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Physiology of Wheelchair Racing in Athletes with Spinal Cord Injury

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Abstract

Wheelchair racing is one of the most popular sporting activities of individuals with spinal cord injury. Athletes with this impairment have unique changes in metabolic, cardiorespiratory, neuromuscular and thermoregulatory systems, which reduce their overall physiological capacity compared with able-bodied individuals or individuals with other types of impairments. This review on spinal cord injury: (i) presents the International Stoke Mandeville Games Federation classification of wheelchair athletes; (ii) describes methods commonly used to characterise anaerobic and aerobic fitness; (iii) presents the findings of physiological studies that have evaluated wheelchair racing performance; (iv) identifies the risks associated with temperature regulation when competing in wheelchair races; and (v) discusses special conditions that can influence wheelchair racing performance.

Currently there is limited research that has examined the relationship between sprint or distance wheelchair racing performance and the anaerobic and aerobic components of physical fitness. Although the descriptive evidence indicates that the profiles of these athletes reflect their training and participation in these specific events, the association between their physiological profiles and real or simulated racing performance is unclear. The generally accepted concept that high values of aerobic and anaerobic power are strongly correlated with endurance and sprint racing performance, respectively, are not necessarily true in this population. Athletes with spinal cord injury have an impaired thermoregulatory capacity, because the compromised autonomic and somatic nervous system functions disrupt control of skin blood flow and sweating below the level of the lesion. As a result, they may be more susceptible to hyperthermia during distance wheelchair racing performance. Wheelchair athletes should follow recommendations advocated for able-bodied individuals to minimise their risks of heat stress during competition. Many athletes with quadriplegia voluntarily induce autonomic dysreflexia (commonly known as boosting) during distance racing events to improve performance. Experimental evidence indicates that boosting can improve performance time by 10% in elite wheelchair marathon racers during simulated racing, as a result of increased oxygen utilisation in the boosted state. However, since boosting can be dangerous to health, the International Paralympic Committee has banned athletes from voluntarily inducing it during competition. The use of antigravity suits to increase lower-body positive pressure can increase the peak oxygen uptake, cardiac output and stroke volume. However, the use of abdominal binders does not influence these physiological responses. An effect of either of these techniques on wheelchair racing performance has not been demonstrated.

1. International Stoke Mandeville Games Federation Classification for Spinal Lesions

Participation in regular physical activity and sport is considered to be an essential part of the rehabilitation process in individuals with chronic disabilities. During the last 5 decades, since the inception of the First International Wheelchair Games for individuals with spinal cord injury in Aylesbury, England, there has been a tremendous growth in competitive sport for those with disabilities.[1,2] Athletes with spinal cord injury, often referred to as wheelchair athletes, are allowed to participate in international competitive events if they have at least 10% loss of function in the lower extremities. An in-depth analysis of the statistics of the 2000 Sydney Paralympic Games indicated that the largest number of competitors participated in athletics, with wheelchair racing being the most widely contested sport.[3]

In international competitions, athletes with different disabilities are classified according to their individual functional capabilities so that they can compete on an equitable basis. This implies that athletes with similar capabilities and not necessarily similar medical disabilities (eg. cerebral palsy) compete in the same class.[1,4-6] The International Stoke Mandeville Games Federation (ISMGF) system is extensively used to classify athletes with spinal cord injuries who are competing in wheelchair sport. This system utilises a combined medical and functional approach to classify athletes into one of eight groups according to specific criteria identified in table I.[1] The classification procedure usually involves an examination of the medical records as well as direct observation of the athlete to evaluate the functional capacity and identify the anatomic level of lesion. Trained classifiers conduct objective tests to evaluate trunk and limb movements as well as the strength of specific muscle groups to classify the athletes. The strength of the

Table I. International Stoke Mandeville Games Federation Classification for spinal injuries[7]

Class	Cord level	Functional characteristics
IA	C4-C6	Triceps 0-3 on MRC Scale. Severe weakness of trunk and lower extremities, interfering with sitting balance and ability to walk
IB	C4-C7	Triceps 4-5. Wrist flexion and extension may be present. Generalised weakness of trunk and lower extremities, interfering significantly with sitting balance and ability to walk
IC	C4-C8	Triceps 4-5. Wrist flexion and extension may be present. Finger flexion and extension permits grasping and release. No useful hand intrinsic muscles. Generalised weakness of trunk and lower extremities interfering significantly with sitting balance and ability to walk
II	T1-T5	No useful abdominal muscles (0-2). No functional lower intercostal muscles. No useful sitting balance
III	T6-T10	Good upper abdominal muscles. No useful lower abdominal or lower trunk extensor muscles. Poor sitting balance
IV	T11-L3	Good abdominal and spinal extensor muscles. Some hip flexors and adductors. Weak or non-existent quadriceps strength, limited gluteal control (0-2). Points 1-20, traumatic, 1-15 polio
V	L4-S2	Good or fair quadriceps control. Points 21-40, traumatic, 16-35 polio
VI	L4-S2	Points 41-60, traumatic, 36-50 polio[a] (Class VI is a subdivision of V, applied only in swimming competitions)

a Each of the following muscle groups is awarded up to 5 points (5 = normal strength) per side: hip flexors, adductors, abductors and extensors, knee flexors and extensors, ankle plantar and dorsiflexors. Potential score, 40 points per side.

MRC = Medical Research Council.

individual muscle groups are graded on a 6-point scale: a score of zero indicates total lack of a voluntary muscle contraction while a score of 5 implies normal strength against resistance throughout the range of motion of the joint. The degree of sensory loss, presence of muscle spasticity, and use of orthotics, all of which play an important role in performance, are also given due consideration in the classification process. From a practical point of view, classes IA and IB are differentiated on the basis of the strength of the triceps, whereas class IC is distinguished on the basis of hand muscle function. Differences in the strength of the abdominal and spinal muscles are used to categorise athletes in classes II and III as they play an important role in maintaining posture and balance in the wheelchair. Muscles of the hip, knee and ankle are used in classifying athletes in classes IV, V and VI (swimming only).

There are several issues associated with the integrated ISMGF classification system for athletes with disabilities.[8,9] The first issue pertains to misclassification of athletes. This system penalises the athlete with a greater impairment but who has enhanced functional capacity and performance because of extensive physical training and practice of specific skills. It is possible that such an athlete would be placed in a higher (more competitive) class and be forced to compete with those who have a lesser impairment. The second issue, termed classification instability, deals with the developing high potential athlete who is recently injured, insufficiently trained or coached and lacks sports skills. Such an athlete may be placed in a specific functional class based on current capabilities, but will likely improve over time necessitating a shift in class. The third issue is an ethical one, where an athlete might not perform at the highest potential during the classification process, thereby obtaining a lower classification and an unfair advantage during competition. The final issue is the subjective nature of the classification process that places special demands on the classifier. Experienced classifiers are expected to perform this subjective task as objectively as possible, often with considerable controversy.

2. Overview of Research Pertaining to Exercise in Spinal Cord Injury

Competitive wheelchair racing performance is dependent upon three characteristics, namely, the athlete, the propulsion vehicle (wheelchair) and the interaction between the athlete and the wheelchair.

Athletes with spinal cord injury have unique changes in cardiorespiratory, metabolic, neuromuscular and thermoregulatory systems that reduce their overall physiological capacity, in contrast to athletes with other disabilities such as amputees who do not face such inherent limitations. To date, the majority of the exercise physiology research that has been conducted on spinal cord injury has focused on their rehabilitation from an overall health perspective.[10,11] Although there is considerable descriptive information on the physiological capacity of athletes with spinal cord injury, this area of research has not kept up with the unprecedented growth experienced in disability sport. Most of the laboratory studies have focused on evaluating the metabolic and cardiorespiratory fitness parameters in this population. During the last decade, efforts have been directed towards quantifying the anaerobic performance of these athletes. Currently, there is a dearth of field studies that have examined the physiological aspects of these athletes during competition.

Earlier reviews have provided a general overview on the medical/physiological,[7,12-16] biomechanical[17,18] and injury [19] profiles of wheelchair athletes. This document will synthesise research pertaining to the *acute physiological responses* of athletes with spinal cord injury and relate this information to wheelchair racing performance. More specifically, this article will: (i) discuss the metabolic factors pertinent to wheelchair racing in individuals with spinal cord injury; (ii) describe tests and methods commonly used to characterise an individual's anaerobic and aerobic fitness; (iii) present the findings of physiological studies that have evaluated wheelchair racing performance; (iv) identify the risks associated with temperature regulation when competing in wheelchair races; and (v) discuss special conditions which can influence wheelchair racing performance. Emphasis is placed on research that has been published since the mid 1980s, which was when the last comprehensive reviews pertaining to the physiological aspects of wheelchair racing were published.[7,13] The list of references was compiled using online searches using the following databases: Medline, Embase and Sport Discus. The following key words were used in the searches: *spinal cord injury (paraplegia, quadriplegia),* in combination with *wheelchair racing, anaerobic power and capacity, aerobic power and capacity, cardiorespiratory responses, metabolic responses, temperature regulation, autonomic dysreflexia*. In addition, the reference lists of published articles were examined to obtain pertinent references on these topics.

3. Energy Systems Specific to Wheelchair Racing Performance

Wheelchair races for those with disabilities range from short sprints such as the 100m dash to the traditional marathon. Theoretically, the energy requirements for such races will depend upon the intensity and duration of the performance.[20] A 100m wheelchair race, which usually lasts between 14 to 20 seconds, is dependent primarily upon the immediate source of energy, namely adenosine triphosphate (ATP) and creatine phosphate (CP). This intramuscular energy source is available in limited quantities and is metabolised very rapidly during the early stages of high intensity exercise. It is considered to be anaerobic in nature because it does not require the utilisation of oxygen. Wheelchair athletes with high levels of intramuscular ATP/CP would have a greater potential for succeeding in such an event. Wheelchair races of longer durations, such as the 200 to 400m events, usually last between 30 seconds to 1 minute 30 seconds. These events are dependent upon both the immediate and short-term energy sources via anaerobic glycolysis, with a greater contribution from the latter the longer the duration of the race. In such events, carbohydrates are metabolised very rapidly in the muscle, resulting in the accumulation of large quantities of lactate and hydrogen ions, which result in muscle fatigue. Wheelchair races exceeding 5000m are dependent primarily upon the long-term sources of energy production. Energy is metabolised from the breakdown of carbohydrates and fats under aerobic conditions without the production of lactate. The availability of oxygen in the mitochondria is crucial to obtain a continuous supply of ATP for

muscular work. The volume of blood transported by the central circulation (cardiac output) and the amount perfusing the exercising muscle play an important role in this process. Usually, as the duration of the race increases, the proportion of energy derived from free fatty acids also increases, with a proportional decrease in the contribution from carbohydrates.

Research pertaining to substrate metabolism during competitive wheelchair sprint or distance racing performance in athletes with spinal cord injury is not available. However, there is limited research that has examined substrate utilisation during prolonged exercise in athletes with spinal cord injury under simulated conditions in the laboratory. The current evidence[21] obtained on a small number of athletes indicates that during 40 to 60 minutes of submaximal wheelchair exercise at approximately 60 to 70% peak aerobic power, the primary substrates utilised are intramuscular glycogen (based on depletion levels in the deltoid), glucose, free fatty acids and glycerol. The contribution of free fatty acids and glycerol increases with the duration of exercise and is accompanied by a moderate decrease in blood glucose utilisation.[21-23] However, some evidence suggests that blood glucose is not utilised during prolonged exercise at these intensities.[24] The increased contribution of fat relative to carbohydrate is indirectly evident as a decrease in the respiratory exchange ratio (ratio between carbon dioxide production and oxygen consumption) which is monitored non-invasively during exercise.[21,23,24] The elevated free fatty acid level during prolonged exercise in individuals with paraplegia suggests that adrenal medullary activity and the subsequent release of these substrates is not adversely affected at the lower spinal lesions.[23] Blood lactate levels increase significantly during the early stages of exercise and tend to decline towards the latter stages of the exercise bout.[22-24] This is most likely caused by an increase in lactate utilisation by other tissues during exercise. During a simulated wheelchair race, individuals with paraplegia accumulate a significantly higher level of blood lactate than those with quadriplegia, implying that the degree of anaerobic metabolism is greater in the former group. This is despite the fact that individuals with paraplegia sustain a higher relative intensity during the race than those with quadriplegia.[25] The overall pattern of substrate utilisation during prolonged exercise in those with spinal cord injury is consistent with that observed in able-bodied individuals.

4. Skeletal Muscle Fibre Types in Spinal Cord Injury: Implications for Wheelchair Racing Performance

It is well documented[26,27] that individuals with spinal cord injury have a preponderance of type IIb (fast glycolytic) motor units in the paralysed limbs with a concomitant reduction in the type I (slow oxidative) motor units. In individuals with quadriplegia, this alteration is evident in the muscles of the lower extremities (vastus lateralis) and the upper extremities (deltoid).[28] The shift in the motor unit characteristics is caused by the lack or reduction of neurological stimuli to the paralysed muscle, as well as the sedentary lifestyle that results from the injury. The overall effect is that the aerobic capacity of the exercising muscles is compromised, which will limit their ability to participate in distance events that are dependent upon long-term sources of energy. However, many athletes with spinal cord injury, including those with low-level quadriplegia, routinely participate in long-distance events such as the marathon and their performance has improved steadily over the years. One of the reasons for this is that the oxidative capacity of the motor units recruited during wheelchair racing is enhanced as a result of extensive physical training.[28,29] It is well documented[30] that able-bodied individuals who succeed in distance running events that are dependent upon aerobic metabolism have a large proportion of type I motor units in the exercising muscle, whereas those who excel in sprint events that are dependent upon anaerobic metabolism have a high percentage of type II motor units. While this characteristic can be attributed to the type of training that these individuals undertake, it is likely that their genetic predisposition is more suited to these sporting events. It is unclear

whether this relationship between motor unit characteristics and wheelchair racing performance is true for individuals with spinal cord injury.

Glycogen depletion studies on able-bodied individuals have demonstrated that the recruitment of motor units during exercise is dependent upon the intensity and duration of the effort.[30] During low intensity efforts, the slow-twitch motor units are primarily recruited whereas during high intensity efforts the slow- and fast-twitch motor units are activated. This differential recruitment pattern is attributed to differences in the activation threshold between the two types of motor units. From a functional standpoint during wheelchair racing, the recruitment of motor units will depend upon the intensity and duration of the race. During short races such as the 100 and 200m sprints, the primary motor units that will be recruited are the type IIb motor units because they have a fast contractile speed and high glycolytic capacity. The energy for these events will be derived mainly from the immediate- and short-term sources, which have a limited capacity, and therefore, the intensity of performance will be reduced as the duration of the race increases. In contrast, during long-distance events such as the wheelchair marathon, the slow-twitch motor units will be recruited because of their high oxidative capacity. However, as the race progresses these motor units will become fatigued and a greater proportion of the fast twitch units will be recruited. Moreover, athletes competing in distance events usually try and increase the velocity towards the end of the race, and as a result, will recruit a greater number of the type II motor units during this period. Research pertaining to motor unit recruitment patterns during wheelchair racing is limited. The glycogen depletion evidence that is available[21] suggests that during submaximal wheelchair exercise at 40 to 60% of peak aerobic power, the slow oxidative motor units in the deltoid muscle are recruited, which is analogous to the trend described in able-bodied individuals.

5. Anaerobic Fitness in Individuals with Spinal Cord Injury

Numerous studies have evaluated the anaerobic and aerobic fitness in individuals with spinal cord injury. While there is considerable information that is available on wheelchair athletes, including wheelchair racers, these studies have several limitations. Firstly, in a vast majority of the studies, the sample sizes within a disability classification are quite small. This limits the generalisations that can be made about the findings to the population under consideration. Secondly, in several studies, descriptive statistics on individuals from different classes are pooled which makes it difficult to evaluate the results. Thirdly, variations in the degree of the spinal cord lesion (complete *vs* incomplete) have a significant impact on the physiological responses, which further confounds the results. Fourthly, differences in the testing modes and protocols make comparisons amongst the studies difficult. Finally, most studies have been conducted on males with a limited database available on females.

The anaerobic power is defined as the maximum amount of power that can be generated using anaerobic (immediate- and short-term) sources of energy production. Anaerobic capacity is defined as the average power that can be developed over a given period of time using the anaerobic energy sources.[20] The Wingate anaerobic test, which was developed in Israel in the mid 1970s for able-bodied individuals,[31] has been adapted for the assessment of anaerobic power and capacity of individuals with spinal cord injury. The two common modes of exercise used for this evaluation are arm cranking (ACE) and wheelchair exercise. Several different instruments have been developed to quantify the power output during wheelchair exercise. These include: (i) computerised stationary wheelchair ergometer (WERG); (ii) wheelchair mounted on a treadmill (WCT); and (iii) wheelchair mounted on rollers (WCR). Details pertinent to these instruments are reviewed elsewhere.[14] Other techniques of evaluating anaerobic power in individuals with spinal cord injury include: (i) quantification of the instantaneous peak power that can be developed during

a wheelchair drag test;[32] and (ii) development of the maximal wheelchair velocity under minimal braking forces which has been shown to be similar to wheelchair racing performance during actual performance.[33]

Currently there is no standardised method for determining the resistance for the Wingate test protocol in individuals with spinal cord injury. Because of the reduced muscle mass resulting from paralysis, the resistance applied is considerably lower than that used for able-bodied individuals. It is generally recommended that the load setting for wheelchair users performing the Wingate test be based upon the functionality, training status and gender of the individuals.[34] The test-retest reliability of the 30-second Wingate wheelchair test in wheelchair athletes has been established. In competitive wheelchair racers with paraplegia evaluated three times during a 5-week interval, the peak power, mean power and maximum velocity demonstrated a high degree of stability.[35] In older competitive male and female wheelchair basketball players, significant reliability coefficients have been reported for the peak power (0.95), mean power (0.92), peak velocity (0.88), mean velocity (0.91) and peak velocity fatigue index (0.62).[36] Nonsignificant correlation coefficients were observed for the mean power (0.49) and fatigue index of the peak power (0.40). No significant differences were observed between the means of the two trials for each of these variables except the mean velocity which was significantly higher in trial two and the peak velocity fatigue index which was significantly lower in trial two. In general, the athletes performed better on the second test, which was most likely caused by a learning effect from the first test. The researchers recommended that future Wingate anaerobic power studies include a complete habituation session before administration of the actual test.

The results from numerous studies that have evaluated the anaerobic power during wheelchair ergometry, including individuals with spinal cord injury, are summarised in an earlier review.[14] The study cited, however, does not indicate the power output relative to body mass (Watts/kg) and the gender of the participants evaluated. To overcome these limitations, the current review presents the absolute and relative values of the peak and average power output in males and females in tables II and III, respectively. The evidence indicates that the peak anaerobic power generated during the Wingate test is inversely related to the level of lesion; that is, the lower the injury level the greater the peak power output. The peak anaerobic power output of a class V wheelchair athlete can exceed that of a class IB or IC athlete by 3 to 4 times.[14,34,37,38] There appears to be no significant difference between able-bodied individuals and those with low level paraplegia (lesion at T8 or lower) for the absolute or relative values of anaerobic power during a 30-second Wingate test. However, the application of torque during wheelchair propulsion seems to be more effective in wheelchair-dependent individuals which could be attributed to their specific training.[39] Gender comparisons[32,36] indicate that: (i) the absolute and relative values of the peak power, mean power and mean velocity are significantly higher in male compared with female wheelchair athletes; (ii) the velocity fatigue index is significantly higher in females compared with males, suggesting a greater decline in force output during the test in females; and (iii) there is no significant correlation between peak power and body mass in either gender. Values of peak anaerobic power output, expressed as a function of lean body mass, are not available for male and female wheelchair athletes. It is hypothesised that the magnitude of this gender difference in peak power would be reduced because of the significantly lower percentage of lean body mass in females.

The fatigue index of elite wheelchair athletes (basketball players) during the Wingate test is 50 to 60%, implying an ~50% decline in power at the end of the test when compared with the peak values during the 30-second period.[14,43] Analysis of the performance during the WERG test indicates that the power profile of athletes becomes less effective at higher velocities, because the force production drops thereby maintaining a constant power output.[45] The validity of the anaerobic capacity mea-

Table II. Anaerobic power in untrained and trained male wheelchair athletes with spinal cord injury

Study	Mode	n	ISMGF Classification	Lesion	Peak power (W)	Peak power (W/kg)	Average power (W)	Average power (W/kg)
Quadriplegia								
Coutts and Stogryn[40]	WERG	2 WA	IA, IB	C6-C7	46	0.77	41.4	0.51
Dallmeijer et al.[41]	WERG	6 UT	IA, IB, IC	C4-C8	121.8[a]	1.47[a]	21.5	0.26
Janssen et al.[42]	WERG	9 UT	I				33.3	0.41
van der Woude et al.[37]	WERG	3 WA		C5/6-C7	23	0.36		
		4 WA		C7	68	1.03		
Paraplegia								
Dallmeijer et al.[41]	WERG	5 UT	II	T1-T5	248.7[a]	3.0[a]	46.9	0.57
		5 UT	III	T6-T10	398.1[a]	5.1[a]	63.7	0.81
		7 UT	IV	T11-L4	310.3[a]	4.0[a]	49.1	0.63
Hutzler et al.[38]	ACE	13 WA	II	T1-T5	280			
		15 WA	III-VI	T6-S5	336			
Hutzler et al.[43]	WERG	11 WB 1 polio		T5-L3	169.3	2.32	149.3	2.07
Janssen et al.[42]	WERG	6 UT	II				70.4	0.85
		15 UT	III				95.9	1.22
		12 UT	IV				114.0	1.45
		2 UT	V				100.5	1.47
Lees and Arthur[35]	WERG	6 WA	II-V	T1-L5	124.8-221.6[b]	2.1-3.7[b]	102.1-148.6[b]	1.7-2.5[b]
Roeleveld et al.[44]	WERG	9 WA		T8-L5			59.4[c]	0.99[c]
Van der Woude et al.[37]	WERG	8 WA		T5-L1	118	1.95	100	1.65
		23 WA		T6-S1	170	2.85	138	2.36
Veeger et al.[32]	WERG	4 WA	II	T1-T5	65.8[d]	0.78[d]		
		7 WA	III	T6-T10	79.8[d]	1.08[d]		
		11 WA	IV	T11-L3	85.4[d]	1.27[d]		
		7 WA	V	L4-S1	79.3[d]	1.34[d]		
Veeger et al.[39]	WERG	9 UT		T8 and below	57.7	0.79	50.2[c]	0.69[c]

a Calculated as the average of the three highest strokes.

b Power output at work loads ranging between 1.2 and 2.4kg during the Wingate tests.

c Power calculated for right hand only.

d Power determined during a wheelchair drag test on a treadmill.

ACE = arm crank ergometry; **ISMGF** = International Stoke Mandeville Games Federation functional classification; **n** = sample size; **UT** = untrained participants; **WA** = wheelchair athletes (mixed group of athletes); **WB** = wheelchair basketball players; **WERG** = wheelchair ergometer.

sured by the Wingate test against a 30-second field sprint test in elite wheelchair basketball players has been demonstrated.[46] The large common variance of 71% between the two tests, implies that both these tests could be useful in evaluating the anaerobic performance in wheelchair basketball players. Wheelchair athletes who participate in events that stress the anaerobic energy system have a higher anaerobic power than those who participate in events that overload the aerobic system.[32,37,40] For example, basketball players whose training and sport involves intermittent exercise have a higher peak power output than track and field athletes whose events and training are not as dependent upon the anaerobic energy system. Furthermore, both these groups of athletes have higher peak power outputs than those who participate in events that are less metabolically demanding such as archery and target shooting.[32]

There is a moderate to high correlation (0.37 to 0.90) between the various indices of anaerobic power

(5-second peak and 30-second average) and the power output during an incremental aerobic exercise test. The proportion of the peak power output attained during incremental exercise and the anaerobic power indices ranged between 17 to 85% and 14 to 81% for the peak and average values, respectively.[14] The large variation amongst these studies can be attributed to the differences in the lesion levels, testing modes and fitness status of the participants. The aerobic contribution of the wheelchair anaerobic power test is estimated as 30%, which is within the range reported for able-bodied individuals.[43] There seems to be a significant relationship between the oxygen cost of the Wingate anaerobic power test and functional classification in wheelchair basketball athletes. This implies that muscle mass available for performance plays an important role in developing anaerobic power.[46]

6. Aerobic Fitness in Individuals with Spinal Cord Injury

6.1 Peak Aerobic Power

The maximal aerobic power [maximal oxygen uptake ($\dot{V}O_{2max}$)] is defined as the maximum amount of oxygen that can be utilised per unit time. It is usually expressed as an absolute value in litres per minute or relative to bodyweight as millilitres per kilogram per minute. The $\dot{V}O_{2max}$ is dependent upon the overall ability to transport, deliver and utilise oxygen, and therefore, is considered to be the best measure of maximal cardiorespiratory fitness. Mathematically, the $\dot{V}O_{2max}$ is determined by the product of the maximal values of the cardiac output (central factor) and the mixed arterio-venous oxygen difference [$(a - \bar{v})O_{2diff}$] (peripheral factor). The former is determined by the product of the maximal values of heart rate and stroke volume during exercise.[20]

The instrumentation used to assess aerobic fitness in individuals with spinal cord injury is similar to that used for the measurement of anaerobic power, that is, ACE, WERG, WCR and WCT. To assess the peak physiological responses during ACE, the individual performs asynchronous incremental cycling exercise with the upper extremities against a resistance at specified cadence. In the case of wheelchair exercise, the individual performs synchronous upper body exercise at a specific work rate (WERG), velocity (WCR) or combination of velocity and slope (WCT). In each of these exercise modes, the intensity is increased at regular intervals until voluntary fatigue is induced. Research has indicated that the mechanical efficiency of WERG (7 to 8%) is considerably less than that of ACE (17 to 18%), which in turn, is lower than that of cycle ergometry (23%).[47] Minimal differences in metabolic efficiency have been reported between asynchronous and synchronous ACE in able-bodied individuals and individuals with paraplegia.[48] In general, the stress of

Table III. Anaerobic power in female wheelchair athletes with paraplegia

Study	Mode	n	ISMGF classification	Lesion	Peak power (W)	Peak power (W/kg)	Average power (W)	Average power (W/kg)
Quadriplegia								
van der Woude et al.[37]	WERG	4 WA		C5-C7, polio	46	1.0	38	0.83
Paraplegia								
Coutts and Stogryn[40]	WERG	1 WA	IV	T10	85	1.73		
Veeger et al. [32]	WERG	48 WA[a]	IC, III-IV		39.4[b]	0.66		
van der Woude et al.[37]	WERG	3 WA		T8, spina bifida	89	1.71	77	1.47
		3 WA		T12-S1, spina bifida, polio				

a Includes one athlete with quadriplegia.

b Power determined during a wheelchair drag test on a treadmill.

ISMGF = International Stoke Mandeville Games Federation functional classification;**n** = sample size; **WA** = wheelchair athletes (mixed group of athletes); **WERG** = wheelchair ergometry.

wheelchair exercise is extremely high when the physiological responses are expressed as a percentage of the peak physiological capacity. When evaluating wheelchair athletes, it is preferable to use wheelchair propulsion instead of ACE because it is specific to the mode of ambulation during the race. Because the muscle mass utilised during upper body exercise is considerably smaller than that recruited during lower body exercise, it is difficult to fully tax the cardiovascular system and attain true $\dot{V}O_{2max}$ values. Hence, the term peak oxygen uptake ($\dot{V}O_{2peak}$) is commonly used instead of $\dot{V}O_{2max}$ when referring to these data.

The validity of the peak physiological responses during different WCT protocols has been demonstrated.[49,50] Studies that have compared ACE with WCT or WCR protocols have indicated no significant differences in the $\dot{V}O_{2peak}$ between the two exercise modes, although peak values of heart rate may be slightly different.[51,52] No significant differences in $\dot{V}O_{2peak}$ have also been reported between 1-minute continuous incremental and 3-minute discontinuous incremental WCT protocols in individuals with paraplegia.[53] The optimum work rate increment for eliciting the $\dot{V}O_{2peak}$ in individuals with quadriplegia during ACE or WERG is between 2 and 6W each minute. Work rate increments of 8W and greater tend to underestimate the $\dot{V}O_{2peak}$.[54,55] Significant test-retest reliability coefficients have been reported for the peak values of the oxygen uptake ($\dot{V}O_2$) [r = 0.98] and heart rate (r = 0.97) during WCR in individuals with quadriplegia and paraplegia.[56] In individuals with quadriplegia, the peak values of the power output and the cardiorespiratory responses are significantly greater when ACE is conducted in the supine posture compared with the sitting posture.[57] It is postulated that these differences are caused by greater ventilation, enhanced venous return and greater trunk stability in the supine position.

Several researchers have adapted valid field tests for able-bodied individuals to assess the $\dot{V}O_{2peak}$ in individuals with spinal cord injury. The Leger and Boucher multistage incremental running test for able-bodied individuals has been validated for measuring the $\dot{V}O_{2peak}$ during wheelchair propulsion in male athletes with class II to V paraplegia.[58] An intra-class correlation of 0.65 has been reported between direct measurements of $\dot{V}O_{2peak}$ during an incremental WCT and the adapted wheelchair test. However, the correlation between the maximal speed attained during the test and the $\dot{V}O_{2peak}$ was not significant (r = 0.18). The authors suggested that other biomechanical, anthropometric and physiological factors were important determinants of the $\dot{V}O_{2peak}$. The short-term (3 to 10 days) and long-term (8 days to 1 month) reliability of the peak heart rate and maximal speed measurements of this adapted field test has also been demonstrated in male athletes with paraplegia.[59] The 12-minute wheelchair propulsion distance (designed on the basis of the Cooper 12-minute run-walk) is significantly related to the $\dot{V}O_{2peak}$ in individuals with paraplegia[60,61] but not with quadriplegia.[60] The correlation coefficient is stronger when the $\dot{V}O_{2peak}$ is expressed relative to bodyweight (0.84 *vs* 0.54), suggesting that body mass plays an important role in determining wheelchair propulsion distance. The strength of the relationship is improved by including other variables such as blood pressure, age and height of the individuals.

The peak physiological responses of sedentary and competitive athletes with spinal cord injury have been extensively documented during the last 3 decades. The reader is referred to earlier publications[7,12,13] for expected peak values of the $\dot{V}O_2$ and heart rate in these individuals. When the physiological responses are evaluated across the whole range of lesion levels (C5 to S1) in individuals with spinal cord injury, the $\dot{V}O_{2peak}$ and other related physiological measures such as heart rate, cardiac output, ventilation rate and blood lactate are inversely related to the lesion level.[62-64] This means that the higher the level of injury the lower the peak responses and vice versa. The physiological responses in well-trained athletes with paraplegia are not necessarily dependent upon the lesion level. Research has demonstrated minimal differences in $\dot{V}O_2$, heart rate and ventilation rate between athletes with high and low level paraplegia.[65] At a given lesion level,

the completeness of the injury also plays an important role in determining the peak physiological responses during exercise. Incomplete lesions may enable the individual to attain higher heart rates and recruit a greater proportion of the muscle mass, thereby resulting in higher $\dot{V}O_{2peak}$ values caused by greater central and peripheral contributions.[24,66]

A summary of several studies published during the last decade in untrained and trained male and female athletes with quadriplegia and paraplegia is available in tables IV to VII. Comparison amongst studies is difficult because of differences in testing mode, protocols, level and completeness of injury. To minimise variations in $\dot{V}O_2$ that could be caused by differences in heart rate at the different lesion levels, the values of the oxygen pulse (O_2 pulse, ratio between absolute $\dot{V}O_2$ and heart rate), which indicates oxygen utilisation per heart beat have also been presented. It is evident that: (i) the values are significantly higher in individuals with paraplegia compared with those with quadriplegia; (ii) there is considerable overlap among the different classes in individuals with quadriplegia and paraplegia; (iii) the values are greater in trained compared with untrained individuals in both these groups; and (iv) the values for well trained individuals with quadriplegia can exceed those observed in untrained individuals with paraplegia. The last three points make it difficult to use this physiological variable to distinguish among the different ISMGF classes.

The cross sectional evidence presented in tables IV to VII clearly indicates that the $\dot{V}O_{2peak}$ of endurance-trained individuals with spinal cord injury is significantly greater than their untrained counterparts.[67,81,82] Although much of this increase can be attributed to the endurance training responses of these individuals, two other important factors must be considered:[7] (i) the genetic predisposition of the individuals for aerobic metabolism (eg. having a preponderance of type I motor units); and (ii) the fitness level of the individuals before the injury. It is likely that individuals who had a high aerobic power before the injury would also adapt more readily to endurance training post injury, which could result in their higher $\dot{V}O_{2peak}$ values.

The $\dot{V}O_{2peak}$ in individuals with quadriplegia is significantly lower than that of able-bodied individuals performing ACE,[63] with the value in the former group being ~40% of the able-bodied individuals. This difference was maintained even when the O_2 pulse was compared between the two groups. However, comparisons between able-bodied indi-

Table IV. Peak aerobic power in untrained and trained male wheelchair athletes with quadriplegia

Study	Mode	n	ISMGF classification	Lesion	$\dot{V}O_2$ (L/min)	$\dot{V}O_2$ (ml/kg/min)	Heart rate (beats/min)	Oxygen pulse (ml/beat)
Untrained								
Bhambhani et al.[67]	WCR	8 RR	IA, B, C	C5-C8	1.06	14.9	118	8.9
Eriksson et al.[66]	WCR	12 UT			0.88	13.9	119	7.5
		6 UT i			1.18	16.4	138	8.7
Janssen et al.[42]	WCT	9 UT	I		1.11	13.6		
Trained								
Bhambhani et al.[67]	WCR	8 RR	IA, B, C	C5-C8	1.43	19.8	121	11.8
Campbell et al.[24]	WCT	1 WA		C7 i	1.39		164	8.5
		1 WA		C7	1.06		113	9.4
Eriksson et al.[66]	WCR	8 RR			1.11	17.4	118	9.6
Ready[68]	ACE	6 WA	IA, IB	C6-C7	1.08	15.0	121.8	8.9
van der Woude et al.[34]	WERG	3 WA		C5-C7	0.67	10.9		
		4 WA		C6-C7	1.30	19.7		

ACE = arm crank ergometer; **i** = incomplete lesion; **ISMGF** = International Stoke Mandeville Games Federation functional classification; **n** = sample size; **RR** = wheelchair road racers; **UT** = untrained wheelchair dependent participants; $\dot{V}O_2$ = oxygen uptake; **WA** = wheelchair athletes (mixed group of athletes); **WCR** = wheelchair roller system; **WCT** = wheelchair mounted on treadmill.

Table V. Peak aerobic power in untrained and trained male wheelchair athletes with paraplegia

Study	Mode	n	ISMGF classification	Lesion	$\dot{V}O_2$ (L/min)	$\dot{V}O_2$ (ml/kg/min)	Heart rate (beats/min)	Oxygen pulse (ml/beat)
Untrained								
Eriksson et al.[66]	WCR	9 UT			1.66	22.0	186	9.0
		6 UT i			1.81	25.4	183	11.9
Janssen et al.[42]	WCT	6 UT	II		1.45	17.5		
		15 UT	III		1.67	21.2		
		12 UT	IV		2.03	25.7		
		2 UT	V		2.14	31.3		
Mossberg et al.[48]	ACE	11 UT[a]		T5-L5	1.40	19.4	180	7.8
Raymond et al.[69]	ACE	10 UT		T5-T12	1.81	26.3	175	10.3
Trained								
Bernard et al.[65]	WERG	7 WA	II, III	T1-T10	1.79	28.5	183	9.8
		6 WA	IV	T11-L3	2.35	34.3	169	13.9
Campbell et al.[24]	WCT	10 WA		T2-L2	1.91		196	9.7
Coutts[70]	WERG	3 WR	III-IV	NA	3.28	51.2	183.7	17.9
Cooper et al.[49]b	WERG	11 WR	II-IV	T3-L1	2.46	37.4	186.5	13.2
Eriksson et al.[66]	WCR	17 WR			2.16	33.6	183	11.9
Gass and Camp[22]	WCT	6 WR	III-IV	T10-L3	2.86	47.5	190	15.0
Hooker and Wells[71]	ACE	6 WR	II-IV	T4-T12 i	2.72	43.1	180.3	15.1
Huonker et al.[72]	WERG	20 UT	II-V	T1-S2	1.76	23.9	161.8	10.5
		29 WA	II-V	T1-S2	2.42	34.5	183.3	13.2
O'Connor et al.[73]	WERG	6WR			2.29	36.2	186	12.3
Okuma et al.[74]	WCT	8 WR		T7-L1	2.44	46.5	169	13.4
Price and Campbell[75]	ACE	11WA		T3-4/L1	2.04	30.5	185	11.0
van der Woude et al.[34]	WERG	8 WA		T3-L1	2.04	32.9		
		23 WA		T6-S1, polio, spina bifida	2.29	38.1		
Veeger et al.[32]	WCT	6 WA	II	T1-T5	1.84	23.0	170	10.8
		10 WA	III	T6-T10	1.97	26.8	175	11.2
		13 WA	IV	T11-L3	2.42	36.9	182	13.3
		7 WA	V	L4-S1	2.38	40.6	182	13.2
Vinet et al.[76]	WCT	8 WA	III-V	T8-L5	2.67	40.6	174	15.3

a Includes one female participant.

b Average of values obtained on incremental speed and incremental resistance protocols.

ACE = arm crank ergometer; **i** = incomplete lesion; **ISMGF** = International Stoke Mandeville Games Federation functional classification; **n** = sample size; **NA** = not available; **UT** = untrained participants; $\dot{V}O_2$ = oxygen uptake; **WA** = wheelchair athletes (mixed group of athletes); **WCR** = wheelchair roller system; **WCT** = wheelchair mounted on treadmill; **WERG** = wheelchair ergometer.

viduals and individuals with paraplegia depend upon the training status of the two groups and the exercise mode. One study[48] indicated a minimal difference in the $\dot{V}O_{2peak}$ during ACE between untrained able-bodied individuals and those with paraplegia despite an 11% higher heart rate in the latter group. However, calculation of the peak O_2 pulse indicated a 25% higher value in the able-bodied individuals. During WERG,[72] the $\dot{V}O_{2peak}$ in able-bodied individuals was ~32% higher than that of untrained individuals with paraplegia but 10% lower than that of trained athletes with paraplegia. The O_2 pulse was lower in the untrained individuals with paraplegia by 20%, with no difference being observed between

the able-bodied individuals and the athletes with paraplegia. Comparisons between trained able-bodied individuals (canoeists) and trained athletes with paraplegia indicate that the former group has a 41% higher $\dot{V}O_{2peak}$ and 75% higher peak O_2 pulse compared with the latter.[75] Since the O_2 pulse is closely related to stroke volume and not the $(a - \bar{v})O_{2diff}$ in healthy individuals[83] and individuals with spinal cord injury, [84] the differences in this variable among the different groups most likely reflect differences in their stroke volume during exercise.

6.2 Physiological Factors Influencing Oxygen Uptake During Exercise

The lower $\dot{V}O_{2peak}$ during incremental exercise in individuals with spinal cord injury is due to alterations in the central and peripheral factors that determine the $\dot{V}O_2$.[63,85-89] At lesion levels above T1, sympathetic stimulation to the myocardium is disrupted and the individual is physiologically unable to attain the age predicted maximal heart rate. This significantly reduces the maximal cardiac output, thereby diminishing the overall capacity to transport oxygen to the tissues. At lesion levels below T1, the myocardium is fully innervated and the individual should theoretically be able to attain the age predicted maximal heart rate. This is clearly evident in the peak heart rate values presented in tables IV to VII. The stroke volume during peak exercise is lower in individuals with spinal cord injury which further contributes to the hypokinetic circulation.[85-87] This reduction is attributed to the venous pooling that takes place in the lower extremities because of the reduction in muscle pump action. Although the respiratory muscle pump in individuals with quadriplegia does facilitate venous return to the heart, the magnitude of the muscle pump is greatly compromised, thereby resulting in the reduced cardiac preload and stroke volume during exercise. Echocardiographic evidence indicates that left ventricular volume is lower in untrained individuals with spinal cord injury compared with able-bodied individuals.[72] However, there is some controversy whether this difference persists when comparisons between sedentary and active individuals with paraplegia are conducted. Some evidence suggests wheelchair athletes with paraplegia have significantly higher left ventricular volumes[72] while other observations[90] indicate no significant difference between the two groups. Left ventricular ejection fraction during WERG in individuals with paraplegia is similar to that observed in able-bodied individuals and does not seem to be affected by the level of physical activity in individuals with paraplegia.[72]

Peripheral oxygen extraction during peak exercise in individuals with spinal cord injury, as reflected by the $(a - \bar{v})O_{2diff}$, is controversial. No significant differences have been reported among able-bodied individuals and individuals with quadriplegia and quadriplegia during peak ACE.[63] The lower $\dot{V}O_{2peak}$ of those with spinal cord injury was attributed primarily to the hypokinetic circulation as a result of lower heart rate and stroke volume during peak exercise. Other evidence[89] indicates that the $(a - \bar{v})O_{2diff}$ is significantly lower in individuals with high level paraplegia compared with able-bodied individuals. The lower $\dot{V}O_{2peak}$ in those with paraplegia was caused primarily by the reduced extraction as the values of cardiac output

Table VI. Peak aerobic power in trained female wheelchair athletes with quadriplegia

Study	Mode	n	ISMGF classification	Lesion	$\dot{V}O_2$ (L/min)	$\dot{V}O_2$ (ml/kg/min)	Heart rate (beats/min)	Oxygen pulse (ml/beat)
Skrinar et al.[21]	WERG	1 WA	IB	C4-5/T6	1.23	17.3	175	7.0
van der Woude et al.[34]	WERG	4 WA		C5-C7, polio	0.74	16.1		
Veeger et al.[32]	WCT	1 WA	IC	C7-C8	0.80			

ISMGF = International Stoke Mandeville Games Federation functional classification; **n** = sample size; $\dot{V}O_2$ = oxygen uptake; **WA** = wheelchair athlete; **WCT** = wheelchair mounted on treadmill; **WERG** = wheelchair ergometer.

Table VII. Peak aerobic power in untrained and trained female wheelchair athletes with paraplegia

Study	Mode	n	ISMGF classification	Lesion	$\dot{V}O_2$ (L/min)	$\dot{V}O_2$ (ml/kg/min)	Heart rate (beats/min)	Oxygen pulse (ml/beat)
Untrained								
Schmid et al.[77]	WERG	10 UT		T1-L5	1.09	18.3	180	6.1
Tahamont et al.[78]	WERG	6 UT		T10-L4	0.90	14.8	150	6.0
Trained								
Hooker and Wells[71]	ACE	1 RR	III	T10	1.99	38.0	167	11.9
Morris[79]	ACE	1 RR		T3-T4	1.32	20.3	161	8.2
Schmid et al.[77]	WERG	13 BB	I-III	T1-L5	1.90	33.7	181	10.5
Vanlandewijck et al.[80]	WCT	10 BB	I	T3-T12	1.67	25.5		
		11 BB	II	T9-T12	2.40	33.9		
		4 BB	III	L1-L5	2.49	36.1		
		9 BB	IV	Amputee, polio, spina bifida	2.62	38.6		
van der Woude et al.[34]	3 WA			T8, spina bifida	1.30	24.9		
	3 WA			T12-S1, spina bifida, polio	1.15	22.8		
Veeger et al.[32]	WCT	8 WA	IC, III-V	C7/8-S1	1.22	20.7	172	7.1

ACE = arm crank ergometer; **BB** = wheelchair basketball players; **ISMGF** = International Stoke Mandeville Games Federation functional classification; **n** = sample size; **RR** = wheelchair road racers; **UT** = untrained wheelchair dependent participants; $\dot{V}O_2$ = oxygen uptake; **WA** = wheelchair athletes (mixed group of athletes, including one athlete with quadriplegia); **WCT** = wheelchair mounted on treadmill; **WERG** = wheelchair ergometer.

and stroke volume were not different between the two groups. It is likely that the aerobic and functional capacity of the active musculature play an important role in determining peripheral oxygen extraction during exercise. Individuals with lesion levels below the T10 level (ISMFG class IV or lower) are able to utilise a large proportion of the trunk musculature during upper body exercise which will result in higher $(a - \bar{v})O_{2diff}$ and $\dot{V}O_{2peak}$ values than those with injuries at higher lesion levels whose ability to utilise these muscles is reduced or non-existent. It is interesting to note that the peak blood lactate level, an index of anaerobic metabolism, is similar in able-bodied individuals and those with paraplegia during ACE.[48,81] However, significantly higher values have been reported in individuals with paraplegia compared with quadriplegia, most likely due to differences in the active muscle mass available for exercise and catecholamine responses between the two groups.[81]

Several studies have evaluated the physiological factors that determine the $\dot{V}O_2$ during submaximal exercise. At the same absolute $\dot{V}O_2$ during ACE, the cardiac output is significantly lower while $(a - \bar{v})O_{2diff}$ is significantly higher in untrained individuals with quadriplegia compared with able-bodied individuals.[91] However, comparisons between individuals with paraplegia and able-bodied individuals indicate no significant differences in the cardiac output and $(a - \bar{v})O_{2diff}$ at the same absolute $\dot{V}O_2$.[87,88] These observations collectively suggest that the exercising muscles in individuals with quadriplegia have the capacity to enhance oxygen extraction when blood perfusion is reduced. This is an interesting phenomenon in light of the fact that the aerobic capacity of the skeletal muscle is compromised in individuals with spinal cord injury.[26-29] During submaximal exercise at the same relative intensity (i.e. percentage of $\dot{V}O_{2peak}$), no significant differences have been reported between able-bodied individuals and those with paraplegia for the cardiac output and $(a - \bar{v})O_{2diff}$. With respect to the cardiac output, research has consistently demonstrated that this is attained by a lower stroke volume and proportionate increase in heart rate in those with spinal cord injury.[86-88]

There is a linear relationship between heart rate and $\dot{V}O_2$ in individuals with spinal cord injury, which is analogous to that observed in able-bodied individuals.[57,82,89,92] This relationship is stronger in individuals with low level compared with high level quadriplegia.[57] The slope of the relationship between heart rate and absolute $\dot{V}O_2$ is significantly greater in those with high-level paraplegia compared with able-bodied individuals during incremental ACE, implying that the increase in heart rate for a unit increase in $\dot{V}O_2$ is higher in the former group. However, when the slope is calculated as a percentage of the $\dot{V}O_{2peak}$, there is no significant difference between the two groups of participants.[89] Furthermore, the slope between percentage heart rate and percentage $\dot{V}O_2$ is not significantly different among individuals with high and low level paraplegia and able-bodied individuals. [92] These findings suggest that training programmes prescribed on the basis of target heart rate measurements need not be altered for individuals with paraplegia.

6.3 Lactate and Ventilatory Threshold

The lactate threshold is defined as the lowest $\dot{V}O_2$ at which a significant amount of lactate accumulates in the blood during a continuous incremental test to voluntary exhaustion.[93] The lactate threshold indicates the maximum amount of energy that can be derived from aerobic sources without the accumulation of lactate. This threshold is determined during the incremental $\dot{V}O_{2peak}$ test using invasive and/or non-invasive methods. Invasively, it is identified as the exercise intensity ($\dot{V}O_2$) at which the lactate level increases exponentially during the incremental test. Although this is the recommended method for identifying the individual lactate threshold, some researchers have utilised a fixed lactate level of 4 mmol/L as a measure of this parameter.

The lactate threshold can also be determined non-invasively from the respiratory gas exchange responses measured during the incremental exercise test.[93] It is generally accepted that when a significant amount of lactate accumulates in the blood, an additional amount of carbon dioxide, above that formed as a result of aerobic metabolism, is released due to the buffering of lactate by bicarbonate in the blood. The increased carbon dioxide production stimulates the peripheral chemoreceptors thereby stimulating the ventilatory drive. The net result is that the ventilation rate, carbon dioxide production and respiratory exchange ratio all increase nonlinearly at the lactate threshold. Improved criteria for detecting this threshold from the respiratory gas exchange measurements are a systematic increase in the ventilatory equivalent for oxygen (ratio between ventilation rate and oxygen consumption), without a concomitant increase in the ventilatory equivalent for carbon dioxide (ratio between ventilation rate and carbon dioxide production). In the scientific literature, this exercise intensity is usually referred to as the ventilatory threshold because the exact physiological mechanism of the relationship between lactate accumulation and the respiratory gas exchange measurements is not completely understood and is currently under debate.

The lactate (ventilatory) threshold is considered to be the best indicator of submaximal aerobic fitness because it represents the exercise intensity at which energy for muscular work can be produced aerobically without the utilisation of anaerobic sources.[20] At this threshold, oxygen supplied by the central circulation is sufficient to meet the requirements of the muscle, resulting in the majority of the energy being produced under aerobic conditions. At exercise intensities that are below this threshold, the individual can continue exercising for prolonged periods because there is minimal accumulation of lactate, a metabolite that is implicated with muscular fatigue. At intensities above the threshold, blood lactate increases in an exponential manner with respect to exercise intensity, and exercise performance is curtailed at the high work rates. Laboratory research on able-bodied individuals has indicated that the lactate threshold may be a better predictor of endurance performance than the $\dot{V}O_{2max}$.[94,95] During prolonged treadmill running, individuals select a running velocity at which the $\dot{V}O_2$ is closely related to that observed at the lactate (ventilatory) threshold. Hence, it is important that athletes com-

peting in endurance events have a high lactate (ventilatory) threshold when the values are expressed relative to the $\dot{V}O_{2max}$.

The validity and test-retest reliability of detecting the lactate threshold from respiratory gas exchange measurements in individuals with spinal cord injury has not been demonstrated. The inter-rater reliability of this detection during WCR in individuals with quadriplegia is 0.90.[62] A summary of studies that have evaluated either the lactate or ventilatory threshold in individuals with spinal cord injury is presented in table VIII. Two of the three lactate threshold studies[96,97] have used the 4 mmol/L value to identify this intensity and did not undertake respiratory gas exchange measurements during the tests. Therefore, the findings of these two studies are not included in the table. The evidence suggests that the absolute power output at the lactate threshold is significantly higher in endurance trained wheelchair racers compared with untrained individuals. However, the relative power output values (expressed as a percentage of the peak value during the incremental test) at the lactate threshold are significantly higher in the untrained individuals compared with the endurance trained racers, primarily because of the lower peak power in the former group.[97] These differences in power output are not reflected by significant differences in the relative values of the heart rate at this threshold. In elite basketball players, the 4 mmol/L lactate threshold occurs at a heart rate of 139 beats/min which corresponds to 57% of the heart rate reserve.[98] The evidence from the remaining study[99] indicates that there is no significant difference in the absolute $\dot{V}O_2$ at the lactate threshold between able-bodied individuals and untrained individuals with high and low level paraplegia during ACE. However, because those with paraplegia have lower $\dot{V}O_{2peak}$ values than able-bodied individuals, the lactate threshold occurs at a higher percentage of $\dot{V}O_{2peak}$ in the former group. This is most likely caused by the intracellular adaptations resulting from the regular upper body training in individuals with paraplegia.

The absolute $\dot{V}O_2$ at the ventilatory threshold in wheelchair athletes with class IA to IC quadriplegia is significantly lower than those with class III

Table VIII. Summary of studies that have evaluated the lactate or ventilatory threshold in individuals with spinal cord injury

Study	Mode	n	ISMGF classification	Lesion	Peak $\dot{V}O_2$ (L/min)	$\dot{V}O_2$ at lactate or ventilatory threshold		
						L/min	ml/kg/min	% peak
Lactate threshold								
Flandrois et al.[99]	ACE	5 HP		T4-T7	1.20	0.76	10.7	63
		4 LP		T12-L2	2.40	1.42	14.8	59
Ventilatory Threshold								
Bhambhani et al.[67]	WCR	8 UT	IA, IB, IC	C5-C8	1.06	0.67	9.0	63
		8 RR	IA, IB, IC	C5-C8	1.43	0.83	11.4	58
Coutts and McKenzie[98]	WCR	2 WA	IA		1.09	0.93		84
		4 WA	IB		0.83	0.69		85
		3 WA	IC		1.03	0.95		92
		4 WA	II		1.95	1.36		69
		4 WA	III		2.43	1.78		74
		7 WA	IV		2.73	1.89		69
		3 WA	V		2.63	1.72		66
Lin et al.[100]	ACE	9 UT	II	T1-T5	0.95	0.58	10.9	61
		11 UT	III	T6-T10	1.05	0.78	13.2	74
		19 UT	IV-V	T11-S2	1.24	0.78	13.5	63
Vinet et al.[76]	WCT	8 WA	III-V	T8 to L5	2.67	1.53	23.1	56

ACE = arm crank ergometry; **HP** = high-level paraplegia; **ISMGF** = International Stoke Mandeville Games Federation functional classification; **LP** = low-level paraplegia; **n** = sample size; **RR** = wheelchair road racers; **UT** = untrained wheelchair dependent participants; $\dot{V}O_2$ = oxygen uptake; **WA** = wheelchair athletes (mixed group of athletes); **WCR** = wheelchair roller system; **WCT** = wheelchair mounted on treadmill.

to V paraplegia. However, when the values are expressed relative to the $\dot{V}O_{2peak}$, the trend is reversed. The mean value of 87% for the athletes with quadriplegia is significantly higher than the value of 69% reported for those with paraplegia. This could be due to loss of sympathetic control to the heart and/or loss of central innervation to the intercostal muscles in those with quadriplegia, which could influence their respiratory response to incremental exercise.[98] During ACE, the absolute $\dot{V}O_2$ at the ventilatory threshold in individuals with class II paraplegia is significantly lower when compared with able-bodied individuals. However, values for the class III and IV individuals are not significantly different from those of able-bodied individuals.[100] The finding that the ventilatory threshold occurs at a significantly higher percentage of $\dot{V}O_{2peak}$ in those with paraplegia when compared with able-bodied individuals is consistent with that reported for the lactate threshold.[82]

The absolute $\dot{V}O_2$ at the ventilatory threshold during simulated wheelchair exercise is significantly higher in competitive wheelchair marathon racers with quadriplegia compared with their untrained counterparts. There is a strong correlation between the absolute $\dot{V}O_2$ at the ventilatory threshold and the $\dot{V}O_{2peak}$ ($r = 0.86$ and 0.81 for untrained and trained individuals, respectively), implying that individuals with a higher level of aerobic fitness were also able to attain a higher $\dot{V}O_2$ before the accumulation of significant amounts of lactate during incremental exercise.[62] When comparing the results by sport participation, track athletes demonstrated significantly higher values for the absolute $\dot{V}O_2$ at the ventilatory threshold and peak exercise when compared with basketball players and other athletes (swimming, table tennis and target shooting). No significant differences were observed among the athletes when the values at the ventilatory threshold were expressed as a percentage of $\dot{V}O_{2peak}$.[98] Similar results were obtained when comparisons between track athletes and tennis players were made.[76] To date, the relationship between the lactate or ventilatory thresholds and wheelchair racing performance has not been examined. Neither has this parameter been evaluated in female wheelchair athletes. Given the importance of this parameter in endurance performance, it is important that such studies be undertaken in the future.

7. Physiological Factors Associated with Wheelchair Racing Performance

During the last few decades, the knowledge gained from research in the field of exercise physiology has played an important role in improving training techniques for able-bodied sport. There is a strong research database that has documented the relationship between sport performance and the five essential components of physical fitness, namely, anaerobic power and capacity, aerobic power, muscular strength and endurance, body composition and flexibility. This information has led to several developments and refinements in testing, training and monitoring of athletes on a continual basis.[101] However, this does not seem to be the case with disability sport. An international study[102] pertaining to the training practices of elite athletes with disabilities (41 wheelchair racers, 20 swimmers and 14 throwers) indicated that a third of the wheelchair racers did not receive any coaching. Possible reasons include inadequate information pertaining to the training principles in wheelchair sport and lack of qualified personnel in this field. To improve training techniques, avoid counteractive training behaviours and avoid the risk of overtraining, it is important that scientific knowledge in the area of disability sport be developed.[103] Although there is considerable descriptive information on the physiological profiles of wheelchair athletes, particularly road racers and basketball players, research that has identified factors associated with improved performance in wheelchair racers is limited. The available evidence suggests that some of the physiological parameters described above can be used to predict success in sprint and distance racing performance.

7.1 Sprint Racing Performance

The relationships between selected measures of anaerobic fitness and sprint racing performance in wheelchair athletes are presented in table IX. There

is a strong inverse correlation between Wingate test results performed during wheelchair exercise and performance during sprint races ranging between 100 to 400m in elite wheelchair athletes.[33,35,37,104] Similar relationships are also observed for other sprint events such as a 30-second sprint, shuttle run and slalom.[42,105] However, it has been reported that Wingate anaerobic power measured during arm cranking is not a good predictor of success in wheelchair basketball players performing a 400m race, slalom and 6-minute wheelchair endurance race.[105] The lack of association between the physiological parameters and performance could be due to the difference in the testing modes: arm cranking for the measurement of anaerobic power versus wheeling during the performance tests. The study cited [105] demonstrated that peak heart rate attained during arm cranking was significantly correlated with wheeling tasks such as a 400m race, slalom and 6-minute wheelchair endurance race. There appears to be no significant relationship between power production and wheelchair marathon times.[34] In general, the indices of anaerobic power vary considerably among the different disabilities and sports disciplines and are strongly influenced by the functionality, number of hours of training and gender of the athletes.[34]

7.2 Distance Racing Performance

Currently there is limited research that has evaluated the physiological responses during competitive wheelchair racing. The results from several field and laboratory studies that have analysed such performance are summarised in table X. The criterion validity of simulated wheelchair racing performed on frictionless rollers in the laboratory against indoor track racing performance has been established.[25] In individuals with quadriplegia and paraplegia, significant correlations between the track and simulated tests have been reported for the racing time (r = 0.79 and 0.82), wheeling velocity (r = 0.81 and 0.88), heart rate (r = 0.89 and 0.88) and blood lactate (r = 0.77 and 0.81) in the two respective groups. The $\dot{V}O_2$ and heart rate during distance racing performance in elite wheelchair racers with quadriplegia and paraplegia range between 90 to 95% of the peak values observed during incremental wheelchair exercise.[107-109] One study[109] reported that the relative intensity at marathon race pace was considerably higher in wheelchair marathoners with paraplegia compared with elite marathon runners. The wheelchair racers sustained a $\dot{V}O_2$ and heart rate that corresponded to 94 and 96% of their respective peak values during incremental exercise, while the marathon runners sustained values of 78 and 85%, respectively. The absolute values of the

Table IX. Relationship between indices of anaerobic power and wheelchair sprint racing performance in wheelchair athletes

Study	Mode	n	Event	Peak power (5 sec)	Average power (30 sec)
Bar-Or et al.[106]	ACE	17 UT	3 × 26m	–0.70[a]	
			Slalom: 3-4 min	–0.64[a]	
Hutzler[105]	ACE	9 BB	428m	–0.10	–0.43
			Slalom	–0.11	–0.38
Lees[33]	WERG	9 WA	100m	–0.79[a]	–0.78[a]
			800m	–0.56[a]	–0.26
Lees[104]	WERG	17 WA	100m	–0.73[a]	
			200m	–0.35	
Lees and Arthur[35]	WERG	7 WA	100m	–0.95[a]	–0.89[a]
			400m	–0.86[a]	–0.88[a]
van der Woude et al.[37]	WERG	20 WA	200m		–0.79[a]
Vanlandewijck et al.[46]	WCR	15 BB	30 sec sprint		0.93[a]

a Indicates significant correlation at the 0.05 level.

ACE = arm crank ergometry; **BB** = basketball players; **n** = sample size; **UT** = untrained wheelchair dependent participants; **WA** = wheelchair athletes (mixed group of athletes); **WCR** = wheelchair roller system; **WERG** = wheelchair ergometer.

Table X. Oxygen uptake ($\dot{V}O_2$) and heart rate during wheelchair racing performance in athletes and nonathletes with spinal cord injury

Study	Mode	n	ISMGF classification	Lesion	Distance	% peak $\dot{V}O_2$	% peak heart rate
Quadriplegia							
Bhambhani et al.[107]	WCR	8 RR	IB, IC	C6-C8	7.5km	94%	93%
Bhambhani et al.[25]	WCR	7 UT	IA, IB, IC	C5-C8	1.6km	76%[a]	86%[a]
Paraplegia							
Asayama et al.[108]	WCR	11 RR	II-V	T4-L5	Marathon	90%	93%
Bhambhani et al.[25]	WCR	6 UT	II-V	T5-L4	3.2km	95%[a]	95%[a]
Crews[109]	WCF	3 RR	II-IV	T4-L3	Marathon race pace	96%[b]	97%[b]
	Running	3 MR				78%	85%
Gayle et al.[51]	WCF	15 UT	II-V	T4-L6	1.6km		100%
Lakomy[110]	WCT	12 WA	II-V	T5-L5	5km	79%	80-90%

a Significant differences between untrained paraplegics and quadriplegics at similar stages of the race.

b Significantly different from the marathon runners tested.

ISMGF = International Stoke Mandeville Games Federation functional classification; **MR** = able-bodied marathon runners; **n** = sample size; **RR** = wheelchair road racers; **UT** = untrained wheelchair dependent participants; **WA** = wheelchair athletes (mixed group of athletes, including two athletes with quadriplegia); **WCF** = wheelchair field test; **WCR** = wheelchair roller system; **WCT** = wheelchair mounted on treadmill.

$\dot{V}O_2$ and heart rate during simulated wheelchair racing are higher in athletes with paraplegia compared with quadriplegia.[25] Contributing factors are the hypokinetic circulation and reduced muscle mass available for exercise in athletes with quadriplegia. However, there is some controversy pertaining to the relative intensity (i.e. $\dot{V}O_2$ expressed as a percentage of the peak value) that is sustained during wheelchair racing between these two groups. One report indicated that this value is significantly higher in individuals with paraplegia[25] while another suggested that it is higher in those with quadriplegia.[110] The reason for this discrepancy is unclear and needs to be elucidated because it has important implications for training. Wheelchair racing for 16 minutes at 60, 70, 80 and 90% of the top speed attained during an incremental $\dot{V}O_{2peak}$ test in well trained male wheelchair athletes does not match the corresponding values of the $\dot{V}O_{2peak}$.[24] Since wheelchair athletes frequently train at a percentage of the peak speed (assuming that it corresponds to the same percentage of $\dot{V}O_{2peak}$) the use of this method for training purposes is discouraged.

The fact that elite wheelchair athletes sustain $\dot{V}O_2$ and heart rate values during the race that are close to their respective peak values during incremental exercise suggests that these two physiological variables might be directly related to successful racing performance. However, the results from several studies that are presented in tables XI and XII indicate that the evidence is controversial. Some researchers[68,111] reported no significant relationship between the $\dot{V}O_{2peak}$ and actual wheelchair racing velocity recorded during a 10km road race in well-trained athletes with paraplegia. Others[107,110] have documented significant correlations between the $\dot{V}O_{2peak}$ and time taken to complete simulated wheelchair races of 5 and 7.5km in athletes with paraplegia and quadriplegia. The former study[107] also demonstrated that wheelchair racing performance was significantly related to the absolute $\dot{V}O_2$ ($r = –0.90$), cardiac output ($r = –0.88$) and stroke volume ($r = –0.82$) during the race, but not the $(a - \bar{v})O_{2diff}$ ($r = 0.42$), age (–0.47) and lesion level (0.58) of the athletes. These observations suggest that: (i) aerobic power is important for successful wheelchair distance racing performance; and (ii) central factors related to oxygen transport play a more important role than peripheral factors in enhancing performance. Besides these metabolic and cardiorespiratory variables, other evidence indicates that in elite wheelchair racers, arm power measured on a steel spring apparatus and total lung capacity play an important role in successful wheelchair marathon racing per-

Table XI. Correlations between peak oxygen uptake ($\dot{V}O_{2peak}$) and distance wheelchair racing performance in individuals with spinal cord injury

Study	Mode	n	ISMGF classification	Lesion	Distance/ duration	$\dot{V}O_{2peak}$ (L/min)	$\dot{V}O_{2peak}$ (ml/kg/min)
Quadriplegia							
Bhambhani et al.[107]	WCR	8 RR	IB, IC	C6-C8	7.5 km	–0.79 *vs* time[a]	–0.73 *vs* time[a]
Bhambhani et al.[25]	WCR	7 RA	IA, IB, IC	C5-C8	1.6km	0.23 *vs* velocity[b]	0.32 *vs* velocity[b]
Rhodes et al.[60]	WCF	16 UT	IA, IB, IC	C5-C8	12 min	0.12 *vs* distance[b]	
Paraplegia							
Bhambhani et al.[25]	WCR	6 RA	II-V	T5-L4	3.2 km	0.52 *vs* velocity[b]	0.50 *vs* velocity[b]
Cooper[111]	WERG	11 RR	II-V	T5-L4	10 km	0.02 *vs* time[b]	
Franklin et al.[61]	ACE	30 UT	II-V		12 min		0.84[a]
Hooker and Wells[71]	ACE	7 RR[c]	II-IV	T4-T12	10 km	–0.06 *vs* time[b]	
Lakomy[110]	WCT	12 WA	II-V	T5-L5	5 km	0.61*vs* velocity[a]	
Rhodes et al.[60]	WCF	14 UT	II-VI	T4-S1	12 min	0.23	

a Correlation significant at the 0.05 level; $p > 0.05$.

b Correlation not significant.

c Includes one female participant.

ACE = arm cranking ergometer; **ISMGF** = International Stoke Mandeville Games Federation functional classification; **n** = sample size; **RA** = recreational athletes; **RR** = wheelchair road racers; **UT** = untrained wheelchair dependent participants; **WA** = wheelchair athletes (mixed group of athletes, including two athletes with quadriplegia); **WCF** = wheelchair field test; **WCR** = wheelchair roller system; **WCT** = wheelchair mounted on treadmill; **WERG** = wheelchair ergometer.

formance.[112] In general, the differences in wheelchair racing velocity of world-class athletes are dependent upon the race distances, as well as the gender and classes of the athletes.[113]

In light of the current research evidence pertaining to wheelchair racing performance in athletes with spinal cord injury, it can be concluded that the generally accepted concept that high values of anaerobic and aerobic power are strongly associated with sprint and endurance racing performance, respectively, are not necessarily true in this population. Further research is needed to elucidate the factors that best predict performance in these events.

8. Temperature Regulation During Wheelchair Racing in Athletes with Spinal Cord Injury

8.1 Temperature Regulation in Spinal Cord Injury

During intense exercise such as a long distance wheelchair race, energy metabolism in the localised muscles increases substantially. A large portion of this energy is unproductive and is released in the form of heat. To maintain core body temperature fairly close to the resting value of 37ºC, it is essential that this thermal energy be dissipated fairly rapidly. Failure to do so would result in symptoms of hyperthermia, which include dehydration, heat exhaustion, heat syncope and heat stroke.[114] The brain initiates three distinct mechanisms to dissipate heat:[114,115] (i) increased circulation to the skin blood vessels thereby facilitating dry heat loss by means of convection and radiation; (ii) stimulation of the sweat glands via the sudomotor centre, which enhances evaporative heat loss; and (iii) redistribution of cardiac output so that there is greater blood flow to the active muscles via vasodilation and a reduction in blood flow to the splanchnic area because of vasoconstriction. Environmental conditions play an important role in initiating the pertinent mechanism for maintaining core body temperature. When exercise is performed in a cool environment, radiation and convection are the primary modes of heat loss. However, when exercise is performed in a hot dry environment, 80 to 90% of the heat is dissipated via evaporative sweat loss. This proportion, however, is significantly reduced when the

relative humidity increases beyond 70%. Under these conditions, there is increased reliance on convective and radiative heat loss. As a result, a greater proportion of the cardiac output is diverted to the cutaneous blood vessels to facilitate heat loss. This reduces the venous return to the heart, and to maintain the cardiac output for the given exercise intensity, heart rate is increased proportionately. This is commonly referred to the 'cardiovascular drift' that occurs during prolonged exercise.

Individuals with spinal cord injury, both athletes and nonathletes, have an impaired thermoregulatory capacity compared with their able-bodied counterparts. This is most likely caused by impairment of the autonomic and somatic nervous systems which disrupts control of skin blood flow and sweating below the level of lesion.[116,117] The consequence of the limited vascular control over a large proportion of the skin is an elevated core body temperature at rest and during exercise, which could adversely affect wheelchair racing performance. It has been demonstrated[117,118] that the degree of thermoregulatory impairment in individuals with spinal cord injury is inversely related to the level and completeness of the lesion. This implies that individuals with quadriplegia will have a lower heat tolerance than those with paraplegia and able-bodied individuals. The elevated core body and skin temperature during prolonged exercise in the heat by those with spinal cord injury could be related to their inability to maintain cardiac output during prolonged exercise.[119,120] Individuals with spinal cord injury have a decreased skin blood flow and sweating capacity for a given core body temperature compared with able-bodied individuals. This could be caused by the reduced afferent feedback that stimulates sweating during exercise.[117] In individuals with paraplegia: (i) oesophageal temperature may be a better index of core body temperature than rectal temperature;[121] and (ii) there may be some inconsistency between the sites where vasomotor control is lost and the areas where sweating capacity is reduced.[122] This difference is observed in several somato-sensory dermatomes, suggesting an anatomical dissociation of the vasomotor and sudomotor efferent pathways. Arm movements such as propelling a wheelchair during racing can substantially enhance evaporative heat loss. However, individuals with paraplegia and incomplete quadriplegia can also dissipate heat via sweating in the arm and head. There is some evidence which suggests that individuals with quadriplegia may have localised head and upper body sweat rates that are higher than those of their able-bodied counter-

Table XII. Correlations between distance wheelchair racing performance and selected physiological variables measured during the race in individuals with spinal cord injury

Study	Mode	n	ISMGF classification	Lesion	Distance	$\dot{V}O_2$ (L/min)	$\dot{V}O_2$ (L/kg/min)	Other variables
Quadriplegia								
Bhambhani et al.[107]	WCR	8 RR	IB, IC	C6-C8	7.5km	–0.90 *vs* time[a]	–0.77 *vs* time[a]	–0.88 *vs* cardiac output; –0.82 *vs* stroke volume; 0.42 *vs* $(a - \bar{v})O_{2diff}$
Bhambhani et al.[25]	WCR	7 RA	IA, IB, IC	C5-C8	1.6km	NS *vs* time	NS *vs* time	
Paraplegia								
Asayama et al.[108]	WCR	11 RR	IA, IB, IC	C6-C8	Marathon	NS *vs* velocity	NS *vs* velocity	
Bhambhani et al.[25]	WCR	6 RA	II-V	T5-L4	3.2km	NS *vs* velocity	NS *vs* velocity	
Lakomy[110]	WCT	12 WA	II-V	T5-L5	5km	0.69 *vs* velocity[a]		

a Correlation significant at the 0.05 level.

$(a - \bar{v})O_{2diff}$ = arterio-venous oxygen difference; **ISMGF** = International Stoke Mandeville Games Federation functional classification; **n** = number of participants; **NS** = correlation not significant; **RA** = recreational athletes; **RR** = wheelchair road racers; **WA** = wheelchair athletes (mixed group of athletes, including two athletes with quadriplegia); **WCR** = wheelchair mounted on rollers, **WCT** = wheelchair mounted on treadmill; **$\dot{V}O_2$** = oxygen uptake.

parts.[118] The use of medications to alter bladder capacity and voiding mechanisms by wheelchair road racers can adversely affect sweating and thermoregulatory function.[123]

Some important points pertaining to research studies in spinal cord injury thermoregulation should be noted. Firstly, some studies [119,124] demonstrated no significant difference or lower core body temperature in individuals with spinal cord injury compared with able-bodied individuals. In both these investigations, the participants with spinal cord injury were exercising at similar relative but lower absolute metabolic rates than the able-bodied individuals. Because changes in core body temperature are proportional to the absolute metabolic rate during exercise,[117] these findings should be interpreted with caution. Secondly, it has been reported[125] that individuals with quadriplegia do not demonstrate signs of hyperthermia when exercising for 30 minutes at 50% $\dot{V}O_{2peak}$ in 23ºC temperature. This is most likely caused by the low $\dot{V}O_2$ during exercise. Thirdly, several investigations[118,125-127] were performed during arm cranking which is a more efficient form of exercise than wheelchair ergometry. It is likely, therefore, that the thermoregulatory stress during wheelchair exercise (which is specific to wheelchair racing) would be greater, which could result in even larger differences between the spinal cord injured and able-bodied individuals. Finally, the exercise duration in these studies ranged between 30 to 90 minutes of continuous exercise. In some cases, the intensity expressed as a percentage of $\dot{V}O_{2peak}$ was not given. Although two studies[120,124] evaluated the responses for 90 minutes, which is slightly lower than the current marathon race time for elite wheelchair racers, the intensity of exercise was only 53% of $\dot{V}O_{2peak}$ or 80% of peak heart rate, which is well below that experienced during competitive wheelchair racing (table X). It is recommended that future thermoregulation studies be designed to simulate not only the environmental conditions experienced during the race, but also the exercise mode, intensity and duration of the effort so that the impact of the findings in terms of wheelchair racing performance is maximised.

8.2 Methods of Minimising Heat Stress During Wheelchair Racing

It is generally accepted that healthy individuals who are aerobically fit (i.e. have a high $\dot{V}O_{2max}$) are at a lower risk for hyperthermia during exercise. This suggests that regular physical training that stresses the cardiorespiratory system plays an important role in minimising the changes in core body temperature during prolonged exercise in hot and humid conditions. Some of the reasons for this include: increased plasma volume, reduced threshold temperature for the onset of sweating, lower skin and rectal temperatures, reduced localised muscle blood flow during submaximal exercise which results in more blood being available for temperature regulation, increased stroke volume and cardiac output during exercise.[114,128]

Research that has examined the effects of heat acclimatisation (i.e. regular exposure to hot and humid conditions) and/or regular physical training on the thermoregulatory responses in athletes with spinal cord injury have not been conducted to date. Until such information is available, recommendations for minimising heat stress in the spinal cord injured population should be drawn from the findings on able-bodied athletes. Athletes and their trainers should consider the following when preparing for competition:[129,130]

(i) Focus on proper aerobic training, which enhances thermoregulatory capacity during exercise. Most researchers agree that training in a cool environment enhances the thermoregulatory responses when exercise is performed at high environmental temperatures

(ii) Avoid training in climatic conditions that can increase the risk of thermal stress. Usually heat loss mechanisms are compromised when environmental conditions exceed 21ºC and 50% relative humidity

(iii) Induce heat acclimatisation with caution and ensure that adequate fluid-electrolyte balance is maintained during this period

(iv) Avoid competing under conditions that are known to increase susceptibility to heat stress; for

example, periods of inactivity due to injury or sickness
(v) Ensure that athletes get adequate rest and sleep when competitions involve travel across different time zones
(vi) Wear lightweight, loose fitting clothes so that sweat can evaporate from the skin surface to facilitate cooling
(vii) Keep an accurate record of body mass to ensure that athletes are not losing excessive amounts of fluid.

It is generally recommended that ~1L of fluid be consumed for each kilogram of bodyweight lost. If fluid loss exceeds 4 to 6% of bodyweight, the intensity and duration of training should be reduced. When weight loss exceeds this amount, a physician should be consulted. Localised cooling devices that are worn on the head may reduce heart rate and oesophageal temperature in wheelchair athletes exercising under thermal stress.[131] These physiological changes are observed despite similar sweating rates with and without the cooling device. Although these preliminary results look promising, further research is necessary to corroborate these findings before these devices can be used during wheelchair racing.

9. Methods for Enhancing Physiological Factors Associated with Wheelchair Racing Performance

9.1 Autonomic Dysreflexia

Autonomic dysreflexia, as the term suggests, is a reflex syndrome that is unique to individuals with spinal cord injury at lesion levels above the major sympathetic splanchnic outflow (injury level above T6).[132,133] The exact mechanism of autonomic dysreflexia is not completely understood. It is postulated that this response is triggered by nociceptive stimuli distal to the lesion level which result in afferent stimuli that transcend the spinal cord, providing collateral connections to the pre-ganglionic cell bodies of the intermedio-lateral horn, thereby resulting in a massive sympathetic discharge. In individuals with high-level quadriplegia, the magnitude of the sympathetic discharge is amplified. This is most likely caused by: (i) denervation hypersensitivity of sympathetic spinal, ganglionic or peripheral receptor sites; (ii) loss of supraspinal inhibitory control; and (iii) formation of abnormal synaptic connections resulting from axonal resprouting. The sympathetic discharge results in peripheral piloerection and vasoconstriction, which is evident in the form of gooseflesh, shivering and pallor distal to the level of injury. In addition, there is a large increase in systemic blood pressure. In an attempt to buffer the increase in blood pressure, the aortic and carotid baroreceptors are stimulated, which in turn activate the parasympathetic nervous system proximal to the lesion level. However, the descending impulses originating from the vasodilatory centre of the medulla are unable to traverse the spinal cord at the level of the lesion, and therefore, peripheral vasoconstriction and systemic hypertension can not be regulated in the normal manner. The elevated blood pressure can result in several serious conditions such as cerebral haemorrhage, aphasia, blindness, cardiac arrhythmias and death.[132,133]

Many athletes with spinal cord injury who compete in wheelchair distance races such as the marathon, voluntarily induce autonomic dysreflexia before or during the event to enhance their performance. The nociceptive stimuli commonly used to induce this reflex are: (i) overdistending the bladder; (ii) sitting on sharp objects; and (iii) use of tight leg straps. This procedure, which is commonly referred to as 'boosting', is done 1 or 2 hours before the actual race performance for the reflex to be fully effective. It is postulated that the elevated blood pressure in the dysreflexic condition would increase the cardiac output, thereby improving racing performance. To date, only one research study has scientifically examined the effects of autonomic dysreflexia on wheelchair distance racing performance.[134] In this study, eight elite male wheelchair marathon racers voluntarily 'boosted' themselves by the method of their choice on two separate occasions. They subsequently performed a 7.5km simulated wheelchair race on a frictionless roller system on each day. Cardiovascular, metabolic and hormonal meas-

urements were undertaken at regular intervals during the tests. The results indicated that 'boosting' improved wheelchair racing time by an average of 10% when compared with the control 'unboosted' race performed on two separate days. Metabolic measurements indicated that the athletes utilised a greater amount of oxygen during the 'boosted' races. The increased $\dot{V}O_2$ was not caused by an increase in cardiac output, as one might expect as a result of autonomic dysreflexia, but was caused by enhanced oxygen extraction from the blood as evidenced by the increased $(a - \bar{v})O_{2diff}$. Systolic blood pressure was elevated before and during the race, but did not reach the dangerous levels according to the World Health Organization classification. No significant alterations were observed in the levels of free fatty acids, glucose and blood lactate between the two conditions, although adrenaline (epinephrine) levels were elevated in the 'boosted' state.[133,135] These findings clearly indicate that 'boosting' provides high level spinal cord injured athletes with an unfair advantage during distance racing events and the practice should be disallowed during competition.

The International Paralympic Committee Medical and Anti-Doping Code considers voluntary boosting to be an unethical and illegal practice. Because boosting can be dangerous to health, athletes are banned from voluntarily inducing this condition during competition.[5] Since it is difficult to differentiate between spontaneous autonomic dysreflexia and voluntary boosting, all athletes who demonstrate signs of the condition are subject to medical examination before the race. This includes measurement of blood pressure, heart rate, dyspnoea, level of sweating, presence or absence of skin blotching, goose flesh, anxiety and tremors. If the athlete demonstrates signs and symptoms that are consistent with autonomic dysreflexia, a re-examination is done after 10 minutes for confirmation. Once this is ascertained, further examination of the athlete with respect to cutaneous, visceral and proprioceptive stimuli known to induce autonomic dysreflexia is considered. Athletes who are deemed at risk may be asked to withdraw from competition after consultation with their representatives and the Chair of the International Paralympic Committee Medical Commission.

9.2 Lower Body Positive Pressure

Many wheelchair racers with spinal cord injury routinely strap their lower extremities to gain a competitive edge over fellow athletes. The most likely reason for this is to reduce venous pooling which could enhance cardiac output thereby improving performance. Direct experimental evidence to support this practice during wheelchair racing is not available. However, several research studies have examined the effects of lower body positive pressure on the physiological responses of individuals with spinal cord injury. These studies have used antigravity suits, abdominal binders and stockings to increase the lower body positive pressure to induce a redistribution of blood flow during exercise. An increase in lower body positive pressure elevates the abdominal pressure as well as the pressure on the lower extremities in individuals with spinal cord injury. This decreases venous capacitance and facilitates venous return to the heart. The increased abdominal pressure also minimises orthostatic hypotension, which could improve the central circulation.[136] It has been demonstrated that the use of antigravity suits can significantly elevate the $\dot{V}O_{2peak}$ during arm cranking and wheelchair exercise in untrained individuals with paraplegia and quadriplegia,[137] as well as wheelchair marathon racers with paraplegia, but not in able-bodied individuals.[138] The mechanism for the increase in $\dot{V}O_{2peak}$ is unclear. In untrained individuals with paraplegia and quadriplegia,[137] a significant increase in cardiac output and stroke volume has been reported, most likely as a result of an enhanced venous return to the heart. In the wheelchair marathon racers, [138] no significant alterations were observed in these two variables during submaximal exercise. It is possible that this discrepancy between the two studies could be caused by differences in the lesion levels and the training status of the individuals.

The use of an abdominal binder does not have a significant effect on the physiological and bio-

mechanical responses during submaximal wheelchair exercise in athletes with paraplegia.[139] No significant alterations in the $\dot{V}O_2$ and heart rate during submaximal or maximal exercise have been reported. Furthermore, the biomechanical variables such as the cycle, stroke and recovery times which could influence wheelchair racing performance were unchanged. The results of this study suggest that the use of an abdominal binder will not aid wheelchair racing performance in individuals with spinal cord injury. A comparison between the use of anti-gravity suits and a combination of abdominal binders and stockings indicated that both these methods of inducing lower body positive pressure have a positive effect on the submaximal cardiovascular responses during ACE.[140] The improvement in performance was greater in individuals with quadriplegia compared with those with paraplegia, with anti-gravity suits inducing a larger improvement than the binder/stocking combination. To gain the maximum benefit from the anti-gravity suits, it is recommended that the pressure be set at a value which is 10 to 20mm Hg below the individual diastolic blood pressure.[140] On the basis of these observations, it may be concluded that the use of anti-gravity suits significantly enhances the cardiovascular responses during submaximal and maximal exercise in individuals with quadriplegia and paraplegia. Theoretically, if distance wheelchair racing performance is associated with peak values of $\dot{V}O_2$ and cardiac output, as previously reported,[107] then this technique should be effective in improving performance.

10. Conclusion

Wheelchair racing is one of the most frequently participated competitive sporting activities by athletes with spinal cord injury. During the last 2 decades, considerable progress has been made in evaluating the anaerobic power and capacity, aerobic power and the lactate/ventilatory threshold in this population. However, research pertaining to the relationship between these physiological factors and wheelchair racing performance is limited. The available evidence suggests that the generally accepted concept that high values of anaerobic and aerobic power are strongly associated with sprint and endurance racing performance, respectively, are not necessarily true in this population. Athletes with spinal cord injury have an impaired thermoregulatory capacity and may be more susceptible to thermal stress when compared with able-bodied athletes. Therefore, they should take precautions to minimise the effects of dehydration, heat exhaustion and heat stroke during distance racing events. Wheelchair athletes with quadriplegia who voluntarily induce autonomic dysreflexia, commonly known as boosting, may enhance distance racing performance by increasing their aerobic power. However, this practice is banned by the International Paralympic Committee not because of its performance enhancing capabilities, but because it could be dangerous to the athletes' health. Other methods of improving exercise performance, such as an increase in lower body positive pressure, may improve the peak physiological responses during exercise but their influence on wheelchair racing performance has not been evaluated.

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