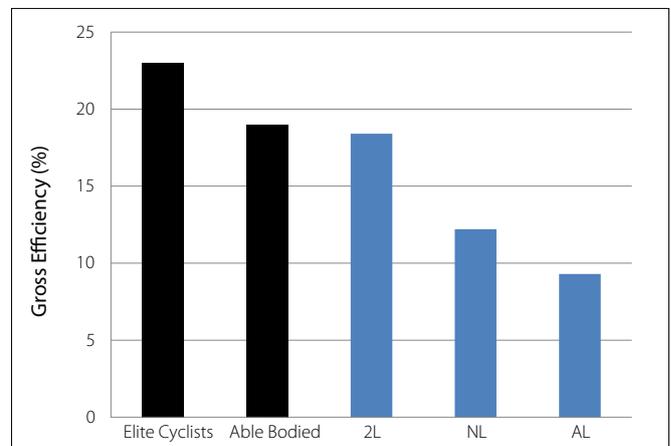


Fig. 1D. Gross efficiency during cycling at 50% of PPO in elite cyclists (dark), in able-bodied non-athletes (dark), with 2L by the subject with CP, and with the NL and AL by the subject with CP (light)

Two 1500m time trials were performed on an outdoor 400m track, with an experienced pacemaker, to allow comparison of race results between 2000 and 2011. The pacemaker attempted to run the pace that the subject believed that he could sustain. During the run, based on verbal communication between the subject and pacemaker (e.g. too slow, just right, too fast), the pace was regulated every 50-100m. During the last 400m, the subject attempted to pass the pacemaker, as he would in competition. Times were recorded every 100m.

Because running is an inherently a bilateral activity, and (as in this subject) CP is often a unilateral pathology the authors felt that it was important to assess differences between his affected leg (AL) and non-affected leg (NL). Accordingly, he performed incremental exercise using both legs 25 W + 25 W per 60 s) and, separately, with both the non-affected and affected legs (25 W + 15 W per 60 s, with 90 s on and 30 s off). Additionally, he performed a 10 min ride at steady state (50% peak power output) on the same cycle ergometer (Lode Excalibur, Groningen, NL), both with 2L and separately with the AL and NL.

Results

At the time of the study, the subject's height and weight were 1.75 m and 63.3 kg respectively, which were unchanged from his competitive years. During his peak competitive years, he trained ~4 times weekly,

totalling ~40 km per week, including 2-3 interval training sessions. He stated that if he trained >4 days per week, he tended to become injured.

During incremental treadmill running, his VO₂max = 64.2 ml.min⁻¹.kg⁻¹, his VO₂@VT=52.8 ml.min⁻¹.kg⁻¹, his VO₂@RCT (respiratory compensation threshold)=60.8 ml.min⁻¹.kg⁻¹. His vMax= 4.58 m.s⁻¹, vVT=3.58 m.s⁻¹, vRCT=4.25 m.s⁻¹, v4mmol=3.39 m.s⁻¹ and vVO₂max=4.33 m.s⁻¹. His gross VO₂max was 4,074 ml.min⁻¹, with VT and RCT at 80% and 95% of VO₂max. These percentages are high compared to the normal range of endurance athletes, possibly because the vVO₂max was limited by the coordination and work capacity of the AL. This finding is duplicated in the cycling results. He remarked that during his competitive career, when he was fatigued, the toe of his AL tended to drop, occasionally causing him to fall.

Comparing his silver medal race in 2000 with his 2011 1500m time trials shows that although he was slower (269 s (4:29) vs 297 s (4:57) and 296 s (4:56)) (Figure 1A), the pattern of pacing was virtually the same (Figure 1B). His final time in the two 2011 1500m time trials represented 91.0% and 91.4% of the velocity achieved during his silver medal race. Although the time trials were slower than his Paralympic race, they were in the general range of races during his late competitive career, which the authors believe provides evidence that the data collected during 2011 is representative of his physiological responses during his peak competitive period in 2000.

During cycling his peak power output (PPO) was 300 W, 105 W and 190 W with 2L, AL and NL, respectively. VO_2max was 3.74 (2L), 3.78 (AL) and 3.71 (NL) $\text{L}\cdot\text{min}^{-1}$. The VO_2max with 2L represented 92% of his running VO_2max . It was remarkable that during 1L cycling with his AL, the pedalling action was very much “whole body”, with a great degree of accessory muscle involvement, whereas with the NL the cycling action looked normal.

The higher CR of our subject is presented in Figure 1C. The CR, with 1% grade, was 257 $\text{ml}\cdot\text{kg}^{-1}\cdot\text{km}^{-1}$ vs 228 $\text{ml}\cdot\text{kg}^{-1}\cdot\text{km}^{-1}$, 211 $\text{ml}\cdot\text{kg}^{-1}\cdot\text{km}^{-1}$ and 188 $\text{ml}\cdot\text{kg}^{-1}\cdot\text{km}^{-1}$ vs ACSM norms, elite Europeans and elite East Africans, respectively.^[3] The GE during cycling is presented in Figure 1D, which depicts the range of elite cyclists, able-bodied individuals, and our subject's values for 2L, NL and AL. GE during 2L steady state cycling at 50% PPO was 18.4% (150 W, $\text{VO}_2=2.42 \text{ L}\cdot\text{min}^{-1}$, RER=0.84) (at the low end of normal), during 1L cycling with NL was 12.2% (95 W, $\text{VO}_2=2.27 \text{ L}\cdot\text{min}^{-1}$, RER=0.90) and during 1L cycling with his AL his GE was 9.3% (53 W, $\text{VO}_2=1.68 \text{ L}\cdot\text{min}^{-1}$, RER=0.95).

Discussion

The main finding of this case study was that an elite runner with CP was characterised by increased CR, which seems to define the mechanism of his reduced (compared to able-bodied runners) performance ability more than other variables in the Joyner model of running. This finding was supported by a decrease in GE during cycling in his AL. He was able to achieve VO_2max even using the AL, apparently by extensive use of the accessory musculature to overcome the strength and coordinative deficit in the AL.

Extrapolating the speed- VO_2 relationship observed in the laboratory his best 2011 performance would require a VO_2 of 74 $\text{ml}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$, representing 115% VO_2max . Assuming that the CR can improve by no more than 8%^[4] and that VO_2max is unlikely to increase more than 2% with the addition of high-intensity interval training^[5], the authors estimated that in Sydney at the 2000 Paralympic Games his CR was no lower than 238 $\text{ml}\cdot\text{kg}^{-1}\cdot\text{km}^{-1}$ and his VO_2max was no higher than 66 $\text{ml}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$. On this basis, his Paralympic performance of 269 s would have required a VO_2 of 79.9 $\text{ml}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$, or 121% of his estimated VO_2max on race day. The percent of VO_2max for both 2000 and 2011 performances are within reason for the relative intensity of elite able-bodied runners. While even his estimated Sydney 2000 race day VO_2max is not equivalent to the $\sim 75 \text{ ml}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$ typically seen in elite middle-distance runners ($\sim 85\%$ as good), his best possible CR is high compared to elite able-

bodied runners ($\sim 75\%$ as good). Thus both the 2011 time trials and the projected values for his 2000 Paralympic performance support the Joyner model of running performance, and suggest that the primary difference between this elite runner with CP and elite able-bodied runners is dominantly related to an increase in the CR.^[1]

His ability to adapt and have a very good performance despite the limitations imposed by CP appears attributable to his ability to recruit accessory muscles to overcome the limitations imposed by CP. This compensatory strategy extracts a cost in terms of reduced GE (e.g. increased CR), which is only possible if VO_2max is reasonably high. A recent study from the authors' laboratory^[6] in children with CP demonstrated that PPO (-25%) and Wingate PO (-40%) were more depressed than VO_2max (-15%) compared to age matched controls. These data support the concept that limitations in mechanical power production (e.g. increased CR, decreased GE) contribute to the performance deficit in CP. They further suggest that therapeutic strategies with CP should include efforts to increase VO_2max so that these patients can have the physiologic capacity to accommodate their neuromuscular deficit. Although not directly measured in this study, it is likely that the spasticity in the AL influenced the stretch-shortening cycle of the AL, decreasing the potential contribution of elastic recoil to propulsion. Further, chronic spasticity of the AL has the potential for reducing muscle blood flow, in much the same way that the extended duty cycle seen in speed skaters contributes to reduced muscle blood flow and VO_2max .^[7]

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