Habilitation Thesis

Exercise in Persons with Spinal Cord Injury Testing - Training - Optimization

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Publication Date:
2012

Permanent Link:
https://doi.org/10.3929/ethz-a-010062184

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Exercise in Persons with Spinal Cord Injury: Testing - Training - Optimization

Habilitation Thesis

presented by
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November 2012
Dedicated to my parents
Acknowledgement

This thesis would not have been possible without the support of so many people and institutions in many ways. First of all many thanks to my academic teacher at the ETH Zurich, Prof. Urs Boutellier, for his interest, encouragement and advice during the preparation of this thesis.

I would also like to thank to all the co-workers of the Institute of Sports Medicine Nottwil headed by Dr. Matthias Strupler for their understanding, patience and daily support. Further I would like to acknowledge the Swiss Paraplegic Centre as well as the Swiss Paraplegic Foundation. These two institutions enabled the use of the whole infrastructure without restrictions and financially supported several of my research projects.

Thanks a lot to all my research colleagues and master students, namely Prof. Ken Hunt, PD Dr. Tanja Kakebeeke, Dr. Helen Berry, Dr. Angela Frotsler, Dr. Vicky Goosey-Tolfrey, Dr. Rob Labruyère, Dr. Gabi Mueller, Christof Leicht and Nadine Stoffel-Kurt for the pleasant collaborations, projects and all the fruitful scientific discussions over the past few years.

I am grateful to all the patients and athletes for their voluntary participation in all the research projects. They spent many hours in the laboratory producing many drops of sweat while exercising and making it possible to gain all the knowledge presented in this work.

Last but not least many thanks to my parents and my family for their perpetual support, not only concerning this thesis but also my whole life.
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1 Introduction

1.1 Spinal cord injury

A spinal cord injury (SCI) leads to considerable physical changes of persons concerned, resulting in an impairment or loss of motor, sensory and vegetative functions. Based on the severity of the spinal damage a SCI is classified as complete or incomplete. In the case of an incomplete SCI some sensation and potentially some motor function below the lesion level are preserved, whereas a complete SCI leads to a total loss of sensation and motor function below the level of injury. Injuries at the level of the cervical spinal cord are termed as tetraplegia and result in impairment of function in arms, trunk, legs and pelvic organs. If the lesion level is located below the first thoracic spinal nerve the impairment is specified as paraplegia. In paraplegia arm function is preserved but function of legs, trunk and pelvic organs may be affected based on lesion level [American Spinal Injury Association, 2002].

Functional independence after a SCI is largely determined by neurological level and completeness of injury. The classification of SCI refers to the most caudal segment of the spinal cord with normal sensory and motor function. In clinical practice, the neurological classification of SCI is performed according to the international standards recommended by the American Spinal Injury Association (ASIA) [2002] and consists of five categories (A to E) according to the ASIA impairment scale (Table 1).

Table 1: ASIA impairment scale

<table>
<thead>
<tr>
<th>Description of categories</th>
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<tbody>
<tr>
<td><strong>A</strong> Complete. No sensory or motor function is preserved in the sacral segments S4-S5.</td>
</tr>
<tr>
<td><strong>B</strong> Incomplete. Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5.</td>
</tr>
<tr>
<td><strong>C</strong> Incomplete. Motor function is preserved below the neurological level, and more than half of the key muscles below the neurological level have a muscle grade less than 3 (=active full range movements against gravity).</td>
</tr>
<tr>
<td><strong>D</strong> Incomplete. Motor function is preserved below the neurological level, and at least half of the key muscles below the neurological level have a muscle grade greater than or equal to 3.</td>
</tr>
<tr>
<td><strong>E</strong> Normal. Sensory and motor functions are normal.</td>
</tr>
</tbody>
</table>

ASIA: American Spinal Injury Association
Epidemiology of spinal cord injury

Incidence of SCI differs widely between countries and is reported to lie between 10 and 83 cases per million inhabitants per year [Wyndaele and Wyndaele, 2006]. The incidence of SCI in the United States was estimated as 40 cases per million population in the year 2006, corresponding to approximately 11'000 new cases of SCI per year [Lim and Tow, 2007], whereas Germany as well as Switzerland reported 30 cases of SCI per million population [Felleiter et al., 2004]. This is at the upper limit of the European incidence range of 10 to 30 cases per million inhabitants [Wyndaele and Wyndaele, 2006].

Most of all spinal cord injuries have a traumatic origin, such as traffic accidents (e.g. automobiles, motorcycles and bicycles), sport accidents (e.g. diving, skiing), falls and violence (e.g. gun shot) [Agarwal et al., 2007; Ho et al., 2007; Jackson et al., 2004]. Non-traumatic spinal cord injuries are scarcely documented but reported to occur in up to 30% of the cases [Agarwal et al., 2007; Zäch and Koch, 2006c], with an increasing tendency during the past few years [Eberhard, 2004]. Reasons for non-traumatic SCI include cancer, infections, arthritis and inflammation of the spinal cord [Zäch and Koch, 2006c].

Cervical injuries (tetraplegia) occurred in 30% [Wyndaele and Wyndaele, 2006] to 50% [Jackson et al., 2004] of the cases reported and complete injuries seem to be more frequent (55%) [Jackson et al., 2004] compared to incomplete SCI. Further, about 80% of the SCI are related to men [De Vivo, 2007; Ho et al., 2007] and the average age at injury currently ranges between 33 and 38 years [De Vivo, 2007; Ho et al., 2007; Wyndaele and Wyndaele, 2006] but seems to increase steadily over time.

Morbidity and mortality

Life expectancy of people with SCI has increased during the past decades [Yeo et al., 1998] but remains still lower than in the general population, even with optimal medical management [Hartkopp et al., 1997]. Age, as well as the level and degree of neurological impairment seem to be important prognostic factors for survival after suffering from a SCI [Catz et al., 2002; Whiteneck et al., 1992; Yeo et al., 1998]. Persons with complete tetraplegia achieve 70% and people with complete paraplegia 84% of the life time expectancy, whereas an incomplete tetra- or paraplegia leads to a life time expectancy of 92% compared to the general population [Yeo et al., 1998]. Especially within the first year after SCI, mortality rate is elevated compared to the general population [DeVivo et al., 1999; Yeo et al., 1998]. However, approximately 80% of the persons with SCI survive the first ten years after injury but survival rate dramatically drops to 50% within 30 years after SCI [Catz et al., 2002; Whiteneck et al., 1992].

Taking into account the past decades, urinary tract infections and other urologic complications were the primary cause of death after SCI. However, with progress in medical management and the considerably increasing life expectancy, there has been a shift in the primary causes of death towards other conditions in the past years [Krause et al., 2004]. Nowadays, the most common causes of death in persons suffering from SCI were diseases of the circulatory system in 40%, and of the respiratory system in 24% of the cases [Garshick et al., 2005]. Unfortunately, suicide is also reported to be a common cause of death in the SCI population [Garshick et al., 2005; Soden et al., 2000], interestingly mainly among the least disabled persons with SCI [Hartkopp et al., 1997]. However, the leading cause of death may vary considerably in people with paraplegia or tetraplegia.
Despite substantial progress in medical management during the past decades patients with SCI are still at greater risk for medical complications compared to the general population, which often leads to rehospitalisations [Cardenas et al., 2004]. Thereby, hospitalisation rate highly correlates to the level and completeness of the neurological impairment, which implies that patients with a complete and/or cervical lesion are concerned most [DeVivo, 2007; Middleton et al., 2004], especially during the first year after SCI [Cardenas et al., 2004; Pagliacci et al., 2007]. The list of known complications in patients with SCI is long and includes for example urinary tract infections [Levi et al., 1995], pneumonia [Fishburn et al., 1990], pressure sores [Levi et al., 1995], spasticity [Skold et al., 1999], pain [Ullrich, 2007], osteoporosis [Frotzler et al., 2008a], disturbed thermoregulation [Khan et al., 2007], deep vein thrombosis [Riklin et al., 2003] and autonomic dysfunction [Garstang and Miller-Smith, 2007]. Some of these specific SCI-related complications - obviously leading to prolonged rehospitalisation times - will be discussed in more detail below (see Chapter 1.2).

1.2 Complications in spinal cord injury

A spinal cord lesion results in an impairment or loss of motor, sensory and vegetative functions. After the occurrence of a SCI muscles below the lesion are paralysed, sensation for pain and temperature, proprioception and sense of touch are lost, gastrointestinal, urologic and sexual dysfunctions are common. Thereby, level and completeness of SCI determine to which extent such complications occur. Below, some more comprehensive information will be given, concerning some of the most frequent complications persons with a SCI are faced with.

Urological complications
The main functions of the bladder are simple, namely urine storage and micturition. This tasks are based on different autonomic and non-autonomic steering mechanisms. A SCI induces a loss of sensory and motor control of the bladder function - the so called neurogenic bladder - which often leads to incontinence and damages of the urinary tract [Zäch and Koch, 2006d]. In this context, urinary tract infections are very common in subjects suffering from a SCI [Trautner and Darouiche, 2002]. This situation is very unpleasant for the patients affected and negatively influences their quality of life. Thus, a well directed, individual bladder management for individuals with SCI is necessary to avoid or at least minimize undesirable side effects of the neurogenic bladder.

Gastrointestinal complications
The main tasks of the gastrointestinal tract include resorption, secretion, peristalsis, defecation and continence. These processes are mainly influenced by the autonomic nervous system. A SCI often results in a disruption of the neuronal innervation of the gastrointestinal tract leading to several gastrointestinal complications. Kirk et al. [1997] for example reported that 76% of a group of patients with SCI complained about gastrointestinal symptoms, whereas Stone and colleagues [1990] reported the occurrence of hemorrhoids in
74%, abdominal distention in 43%, autonomic hyperreflexia in 43% and problems with defecation in 20% of subjects with SCI. A chronic gastrointestinal problem was found in 27% of the patients investigated. It seems obvious that such complications decrease not only quality of life in the subjects concerned but also lead to a high number of rehospitalisations. Thus, arrangements in the sense of a neurogenic bowel management aim to minimise the above mentioned complications and should allow a regular, sufficient and temporally limited defecation for patients with SCI [Zäch and Koch, 2006a].

Autonomic dysreflexia
Autonomic dysreflexia is an acute syndrome of excessive, uncontrolled sympathetic output caused by spinal reflex mechanisms. In general, subjects with SCI at or above the sixth thoracic neurologic level are affected [Blackmer, 2003]. Typical symptoms include high blood pressure, headache, flushing, sweating in the head and neck region, mydriasis and nasal congestion. Autonomic dysreflexia is caused by a noxious stimulus (e.g. full bladder or abdominal distension) below the lesion level. This stimulus produces an afferent impulse leading to a generalized sympathetic response and as a consequence to a vasoconstriction below the neurologic lesion [Karlsson et al., 1998]. The vasoconstriction is responsible for a rapid increase in blood pressure (up to 300mmHg for the systolic and 220mmHg for diastolic pressure [Karlsson, 1999]) causing the above mentioned symptoms. With a damaged spinal cord a descending inhibitory parasympathetic response via the central pathways to modulate the rise in blood pressure is impossible, which finally results in an excessive parasympathetic output and peripheral vasodilatation [Blackmer, 2003]. Autonomic dysreflexia is a severe complication in subjects with a high spinal cord lesion and can lead to life-threatening situations.

Musculoskeletal complications
Adaptations after SCI include a loss of muscle mass [Lotta et al., 1991; Spungen et al., 2000] in the paralyzed limbs. Olive and colleagues [2003] found a 38% reduction of muscle volume in the paralysed legs 10 years after the occurrence of a traumatic SCI. At the same time the diameter of the femoral artery was concomitantly reduced by 36% [Olive et al., 2003] These adaptations lead to a reduced blood flow and blood redistribution compared to able-bodied subjects [Theisen et al., 2001]. Moreover, a shift towards type-II muscle fibres in the legs can be found in people with chronic paraplegia [Burnham et al., 1997].

A consequence of sublesional muscle loss and paralysis is reduced mechanical loading on bones [Rittweger et al., 2006] resulting in a distinct bone loss in subjects with SCI termed “immobilization” or “disuse” osteoporosis [Ubelhart et al., 1995]. Compared to able-bodied controls sublesional bone mineral density decreases on average by 41% within the first year after SCI. The consequence of the lowered bone mass and microarchitectural deterioration of bone tissue is an enhanced bone fragility with a concomitant fracture risk [WHO technical report series, 1994] causing severe secondary complications such as pressure sores and infections [Freehafer, 1995; Ragnarsson and Sell, 1981] in subjects with SCI.
Pressure sores
Immobilisation and the loss of sensitivity in the paralysed limbs seem to be the main reasons for the occurrence of pressure sores in patients with SCI. The incidence was reported to be between 13% and 40%, whereas persons with a complete tetraplegia showed the highest and subjects with an incomplete paraplegia the lowest frequency of occurrence [Young et al., 1982]. Thus, pressure sores are one of the most common reasons for rehospitalisations in the spinal cord injured population [Cardenas and Hoffmann, 2004; Savic et al., 2000], provoking prolonged hospitalization and rehabilitation times, increased health care costs and decreased quality of life of patients affected by this complication.

Shoulder pain
Compared to able-bodied persons the upper extremities of wheelchair users are excessively involved in tasks of daily living, such as propulsion of the wheelchair and transferring from the wheelchair e.g. into a car. Thus, it seems not surprising that overuse symptoms of the upper extremities, in particular of the shoulder, often occur in this population [Sie et al., 1992; Silfverskiold and Waters, 1991]. In fact, Sie et al. [1992] found up to 64% of subjects with paraplegia complaining about pain in the upper extremities, mainly in the shoulder. However, the occurrence of shoulder pain may highly impact quality of life and result in long-lasting therapies or high surgery costs.

Spasticity
According to Lance [1980] spasticity is defined as a velocity-dependent increase in muscle tone to passive stretching, leading to an intermittent or continuous involuntary activation of muscles [Pandyan et al., 2005]. Spasticity is a typical phenomenon, which occurs in patients with a lesion of the upper motor neuron. Spasticity is often related to a decreased quality of life in patients with SCI as motor activity can be hindered, pain often occurs and digestion, circulation as well as normal breathing are negatively affected. The later can cause severe problems, especially in subjects with tetraplegia [Zäch and Koch, 2006b]. However, one has to keep in mind, that spasticity may also have some positive effects such as an increased venous return, a decelerated muscle atrophy [Zäch and Koch, 2006b] or a positive impact on bone mineral density in the paralysed limbs [Demirel et al., 1998; Eser et al., 2005]. Thus, the medical an paramedical personnel has to take into consideration the advantages and disadvantages of spasticity while treating and nursing a patient with SCI.

Respiratory complications
Based on a lesion-dependent loss of respiratory muscle innervation, respiratory muscle function is decreased in individuals with SCI. The consequences of this impairment are reduced respiratory muscle volumes and strength [Sipski and Richards, 2006] causing complications such as dyspnoea, pneumonia or sleep-disordered breathing [Brown et al., 2006]. Respiratory complications are still one of the leading causes of death (approximately 25%) in persons with SCI [van den Berg et al., 2010]. Thereby, a higher neurological level and completeness of SCI is associated with an increased number of pulmonary infections [Haisma et al., 2007] and consequently a higer mortality risk. In general, respiratory complications imply prolonged hospitalization times [Winslow et al., 2002] and a considerable reduction in quality of life [Tator et al., 1993]. Thus, the goal of a respiratory management
and prevention program in patients with SCI should be to reach and preserve the highest possible function of the respiratory pump.

Thermoregulatory dysfunction
The impairment of the autonomic and somatic nervous system in subjects with SCI leads to a decreased thermoregulatory capacity compared to able-bodied individuals [Bhambhani, 2002]. This means that skin blood flow and the ability to sweat below the lesion is disturbed [Holme et al., 2001; Sawka et al., 1989], whereas individuals with higher lesion levels and a complete lesion are affected most [Petrofsky, 1992; Sawaka et al., 1989]. The consequence of the lower heat tolerance of individuals with SCI is a decreased exercise performance compared to able-bodied subjects, especially when exercising under hot environmental conditions [Bhambhani, 2002].

1.3 Consequences for exercise performance

Exercising on a regular base is very important for individuals with a SCI to prevent from risk factors such as cardio-vascular diseases [Franklin et al., 2003], to decrease depression [Dunn et al., 2005], pain and stress [Ditor et al., 2003] as well as to increase quality of life [Ditor et al., 2003; Martin Ginis et al., 2010; Stevens et al., 2008]. In this context, it seems clear that some of the above mentioned complications (e.g. pressure sores, shoulder pain) make exercise almost impossible or at least difficult, whereas others (e.g. thermoregulatory dysfunction, reduced respiratory muscle function) may negatively influence exercise performance of wheelchair users compared to able-bodied individuals. Thereby completeness and the level of the lesion play an important role to which extent exercise performance is affected. Especially in persons with a lesion level above Th6 the cardiovascular response to exercise is often limited due to the disruption of the sympathetic nervous system [Schmid et al., 1996]. Blood pressure, peak heart rate, stroke volume, cardiac output and peak oxygen uptake are diminished [Bhambhani, 2002]. In contrast, individuals with a low lesion paraplegia show similar cardio-vascular adaptations of heart rate and blood pressure during exercise compared to able-bodied persons, whereas stroke volume is reduced due to the reduced muscle pump action in the lower extremities leading to a venous blood pooling [Bhambhani, 2002; Jacobs et al., 2002]. Nevertheless, it is amazing that highly trained wheelchair athletes are able to reach peak oxygen uptakes of over 50ml/min/kg [Schmid, 2002] corresponding to values of endurance trained able-bodied subjects.

Beside cardio-vascular adaptations a SCI also affects respiratory muscle function [Sipski and Richards, 2006], which not only leads to the above mentioned complications but also may limit exercise performance. Thereby, respiratory volumes and strength are dependent on lesion level and completeness [Almenoff et al., 1995], making subjects with a complete tetraplegia affected most. In some patients exercise performance may also be reduced due to severe spasticity as this may limit physiologically correct respiratory manoeuvres leading to a reduction in oxygen uptake [Zäch and Koch, 2006b].
The decreased thermoregulatory capacity as a further exercise limiting factor in persons with SCI during exercise [Bhambhani, 2002] has to be taken into account as well, especially under extreme hot or cold environmental conditions. Thus, to wear warm clothes during winter sports to prevent from frostbite as well as to implement adequate cooling methods to avoid heat stroke and to optimize exercise performance in a hot environment [Hagobian et al., 2004] are strongly recommended.

Further, in view of complications such as urinary tract infections, incontinence, spasticity, gastrointestinal problems or pain one has to keep in mind that individuals with a SCI are often treated with medication. Some of these substances might influence e.g. thermoregulation or exercise performance in general. Especially in the field of elite sports, one has to be aware of the fact, that some substances might be banned as specified on the doping list. Additionally, some athletes use the phenomenon of the autonomic dysreflexia to enhance exercise performance by filling their bladder. This method is called “Boosting” and may increase exercise performance up to 10% [Bhambhani, 2002]. However, the rapid and massive increase in blood pressure [Karlsson, 1999] is dangerous and can cause life-threatening situations. Thus, such unfair and dangerous practices are forbidden according to the regulations of the International Paralympic Committee [IPC, 2000].

In summary, the specific complications related to a SCI demand special attention when exercising with this special population. For example, in a subject with complete tetraplegia, training guidance often has to be done based on rating of perceived exertion or lactate concentrations as heart rate is not a reliable parameter due to the missing sympathetic innervation of the heart [Goosey-Tolfrey, 2010]. Additionally respiratory, thermoregulatory and medical aspects have to be taken into account as well. However, in general, also subjects with a SCI are trainable and exercise performance can be determined based on standardized exercise tests. Although several limitations and restrictions exist, physical activity on a regular base plays a key role in reducing health-related risk factors and complications in this population [Mohr et al., 1997a].

Thus, the present work deals with the issue “exercise in persons with SCI” and aims to show possibilities of exercises testing, implementation of new training methods into daily clinical practice as well as methods to optimize exercise performance of patients and athletes suffering from a SCI. Whereas Chapter 2 focuses on “exercise testing”, the content of Chapter 3 refers to “respiratory muscle training”. Chapter 4 relates to “functional electrical stimulated cycling” and Chapter 5 finally deals with the topic “nutrition in persons with SCI” in conjunction with exercise. Hopefully, practical application of the following study results will finally be a little contribution towards a better quality of life in patients with SCI and may offer some possibilities to athletes with SCI to compete at their highest performance level possible.
2 Exercise testing in persons with spinal cord injury

A recently published meta-analysis [Martin Ginis et al., 2010] showed a significant positive relationship between physical activity and subjective well-being in individuals with a spinal cord injury (SCI). Thus, regular exercise seems to play a key role in reaching and preserve a high quality of life, especially for persons with disabilities such as SCI who tend to report lower ratings for their quality of life compared to able-bodied subjects [Dijkers, 1997]. In order to objectively determine the actual fitness level of a subject with SCI and to guide or optimize the training process, exercise testing seems to be a helpful tool. In view of the above mentioned important role of regular physical activity for subjective well-being in persons with SCI, a regular examination of the actual fitness level seems to be of importance not only for athletes but even more for patients. However, to generate adequate training recommendations valid and reliable testing methods and concepts are needed. Further, the special physiological characteristics of persons with a SCI (for details refer to Chapter 1 above) should be considered. As a consequence, the development of new sport-specific testing methods or adaptations of already existing exercise tests to meet the requirements of the population with SCI are necessary and the only way to optimally support patients and athletes during their training process. Additionally, the application of valid measurement equipment is a prerequisite to achieve reliable data and new devices should be critically evaluated before a routinely use in daily clinical practice is recommended.

The first study of this chapter aimed to assess the accuracy of a new portable ergospirometric device [Perret and Mueller, 2006]. The following studies dealt with the development of simple [Mueller et al., 2004] and more sophisticated [Strupler et al., 2009] new exercise testing methods and its implementation into wheelchair sports [Perret et al., 2012]. The final study presented in this chapter compared the blood lactate elimination kinetics after exhaustive upper body exercise between able-bodied persons and subjects with SCI [Leicht and Perret, 2008]. Such knowledge is of importance to further optimize the training process of subjects with SCI, especially in the field of Paralympic elite sports.
### 2.1 Validation of a new portable ergospirometric device (Oxycon Mobile®) during exercise

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**Introduction**

Today automated metabolic gas analysis systems are commonly used to determine physical fitness in athletes and patients under laboratory conditions [Macfarlane, 2001]. For sports which are impossible to effectively simulate in a laboratory setting [Smekal et al., 2000] it may be advantageous to measure gas exchange and ventilatory variables during a sport specific field test. For this purpose, powerful and lightweight portable metabolic systems are required. In the past, several studies with different devices were conducted [Crandall et al., 1994; Hausswirth et al., 1997; King et al., 1999; Lothian et al., 1993; Lucia et al., 1993; McLaughlin et al., 2001; Melanson et al., 1996; Meyer et al., 2001; Novitsky et al., 1995; Schulz et al., 1997] with some limitations to the accuracy of the measured data compared to laboratory-based systems or the Douglas bag method [Macfarlane, 2001].

The aim of the present study was to test the accuracy of a new portable spirometric-telemetric device (Oxycon Mobile®) over a wide range of exercise intensities against a stationary apparatus (Oxycon Pro®) which was validated some years ago against the Douglas bag method as a gold standard [Rietjens et al., 2001]. That study reported that the OP was a fast and accurate system for measurements of metabolic parameters during low- and high-intensity exercise [Rietjens et al., 2001]. Thus, we were confident that the OP is a reliable reference system and a helpful tool to determine physical fitness in athletes and patients under laboratory conditions.

To avoid possible negative effects due to athletes’ nutrition, training, test preparation or day-to-day variability, in contrast to other studies [Crandall et al., 1994; Hausswirth et al., 1997; Lothian et al., 1993; Lucia et al., 1993; McLaughlin et al., 2001; Melanson et al., 1996; Meyer et al., 2001; Schulz et al., 1997], we designed a special face mask which allowed the simultaneous measurement of respiratory variables by both devices.
Materials and Methods

Subjects
Fifteen male endurance trained subjects (VO₂peak: 58.8±5.2 ml·min⁻¹·kg⁻¹) participated in the study. Their average age was 35.1±7.4 years, height 180.9±6.6 cm, and weight 77.5±7.9 kg. The study was approved by the local ethical committee. Written informed consent of the subjects was obtained prior to the start of the study.

Equipment

Oxycon Pro®
The Oxycon Pro® (OP) (Jäger, Würzburg, Germany) is a stationary ergospirometer and consists of a transducer holder with a turbine inside attached to a face mask. The rotation of this turbine is detected optoelectrically and allows determination of minute ventilation. Expired air is analyzed for oxygen and carbon dioxide concentrations via a sampling line connected to the transducer holder. Oxygen and carbon dioxide concentrations are measured by a paramagnetic and an infrared absorption analyzer, respectively. All data measured are recorded and monitored on a personal computer.

Oxycon Mobile®
The Oxycon Mobile® (OM) (Jäger, Würzburg, Germany) is a new, light-weight (950 g) portable spirometric device based in general terms on the same technology as the OP. In contrast to the OP, the OM measures the oxygen concentration through an electrochemical sensor and data are transmitted telemetrically and recorded on a personal computer. During exercise, the battery operated OM can be comfortably strapped to the chest or the back of a subject and allows continuous data sampling for up to four hours.

To control for the possible effects of day-to-day variability in exercise performance and to allow for the simultaneous measurement of metabolic variables by the two devices, the transducer holder of the OM was modified by creating an additional hole for the sampling line of the OP. The modification of the OM transducer holder allowed simultaneous gas sampling by the OM and OP and allowed both systems to measure respiratory volumes by the same turbine.

Warm up time of both devices was at least 60 min. Immediately before the start of an exercise test (see below), the turbine was calibrated by a 3-l syringe (Hans Rudolph Inc., Kansas City, US) and the gas analyzers were calibrated with the same certified calibration gas mixture of 5% CO₂, 16% O₂ and 79% N₂ (Pangas, Dagmersellen, Switzerland). After the calibration procedure, subjects were equipped and connected to the OM and the OP before testing was started with both devices at exactly the same time. Gas exchange and ventilatory variables were measured breath by breath and averaged over 15 s for data analysis.

Experimental procedure
Subjects completed two exercise tests on an electromagnetically braked cycle ergometer (Ergometrics 900 S, Ergoline, Bitz, Germany) on two different occasions. The first test was an incremental exercise test (IET). After a resting period of 5 min, subjects started cycling at 100W. Every 3 min the load was increased by 50W until volitional exhaustion. Maximal
power output was defined as the highest workload subjects were able to sustain for the whole 3 min exercise stage.
The endurance test (EET) consisted of a 5 min resting period, followed by 15 min continuous cycling at 100W, 150W and 200W, respectively. This test was performed to control the differences (e.g. drift) of data measured between the OM and the OP during submaximal, steady state exercise over a longer time period.

During the resting periods and the cycling tests, gas exchange variables (VO\textsubscript{2}, VCO\textsubscript{2}, RER) were measured continuously breath by breath in parallel by the two different ergospirometric devices.

Statistics
The mean of the 15 s values from VO\textsubscript{2}, VCO\textsubscript{2} and RER over the last 2 min of each exercise stage during the IET and the last 5 min of each stage during the EET were calculated at rest and for each power output. Comparisons between the OM and the OP variables of gas exchange at rest and for each power output were made using ANOVA for repeated measures. Where a significant main effect was observed, a Bonferoni post-hoc test was used to locate the significant differences. Values were considered to be significantly different if \( p < 0.05 \).

In order to show the difference between the two devices, Bland-Altman plots were used over the complete range of measured variables [Bland and Altman, 1986]. These data were presented graphically, comparing the difference between the devices (bias) versus their average value.

Results

Compared to the OP, the OM showed significantly lower values for VO\textsubscript{2} at 200W (2680±155ml·min\textsuperscript{–1} vs. 2800±156ml·min\textsuperscript{–1}) and 250W (3228±167ml·min\textsuperscript{–1} vs. 3363±173ml·min\textsuperscript{–1}) during the IET, whereas at 300W (3767±233ml·min\textsuperscript{–1} vs. 3923±220ml·min\textsuperscript{–1}) only a tendency (\( p = 0.07 \)) for significance was seen (Fig. 1a). During the EET no significant differences for VO\textsubscript{2} were found (Fig.1b).

Further analyses of agreement between the two devices in measuring gas exchange variables were accomplished according to the method described by Bland and Altman [1986]. The mean difference (bias) between the two methods against their mean and the limits of agreement (mean ± 2 SD of the differences) are graphically presented as in Fig. 1c for the IET and in Fig. 1d for the EET for VO\textsubscript{2} over the complete range of values measured.

The bias was –110±127ml·min\textsuperscript{–1} for the IET (Fig. 1c) and –55±67ml·min\textsuperscript{–1} for the EET (Fig. 1d). For VCO\textsubscript{2} no significant differences were found in either the IET (Fig. 2a) or the EET (Fig. 2b) although the OM seemed to systematically measure slightly higher values for VCO\textsubscript{2} compared to the OP. Therefore, corresponding Bland-Altman plots for VCO\textsubscript{2} showed a positive bias of 43±57ml·min\textsuperscript{–1} and 20±74ml·min\textsuperscript{–1} for the IET (Fig. 2c) and the EET (Fig. 2d), respectively.
Figures 1a to 1d: Absolute VO\(_2\) measured by Oxycon Mobile (OM) and Oxycon Pro (OP) during the incremental (IET) (a) and the endurance exercise test (EET) (b) at different power outputs. Corresponding Bland-Altman plots showing relationship between mean measured values for VO\(_2\) and the difference measured by OM and OP during IET(c) and EET(d). * p < 0.05.

With regard to RER for all workloads during the IET (Fig. 3a) and during the EET (Fig. 3b), a significant overestimation of RER by the OM was found, which is also reflected in the corresponding Bland-Altman plots with a bias of 0.05 ± 0.03 for the IET (Fig. 3c) and a bias of 0.04 ± 0.03 for EET (Fig. 3d).

**Discussion**

The main finding of the study showed that VO\(_2\) was significantly underestimated at high power outputs and RER significantly overestimated at all workloads tested by the OM compared to the OP. The VCO\(_2\) measured by the OM showed constant but not significantly higher values.

Previous studies revealed inconsistent results comparing gas exchange variables measured by different portable units against reference systems. Some investigators found significantly higher [McLaughlin et al., 2001; Wideman et al., 1996] or lower [Beneke et al., 1995; Lothian et al., 1993; Peel and Utsey, 1993] values for VO\(_2\) whereas others showed no significant differences [Hausswirth et al., 1997; Lucia et al., 1993; Schulz et al., 1997]. While the OP
and OM devices were allowed to warm up for 60 min and then calibrated immediately prior to each exercise test using the same calibration gas; these data found that the VO$_2$ values measured by the OM were significantly lower than the OP at higher workloads during the IET (Fig. 1a). These differences were possibly due to the different oxygen sensors used in the two tested devices (electrochemical in the OM vs. paramagnetic in the OP).

Figures 2a to 2d: Absolute VCO$_2$ measured by Oxycon Mobile (OM) and Oxycon Pro (OP) during the incremental (IET) (a) and the endurance exercise test (EET) (b) at different power outputs. Corresponding Bland-Altman plots showing relationship between mean measured values for VCO$_2$ and the difference measured by OM and OP during IET (c) and EET (d).

Older portable systems used in previous studies (e.g. [Crandall et al., 1994; Lucia et al., 1993; Peel and Utsey, 1993]) were not able to measure VCO$_2$. Studies with more sophisticated devices showed inconsistent results [Hausswirth et al., 1997; King et al., 1999; Wideman et al., 1996]. The data for VCO$_2$ measured by the OM were slightly but not significantly higher at all workloads (Fig. 2a and b).

As a consequence of the lower VO$_2$ and these slightly but not significantly higher VCO$_2$ values in our study, RER was significantly overestimated by the OM compared to the reference system (Fig. 3a and b). Similar results for RER were also found in other studies [King et al., 1999; Wideman et al., 1996].
Figures 3a to 3d: Respiratory exchange ratio (RER) measured by Oxycon Mobile (OM) and Oxycon Pro (OP) during the incremental (IET) (a) and the endurance exercise test (EET) (b) at different power outputs. Corresponding Bland-Altman plots showing relationship between mean measured values for RER and the difference measured by OM and OP during IET (c) and EET (d). * p < 0.05; ** p < 0.01; *** p < 0.001.

Validity analysis suggested by Bland and Altman [1986] showed a relatively large bias with fairly high standard deviations, especially for the measurements of RER (Fig. 3c and d). We therefore dissuade from comparing gas exchange variables collected by the OM with data measured under laboratory conditions by an accurate reference system such as the OP. Furthermore, the extrapolation of information about substrate utilization during different exercise durations and intensities from data measured by the OM seems inappropriate.

Conclusions

Compared to the OP as a reference system, the OM significantly underestimates VO\textsubscript{2} at high workloads above 200W and overestimates RER at all workloads tested during incremental and steady state endurance exercise. This must be considered if data measured by the OM is used for comparison of test results or metabolic calculations like energy costs of exercise.
2.2 A new test to improve the training quality of wheelchair racing athletes

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Introduction

Exercise testing can be a helpful instrument to prescribe and control training. Indeed, in order to obtain reliable test results, sport-specific testing methods are needed [Bhambani, 2002] especially for athletes with physical disabilities. Furthermore, in order to assess training progress, high test–retest reliability is essential. This reliability seems to be most pregnant at racing speed over racing distance [Schabort et al., 1998b]. Nevertheless, an increased sensitivity of awareness, also at lower intensity levels, is fundamental for competitive athletes in their daily training routine. This allows an improved guidance of the athlete’s training by the coach, because the athlete can differentiate better between intensities. Several studies showed that exercise testing based on rate of perceived exertion (RPE), is valid and useful for exercise prescription and regulation of exercise intensity [Ceci and Hassmen, 1991; Okura and Tanaka, 2001], especially for moderate levels [Dunbar et al., 1992] and adolescents [Perez-Landaluce et al., 2002]. Schabort et al. [1998a] obtained more reliable results for tests with self-chosen intensities in regard to tests performed at fixed workloads. Trustworthy results have also been found for set distances performed in the shortest time possible [Schabort et al., 1998a]. Goosey et al. [2000] reported that self-chosen stroke frequencies are the most economical. All these facts show that a precise awareness of the different intensity levels is important in competition and training in order to economize the motor function and thereby increase the performance level. We are aware of the fact that for scientific publications, it is much easier to calculate the reproducibility of clearly defined and measurable data. On the other hand, for practical use, other factors, such as a precise awareness, are equally important and have to be considered as well.

In our opinion, the ability to choose a certain training intensity level consciously is one of the most important skills needed in order to become a topclass athlete. In this way training quality can be improved, allowing performance to increase. Additionally, we believe that a well-developed coenaesthesia helps the athlete to prevent from injury. A reduced injury rate results in less training drop-outs, leading in its way to an improved training quality. The objective of the current study was to design a new test, based on the awareness of usual training intensities, which has in its way a high relevance to daily training practice. Therefore, a new test for wheelchair racing athletes was designed, at which the reproducibility of different test parameters at habitual, but subjectively chosen intensity bouts was tested.
Subjects and methods

Subjects
We tested 11 competitive wheelchair racing athletes. Detailed information of their anthropometrical data, impairment and training volume is given in Table 1. The local ethics committee approved the study. All 11 participants, as well as their parents (in case subjects were under 18 years of age before the start of the study), were informed about the procedures and gave written consent before participation.

Methods
Within a period of 3±1 days, every athlete completed two identical tests in their own racing chair on a nearly frictionless training roller (Spinner, New Hall’s Wheels, Cambridge, USA). The tests consisted of five 1500m bouts, at subjectively chosen, habitual training intensities, with a rest of 2 min between each bout. These training intensities were defined as 1=warm up/cool down, 2=extensive aerobic training, 3=intensive aerobic training, 4=training in the area of the anaerobic threshold, 5=race intensity. As in every sports discipline, athletes and coaches use a certain scale and description for their different intensity levels. Even if these levels were somehow defined by words, they are not necessarily the same for everyone. Our goal was to assess the reproducibility of these intensity levels, in order to get a measure on how exactly these athletes were able to implement training guidelines. They were told to maintain subjectively a constant velocity during every bout, after which they were asked for RPE by means of a Borg-Scale ranging from 6 to 20 [Borg, 1982]. The athletes were blinded to any of the collected data during the whole test, since we wanted them to perform only in reference to their own awareness. The only information they got throughout the test were verbal indications of the 500 and 1000m markers and the end of each bout. Verbal encouragement was only given during the last 200m of bout 5, because it has been proven that frequent encouragement leads to a significantly higher maximum effort [Andreacci et al., 2002]. We measured the time to complete the 1500m (in min) using a stopwatch. Average speed (km·h⁻¹) was measured by a speedometer (CicloMaster, Hochschorner GmbH, Krailling, Deutschland), which was calibrated and mounted on the training roller. Stroke frequency (min⁻¹) was measured by a hand counter, on which each single stroke of the whole test (on each bout) was registered. Mean stroke frequency was calculated afterwards. Heart rate (HR) (bpm) was registered every 5s during the whole test time by means of an HR monitor (Polar Vantage NV, Polar Electro, Kempele, Finland). Mean HR values of the last minute of each bout were calculated. Capillary blood samples to measure lactic acid concentrations (mmol·l⁻¹), were collected from the earlobe before the test, immediately at the end of each bout, as well as at 2, 4 and 6min after the end of bout 5. Blood samples were analysed enzymatically by a lactate analyser (Super GL Ambulance, Ruhrtal Labor Technik, Möhnsee, Germany). All athletes performed no or only low intensity exercise the day before each test and recorded sleep, food, beverages, supplements and medication during the 24h before both tests. Participants were asked to replicate test preparations for the second test. Nutrition and training protocols were checked before both tests by a questionnaire. At the second test, preparation was compared with the 24h before the first test. If test preparations were not strictly adhered to, the test was cancelled. Additionally, all athletes were told to refrain from caffeine intake on both test days, because caffeine intake may increase exercise performance [Spriet, 1995].
Table 1: Anthropometric data, impairment and training information

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age [y]</th>
<th>Height [cm]</th>
<th>Weight [kg]</th>
<th>Lesion level</th>
<th>ASIA / Impairment</th>
<th>Impairment since [y]</th>
<th>Training [h-week⁻¹]</th>
<th>Training since [y]</th>
</tr>
</thead>
<tbody>
<tr>
<td>m</td>
<td>26</td>
<td>153</td>
<td>52</td>
<td>L3</td>
<td>C</td>
<td>26</td>
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</tr>
<tr>
<td>m</td>
<td>17</td>
<td>172</td>
<td>58</td>
<td>Th12</td>
<td>Spina bifida</td>
<td>17</td>
<td>12</td>
<td>5</td>
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<tr>
<td>m</td>
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<td>140</td>
<td>50</td>
<td>L3</td>
<td>A</td>
<td>15</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>f</td>
<td>13</td>
<td>138</td>
<td>38</td>
<td>Unknown</td>
<td>Spina bifida</td>
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<td>6</td>
<td>3</td>
</tr>
<tr>
<td>f</td>
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<td>170</td>
<td>46</td>
<td>L1</td>
<td>B</td>
<td>3</td>
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<td>1</td>
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<tr>
<td>m</td>
<td>23</td>
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<td>L1</td>
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<td>8</td>
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<tr>
<td>m</td>
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<td>180</td>
<td>60</td>
<td>-</td>
<td>Cerebral paresis</td>
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<td>3</td>
<td>2</td>
</tr>
<tr>
<td>f</td>
<td>36</td>
<td>162</td>
<td>47</td>
<td>-</td>
<td>Right leg amputee</td>
<td>17</td>
<td>10</td>
<td>3</td>
</tr>
<tr>
<td>m</td>
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<td>191</td>
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</tr>
<tr>
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<td>L4</td>
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</table>

Mean±SD 20.5 ± 6.4  164.7 ± 16.3  54.1 ± 9.2  14.0 ± 7.9  6.8 ± 2.7  3.9 ± 2.8

SD: standard deviation; m: male; f: female; L: lumbar; Th: thoracic; ASIA: American Spinal Injury Association
Statistical analysis
In order to get a measure on how reproducible the subjectively chosen intensity levels (1–5) were, we calculated the root-mean-squared coefficients of variation (CV) of the overall time (for 1500m), average speed, stroke frequency, HR, RPE and the concentration of lactic acid on every bout, according to the duplicate measurement method described by Glüer et al. [1995].

Results
Mean CVs (%) of the overall time (for 1500m), average speed, stroke frequency, HR, RPE and concentration of lactic acid of bouts 1–5 are shown in Table 2. CVs were smallest for the HR and stroke frequency parameters and for values of bout 5. This newly designed wheelchair test resulted in CVs ≤9.5% for nearly all parameters. Exceptions were the lactic acid parameters on all bouts, the average speed together with the RPE on the two lowest intensity bouts and the time of bout 2.

Table 2: Coefficients of variation (%)

<table>
<thead>
<tr>
<th></th>
<th>Bout 1</th>
<th>Bout 2</th>
<th>Bout 3</th>
<th>Bout 4</th>
<th>Bout 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time for 1500m</td>
<td>9.4</td>
<td>10.7</td>
<td>9.5</td>
<td>9.5</td>
<td>2.8</td>
</tr>
<tr>
<td>Average speed</td>
<td>12.0</td>
<td>10.9</td>
<td>8.5</td>
<td>7.8</td>
<td>2.6</td>
</tr>
<tr>
<td>RPE</td>
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<td>10.7</td>
<td>6.9</td>
<td>6.0</td>
<td>3.5</td>
</tr>
<tr>
<td>Heart rate</td>
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<td>6.4</td>
<td>6.3</td>
<td>6.1</td>
<td>3.1</td>
</tr>
<tr>
<td>Lactic acid</td>
<td>20.6</td>
<td>16.8</td>
<td>25.2</td>
<td>29.7</td>
<td>23.2</td>
</tr>
<tr>
<td>Stroke frequency</td>
<td>6.7</td>
<td>7.7</td>
<td>7.2</td>
<td>6.5</td>
<td>7.9</td>
</tr>
</tbody>
</table>

RPE: rate of perceived exertion

The absolute values of the different subjects showed large variations to one another due to a large range of age and training practice of the tested athletes. However, this does not affect the calculation of the CVs, since all of the subjects’ values were only compared to themselves. Means±SD of test 1 and test 2 of bouts 1–5 are shown for the overall time (for 1500m) (Figure 1), RPE (Figure 2), HR (Figure 3) and stroke frequency (Figure 4).
Figure 1: Mean±SD for the time to complete 1500m at five different exercise intensities on two separate test days

Figure 2: Mean±SD for the rate of perceived exertion at five different exercise intensities on two separate test days
Figure 3: Mean±SD for the heart rate at five different exercise intensities on two separate test days

Figure 4: Mean±SD for the average stroke frequency at five different exercise intensities on two separate test days
Discussion

According to Devillard et al. [2001] exercise tests with wheelchair ergometers that deliver parameters with CVs <9.5% are suitable for practical use. As shown in Table 2, our results comply with this recommendation, in particular the high reproducibility of the upper intensity bouts. Measurement of lactic acid concentrations showed a too low reproducibility to be of any practical use.

Levels of suitable CVs for a test validation depend on many different factors. We therefore recommend considering sports discipline, level, age and number of subjects before assessing CV values. Even though we only had 11 subjects of rather different proficiency levels, most CVs are in the above-mentioned range. If the test parameters above are used for monitoring training progress and to improve awareness of exercise intensity (which cannot be measured or defined exactly), the reproducibility of our tested parameters is quite high. We therefore conclude that our test is valid for wheelchair racing athletes, in particular for inexperienced athletes. In this last group, potential training progress is still bigger than in elite athletes. At the same time, this progress is large enough to be detected by our testing procedure.

We realize that standardizing and testing of a certain sensation is rather difficult and also depends on a lot of individual factors. It has been reported that the psychological factors may also play an important role during testing [Hickey et al., 1992; Jeukendrup et al., 1996]. There are no conclusive data available on the physiological and psychological contribution to variability in athletic performance [Kuipers et al., 1985]. We therefore recommend the best possible standardized conditions before and during testing. This is one of the most important requirements in order to get reliable performance test results, even if the tested intensities are based on personal feelings. In this study, we standardized test conditions, nutrition and test preparations as described under Methods.

It should furthermore be noted that even in trained athletes [Hickey et al., 1992], learning effects have been observed in many exercise tests, especially between the first and the second trial [Hutzler et al., 2000]. In order to measure the training progress and not learning effects, we recommend performing an initial ‘learning trial’ test. In this way, one can learn in which amount the intensity has to be increased between the different bouts and one can get familiarized with the testing conditions. The second test will then deliver a more realistic basic level, which can later be compared with further tests, in order to detect a training progress.

As shown in Table 2, a rather high reproducibility resulted from our tests, even without a learning trial. Nevertheless, in order to achieve an even better reproducibility, a learning trial would be an additional benefit, even more so for young athletes. For a meaningful training control, we consequently recommend a learning trial before using this test in practice.

Eston and Williams [1988] reported that RPE ratings are a useful tool with high reproducibility for high intensity exercise, and only a small amount of practice with the scale is needed for low intensity levels. This finding confirms our CVs found for RPE ratings, which range from 14.4% on bout 1 to 3.5% on bout 5 (Table 2). Furthermore, it has been reported that RPE ratings are also related to goal orientation, especially in young athletes between 11 and 15
years [Stephens et al., 2000]. Since we tested two subjects of this particular age group, this could also have extended our range of CVs for RPE, even more so at the low-intensity bouts.

In young subjects, a close relationship of RPE, HR and relative exercise intensity has been noted [Eston and Williams, 1986]. HR seems to be a reliable test parameter, even when athletes choose their own intensity, blinded to time, HR, velocity and stroke frequency. Our CVs of HR are nearly constant around 6% from bout 1 to 4 and with 3.1% during bout 5, even lower. Similar results were also shown by other groups [Bhambani et al., 1991; Washburn and Montoye, 1985] who reported increased HR reliability with increased power output. Concerning HR, one has to pay special attention to athletes with lesion levels above Th6, if activity of the sympathetic system is partly suppressed. As we had only one athlete (Th5) with a lesion above Th6 among our subjects, this factor should not play an important role in the outcome of our study, since the HR is not influenced a lot in this particular case.

Even though HR reliability is rather high in our newly designed test, we do not recommend daily training control based on HR, particularly not for young athletes, because they should first achieve a stable awareness of their exercise intensity. Also, if attention is focused too much on HR, the main training goal will be delayed, because stress sensation is suppressed due to concentration on an external device (e.g. a heart rate monitor). In daily training practice, HR can also be greatly influenced by factors such as heat, wind, fluid intake and boosting in athletes with high lesion levels. These factors are standardized in most laboratory trials and are therefore more reliable than in daily training practice on the track.

Another important finding concerning training practice with young athletes is, that the development of metabolic adaptations in adolescents is not yet completed [Mahon et al., 1996]. In the same way, muscular differentiation in type I and type II fibres, as well as lactic acid metabolism, are still immature. Therefore, with young athletes, high intensity anaerobic exercise bouts should not be performed as often as with elite athletes. Aerobic training is recommended for adolescents, and more so for the spinal cord injured, because it enhances thermoregulatory capacity during exercise; something that is of high importance for this particular group [Bhambani, 2002]. Further studies reported other special conditions for adolescent athletes. Beneke et al. [1996] found that neuromuscular coordination has a higher influence on performance in young athletes than metabolic parameters. Williams et al. [1990] studied lactic acid metabolism in children. They found that they can exercise at intensities close to their peak oxygen uptake, without accumulating high levels of blood lactate. They therefore recommend not using the 4mmol blood lactate level to assess and monitor exercise performance in children between 11 and 13 years. Additionally, in spinal cord injured athletes, accumulation of lactic acid also depends on the lesion level and is significantly higher for quadriplegics than for paraplegics [Bhambani, 2002]. All these factors, concerning lactic acid accumulation and degradation, could probably have influenced our values if six of our athletes would have been between 13 and 18 years of age. We hence conclude not to use lactic acid measurements in wheelchair athletes younger than 18 years.

In the same way, it is of no practical use to measure oxygen uptake (VO$_2$) in athletes younger than 17 years [Kemper and Verschuur, 1987]. Peak VO$_2$ also has a weak relationship with the 1500m running performance of 14–18 years old boys and girls [Almarwaey, 2003], of which exercise time and distance corresponds to our tests with 1500m wheelchair racing bouts. In addition, CVs from VO$_2$ measurements are not as small as CVs from peak power output measurements [Kuipers et al., 1985]. Considering the effort and
discomfort of VO$_2$ measurements, combined with the above arguments, we do not recommend VO$_2$ measurement for testing adolescent athletes. This is the cause why we did not measure VO$_2$ in our study.

In an international study [Liow and Hopkins, 1996] on disabled athletes, it is reported that a third of all wheelchair racing athletes did not receive any coaching nor have any scientific knowledge in order to improve their training techniques. This shows potential for future support of these athletes in order to avoid the risk of overtraining, especially in the area of disabled sports [Chow and Mindock, 1999]. We are confident that this study can contribute further knowledge and help athletes and coaches to improve the training quality. We recommend the execution of the 5x1500m test at regular intervals, especially for young wheelchair racing athletes. Since the implementation of this test is very easy and noninvasive (we recommend omission of lactic acid analysis), coaches are able to perform it with their athletes, without assistance of any medical or scientific staff.

For practical use, we recommend to carry out this test every 5–6 weeks, in the above-described way (see Methods), particularly during the winter months. Especially, in this time of the year, there is no training control by means of competitions, but most athletes want to know ‘where they stand’. To train awareness of training intensities, but also to report training progress by a simple but valid testing method, we advise to predominantly perform this test during the first 2 or 3 years of an athletes’ sporting career, even though further research is still needed to measure the improvement of the training quality exactly and to quantify it somehow.

Of course, it is not a very sensitive test to detect small progress in exercise performance and it is therefore not suitable for highly trained athletes. This is the reason why we principally recommend it for junior or inexperienced athletes, in order to educate awareness of the different training levels. Nevertheless, the higher the performance level of an athlete is, the better these values will be able to be reproduced and the easier it will be to detect small changes. But to really prove this assumption and to see the difference of CVs in, for example, highly trained athletes, further studies are needed.

**Conclusions**

We conclude that our newly designed test is, with exception of the lactic acid data, suitable for practical use. We recommend a learning trial, after which a periodical conduction of the test allows for a better monitoring of the training progress, in particular during the winter months. It additionally trains the awareness of the specific training intensities. Given that the implementation of this test is rather easy, coaches are able to perform it with their athletes without further assistance. We find it to be a helpful tool for improving awareness of the individual training intensities, and for pursuing the development of the training process, as particularly the high-intensity bouts are well reproducible.
2.3 Heart rate based lactate minimum test - a reproducible method

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**Introduction**

A well-developed endurance capacity is a prerequisite for optimal exercise performance [Farrell et al., 1979] in many sports, but is also important for patients during rehabilitation. The success of endurance training depends on the duration, intensity and frequency of training [Neufer, 1989]. For an adequate training control and specification of training various parameters – such as heart rate (HR), rating of perceived exertion (RPE), blood lactate concentration, maximal lactate steady-state (MLSS) and ventilatory thresholds – are used [Green et al., 2005]. MLSS is an excellent parameter for predicting endurance performance, for assessing fitness level, and for designing training programmes [Kindermann et al., 1979]. MLSS has been defined as the highest workload that can be sustained over time without continuous blood lactate accumulation [Beneke, 1995]. Thereby, lactate should not increase >1mmol·l⁻¹ during the last 20min of a 30min constant load endurance test [Beneke et al., 2000; Billat et al., 2003; Jones et al., 1998]. In order to accurately determine MLSS, several exercise tests on different days are necessary. Thus, such a method is not suitable for practical use [Beneke, et al., 2000].

However, studies from various authors suggest that work rate and/or HR at the lactate minimum (LM), where an equilibrium between blood lactate accumulation and elimination exists, correspond to the work rate and/or HR at MLSS [Bacon and Kern, 1999; MacIntosh et al., 2002; Tegtbur et al., 1993; Tegtbur et al., 2001]. Therefore, the LMT would represent a valuable tool for designing exercise training prescriptions.

In general, the lactate minimum test (LMT) consists of two parts: the first part is used to induce a distinctive blood lactate accumulation; the subsequent second part is an incremental test starting at a moderate intensity in accordance with the guidelines for aerobic training [ACSM guidelines, 2000]. Under these aerobic conditions during the early phase of the second part, lactate is metabolised until the LM is reached. LM denotes the stage at which there is an equilibrium between blood lactate appearance and elimination. Thereafter, blood lactate concentration starts to rise again with increasing workload.

So far, only MacIntosh et al. [2002] have examined the reproducibility of the LMT; they demonstrated high reproducibility of a workload-based LMT. However, Heck et al. [1989] showed that workload at LM depends on the increase of workload in a workload-based test protocol. The effect on HR at LM was not investigated in their study. In order to meet the requirements of daily clinical use, we developed a new HR-based test protocol. In particular,
the new test mode should provide the basis for a HR-controlled training, because HR is commonly used to estimate exercise intensity.

However, to know how reliable a new test protocol is, information about its reproducibility is important. Thus, the aim of the present study was to evaluate reproducibility of our new HR-based LMT protocol. Further, a constant HR test at an intensity corresponding to the HR at the LM (LMHR) was performed to compare lactate concentrations from the LMT with values observed during endurance exercise at LMHR.

Methods

Subjects
Twenty healthy and endurance-trained individuals (13 men, 7 women) participated in the study. The subjects' characteristics were: age 30.3±7.9 years, height 175.9±8.6cm, weight 66.9±9.0kg, VO2peak 58.6±7.8ml·min⁻¹·kg⁻¹ and maximal work rate 5.2±0.9W·kg⁻¹. The study was approved by the ethics committee of Lucerne, Switzerland. Written informed consent was obtained from each subject prior to the start of the study.

Subjects were asked to abstain from strenuous training sessions on the day before each test. Training and nutrition on the days before the test and on the test days were kept uniform and recorded.

Testing protocol
At present, no standardised protocol for LMT exists, and the following elements of LMT may be varied: i) the duration and strain needed to induce lactate accumulation in the first part of the test, ii) the work rate at the start of the second part of the test, iii) the duration and the increment of the single stages during the second part of the LMT, and iv) the mode of LM determination. These points are briefly discussed below to justify our applied protocol, which consisted of two successive incremental tests (Fig. 1).

i) The first part of our LMT protocol was a Conconi test [Conconi et al., 1996] to volitional exhaustion. This test consists of an incremental protocol, with increments of 20W and duration of 2min for the first bout. Since work per bout stays equal, the duration of the bouts decreases throughout the test, causing lactic acidosis within a few minutes. In the literature, various types of strains have been used to induce lactic acidosis, including different kinds of ramp protocols, incremental tests, or short runs of high intensity [Bacon and Kern, 1999; Tegtbur et al., 1993; Tegtbur et al., 2001]. Smith et al. [2002] showed that different protocols (ramp vs. maximal exercise bouts of short duration) did not influence power output, HR or blood lactate concentration at LM.

ii) The second part of our protocol was HR-based with the intention to detect HR at LM for training prescriptions. It is crucial to choose a moderate aerobic intensity for the initial work rate of the second part. Therefore, we chose HR at rest plus 60% of heart rate reserve (HRR) as a target HR for the first stage, meeting the guidelines for aerobic training [ACSM guidelines, 2000].
iii) The duration of the stages of the second part of our LMT was set at 5 min in order to reach a steady state with constant HR at the end of each stage. In the literature, ramp protocols with stage durations of 3 or 5 min or fixed distances have been described [Bacon and Kern, 1999; MacIntosh et al., 2002; Tegtbur et al., 1993; Smith et al., 1998; Carter et al., 1999]. All these protocols used fixed incremental workloads, whereas in our study workload was individually adjusted to reach the target HR, such that an increase in HR of 8% of HRR was reached (for a detailed description of our protocol see below).

iv) In the present protocol, LM corresponds to the HR at the stage before blood lactate starts to increase again. At this stage the rate of removal of lactate from blood is greater than or equal to the appearance rate of lactate. Different procedures for determining LM have been reported in the literature [MacIntosh et al., 2002; Tegtbur et al., 1993]. The nadir of the lactate curve is either calculated or determined visually. MacIntosh et al. [2002] as well as Smith et al. [2002] suggested that both methods of analysis revealed similar results, although different exercise protocols were used in their studies.

Figure 1: Schematic protocol of the lactate minimum test, consisting of two successive incremental tests. The first part is a ramp test (Conconi protocol) and the second part is an incremental test based on predicted heart rates which lasts until blood lactate concentration begins increasing again.
Taking all these issues into account, we applied the following protocol: HR was recorded after the subject had been sitting on the ergometer for 2min (HR at rest). After a 5min warm-up at 100W, the protocol described by Conconi et al. [1996] was followed, starting at 100W, with a first bout of 2min and increments of 20W between bouts. Subjects received verbal encouragement to perform until exhaustion. Peak HR at the end of the first part of the test was defined as HRmax. The second part of the LMT started immediately after the first part ended, and was guided by the subject’s HR. For each stage a target HR was calculated. Target HR for the first stage was calculated from HR at rest plus 60% of HRR, which was determined on the basis of HR at rest and HRmax. For the following stages the increments of HR were 8% of HRR, but with a maximal augmentation of 10 bpm. Work rate was adjusted to reach the predicted HR within 3min and to keep it constant for the last 2min of the stage.

Mean HR during the last minute of each stage was used for analysis. In the second part of the test, the first stage of the LMT lasted 7min, and the following stages each lasted 5min. For lactate measurement 20ml of blood was taken from the earlobe and analysed immediately (Super GL Ambulance, Ruhrtal Labor Technik, Möhnesee, Germany). Lactate was measured during the warm-up, at the end of the first part of the test, and at the end of each stage of the second part. The test was terminated when blood lactate concentration increased by >0.2mmol·l⁻¹ compared with the previous stage. HR of the stage before lactate increased again (LM) was defined as LMHR.

To evaluate the reproducibility of LMT, subjects completed four tests, with an interval of 48h to 1 week between tests. The tests were performed on an electronically braked stationary cycle ergometer (Ergometrics900, Ergoline, Bitz, Germany). During the whole LMT, HR was measured by ECG (Cardio Soft, GE Medical Systems, Freiburg, Germany), and oxygen uptake (VO₂) was measured breath by breath by OxyconPro (Jaeger GmbH, Hoechberg, Germany). The highest 15s average for VO₂ was determined as VO₂peak.

Constant heart rate trial
On a separate occasion, the subjects performed an endurance test on the cycle ergometer at the mean LMHR resulting from the four preceding LMTs. Warm-up was standardised, with 5 min of cycling at 80%, followed by 3min at 90% and 3min at 100% of the work rate at LM. Thereafter, work rate was adjusted to reach the intended HR (LMHR ±3 bpm) within the next 5min. Subsequently, if necessary, work rate was adjusted to keep HR within these limits for the following 20min. Blood samples for lactate measurement were taken at the end of each stage of the warm-up and then every 5min until the end of the test.

Statistics
For the evaluation of reproducibility of LMT, mean coefficients of variation (CV) were calculated for HRmax, LMHR, maximum blood lactate concentration (LAmx), lactate concentration at LM (LMLA), VO₂ at LM (LMVO₂) and work rate at LM (LMWR) [Glüer et al., 1995]. Individual CVs for LMHR were presented for each subject as the ratio of the standard deviation to the mean. Data for HRmax, VO₂peak, LMVO₂, LAmx, LMLA and LMWR are presented as mean (SD) of the four tests for each subject. Further, LMHR and LMVO₂ were also calculated as a percentage of HRmax and VO₂peak, respectively. Data from the constant HR trial are presented as mean±standard deviation. Coefficients of correlation were calculated using the Pearson correlation matrix.
Results

The mean CVs for all subjects were 2.1% for LMHR, 1.7% for HRmax, 15.5% for LAmx, 17.4% for LMLA, 7.3% for LMVO₂ and 6.8% for LMWR. Individual values for LMHR at the four LMTs and corresponding individual CVs are given in table 1. Further, mean values for HRmax, VO₂peak, LMVO₂, LAmx, LMLA and LMWR of the four tests for each subject are shown in table 2.

Table 1: Individual values of lactate minimum heart rate (LMHR) of the four lactate minimum tests (LMT), as well as individual coefficients of variation (CV) for LMHR and oxygen uptake at lactate minimum (LMVO₂)

<table>
<thead>
<tr>
<th>Subject</th>
<th>LMHR of LMT1</th>
<th>LMHR of LMT2</th>
<th>LMHR of LMT3</th>
<th>LMHR of LMT4</th>
<th>CV [%] of LMHR</th>
<th>CV [%] of LMVO₂</th>
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<td>2.48</td>
<td>8.84</td>
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</table>

The LMHRs ranged from 84 to 91% of HRmax, with a mean of 88.1±2.1%. Mean LMVO₂ was 68.7±3.7% of VO₂peak. Mean HR at rest was 64±10 bpm. All subjects were able to perform the constant HR test for 20min at LMHR after the standardised warm-up. Lactate increased during the standardised warm-up and remained within a range of 1mmol·l⁻¹ in all subjects until the end of the constant HR test (Fig. 2). The individual mean values of lactate during the last 20min of the constant HR test ranged between 0.99 and 5.28mmol·l⁻¹ (2.65±1.20mmol·l⁻¹). The mean difference of individual lactate concentrations between the constant HR test and the mean LMLA was 0.55±0.49mmol·l⁻¹. A significant correlation (r=0.84, p<0.01) was found between LMLA and the lactate level during the constant heart rate trial.
Mean VO\textsubscript{2} during the last 20min of the constant HR trial was 41.5±7.2ml·min\textsuperscript{-1}·kg\textsuperscript{-1} compared with 38.7±2.8ml·min\textsuperscript{-1}·kg\textsuperscript{-1} at LM (table 2).

Table 2: Means±standard deviation of the four lactate minimum tests for each subject

<table>
<thead>
<tr>
<th>Subject</th>
<th>HR\textsubscript{max}</th>
<th>VO\textsubscript{2peak}</th>
<th>LMVO\textsubscript{2}</th>
<th>LA\textsubscript{max}</th>
<th>LMLA</th>
<th>LMWR</th>
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<td>[bpm]</td>
<td>[ml·min\textsuperscript{-1}·kg\textsuperscript{-1}]</td>
<td>[ml·min\textsuperscript{-1}·kg\textsuperscript{-1}]</td>
<td>[mmol·l\textsuperscript{-1}]</td>
<td>[mmol·l\textsuperscript{-1}]</td>
<td>[W]</td>
</tr>
<tr>
<td>1</td>
<td>178 ± 4</td>
<td>53.5 ± 1.7</td>
<td>38.0 ± 2.5</td>
<td>8.9 ± 1.6</td>
<td>2.2 ± 0.5</td>
<td>168 ± 13</td>
</tr>
<tr>
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<td>199 ± 3</td>
<td>62.9 ± 4.3</td>
<td>41.5 ± 3.0</td>
<td>6.2 ± 2.1</td>
<td>1.2 ± 0.2</td>
<td>200 ± 12</td>
</tr>
<tr>
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<td>3.7 ± 1.1</td>
<td>196 ± 12</td>
</tr>
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<td>7.8 ± 1.5</td>
<td>1.6 ± 0.4</td>
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<tr>
<td>5</td>
<td>182 ± 5</td>
<td>58.4 ± 2.6</td>
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<td>7.6 ± 1.5</td>
<td>1.9 ± 0.3</td>
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<td>3.0 ± 0.7</td>
<td>223 ± 14</td>
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<tr>
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<td>61.4 ± 2.2</td>
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<td>9.7 ± 1.5</td>
<td>2.9 ± 0.6</td>
<td>210 ± 15</td>
</tr>
<tr>
<td>8</td>
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<td>3.3 ± 0.6</td>
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<td>4.8 ± 0.8</td>
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<tr>
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<td>186 ± 12</td>
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</table>

HR\textsubscript{max}: maximal heart rate; VO\textsubscript{2peak}: peak oxygen consumption; LMVO\textsubscript{2}: oxygen consumption at lactate minimum; LA\textsubscript{max}: lactate maximum; LMLA: lactate at lactate minimum; LMWR: work rate at lactate minimum; SD: standard deviation
Figure 2: Individual blood lactate curves during the constant heart rate trial.

Figure 3: Correlation of lactate at lactate minimum (LMLA) vs. lactate during the constant heart rate trial (CTLA).
Discussion

Reproducibility
The purpose of our study was to investigate the reproducibility of LMHR with a new HR-based LMT protocol. The main finding was that this protocol showed a high reproducibility for LMHR (CV = 2.1%; individual SD = 3.2 bpm) evaluated in four tests. Individual LMHR was within a range of 2 to 11 bpm, which corresponded to individual CVs between 0.59 and 3.33% over the four tests (table 1). In the literature, CVs of <6% for HR were estimated to represent very high reproducibility [Bhambhani et al., 1991; Jeukendrup et al., 1996]. Jeukendrup et al. [1996] compared different performance tests and interpreted CVs of <3.5% as highly reproducible. In the present study an even higher reproducibility was found for HRmax (CV = 1.7%; SD = 2.7 bpm). This was probably due to well-trained and highly motivated subjects performing to exhaustion in every single test. Work rate at LM showed good reproducibility (CV = 6.8%). Lactate concentration at LM was less reproducible (CV = 17.4%). The CV for work rate at LM may be an expression of daily variation of physical capacity. The high CV for LMLA might be due to the dependence of lactate metabolism on nutrition, training sessions in between test days and current level of glycogen stores, as well as hormonal and vegetative conditions of the subject.

The advantage of a HR-based protocol is the constant HR value at the end of each stage. If LMTs are performed based on predetermined workload or velocity increments, a HR drift within a single step can often be observed. If HR-based training prescriptions are deduced from such a test, it may cause misleading interpretations.

Testing protocol
In contrast to the studies described so far [Jones and Doust, 1998; Bacon and Kern, 1999; MacIntosh et al., 2002; Smith et al., 2002; Tegtbur et al., 1993, Tegtbur et al., 2001], we decided to use a HR-based test protocol, which should be a helpful and practical tool for making HR-based training prescriptions. The HR-based test protocol also meets the observation of sports practice, where athletes perform at a constant HR, for example, during cycling time trial competitions [Palmer et al., 1999; Hoogeveen et al., 1997]. The proposed HR-based protocol can be used in different sports disciplines and over a wide range of fitness levels. A further advantage of LMT is the independence of absolute lactate concentrations, which means that individual lactate kinetics are considered [Tegtbur et al., 1993; Tegtbur et al., 2001; Carter et al., 1999; Hoogeveen et al., 1997].

Determination of LM seems to depend on the test protocol and data analysis used during the second part of the LMT. Heck et al. [1989] found higher work rates at LM after higher blood lactate concentrations at the end of the first part of the LMT. In their study the stages of the second part lasted only 1.5 min. This is too short to achieve a steady state in lactate metabolism before the workload is again increased, and probably led to the above mentioned higher work rate at LM. The choice of the initial work rate of the second part of the LMT is also important. Different initial workloads were found in the literature [Tegtbur et al., 1993; Tegtbur et al., 2001]. However, when the starting speed was low, subjects reached a lactate steady state at several stages [Carter et al., 1999]. Therefore, Carter et al. [1999] concluded that LM was profoundly influenced by the starting speed. It is thus important to choose a moderate initial exercise intensity. The results of the present study seem to indicate that our initial target HR during the second phase of the LMT was adequate. In the case of the use of
ramp protocols during this second part of the LMT, mathematical analysis by means of curve fitting is suitable [Jones and Doust, 1998; Tegtbur et al., 1993]. However, if stages of 3 to 5 min are applied, LM is denoted by the last stage before lactate concentration starts to rise again.

Lactate, HR and work rate at LM
Tables 1 and 2 show that the absolute individual values of HRmax, LMHR and LMLA have considerable interindividual differences. The large range of 29 bpm for individual HRmax and LMHR emphasises the interindividual differences for training HRs as well. Similar results with weak correlation of HRmax and age are found in the literature [Whaley et al., 1992]. The large range of the individual LMLA (1.2–6.8 mmol·l⁻¹) would tend to support the conclusion of Tegtbur et al. [2001] and Myburgh et al. [2001] that there exists no fixed lactate threshold (e.g. 4 mmol·l⁻¹), but that threshold values are individual.

There exists a significant correlation between LMLA and the lactate level during the constant HR test at an intensity corresponding to LMHR (Fig. 3). This finding is not surprising to us, as intensity (HR) during the constant HR trial corresponds to the intensity detected at LM. However, the similar lactate concentrations at LM as well as those during the constant HR trial indicate that the metabolic demands are comparable. As all our subjects were able to perform the last 20 min of the constant HR test at LMHR, the intensity at LMHR could not have been higher than MLSS. Indeed, although lactate concentrations increased to values of between 0.93 and 5.25 mmol·l⁻¹, they remained constant during the last 20 min of the constant ride (i.e. within 1 mmol·l⁻¹).

LM in relation to HRmax and VO₂peak
The oxygen consumption at LM with 68.7±3.7% of VO₂peak in our study was far below the 81.4% found by Bacon and Kern [1999]. In their study exercise tests were performed on a treadmill with increments of 3 min. We suppose that the different testing mode is one of the reasons for these different results.

Limitations of the study
The abovementioned, lower VO₂ obtained at LM compared with the data of Bacon and Kern [1999] might also indicate an underestimation of MLSS based on LM in some cases in the present study. Therefore, we cannot exclude the possibility that several subjects would have been able to perform at a higher intensity during the constant HR trial without a continuous accumulation of lactate. This assumption is supported by the fact that in some subjects lactate concentrations tend slightly to decrease towards the end of the endurance test (Fig. 2). Thus, although the LMHRs determined in the present study provided HRs leading to steady-state conditions in blood lactate levels, we are far from assuming that the present LMHR corresponds to HR at MLSS. Therefore, further studies are necessary to elucidate in detail the relationship between LMHR and MLSS.

Moreover, the accuracy of LMHR would possibly be further improved by making smaller steps in target HRs. Possibly, in certain cases, the relatively small number of steps during the second part of the LMT may have contributed to an underestimation of LMHR. Further, a higher number of stages might also contribute to a further decrease in the intra-individual
variability observed. Finally, one cannot exclude the possibility that the work duration during moderate intensity exercise (7 min for the first step and 5 min afterwards) was too long, leading to an important recovery mimicking what has been seen in the study of Carter et al. [1999]. In such a case, the lower concentration observed during the second part of the LMT would not correspond to MLSS and the increase of blood lactate level during the next step may indicate the reaching of a new steady state at a higher blood lactate concentration but not an imbalance between lactate appearance and disappearance. In order to prove these assumptions, further studies are needed, which may lead to future improvements in our HR-based LMT protocol as well as to a better understanding of the relationship between data at LM and MLSS.

**Conclusions**

Based on the findings of our study, we conclude that our proposed LMT with a HR-based test protocol is a reproducible method for assessment of LM and LMHR. Further investigations should evaluate the relation between LM and MLSS.
2.4 Correlation of heart rate at lactate minimum and maximal lactate steady state in wheelchair racing athletes

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Introduction

The maximal lactate steady state (MLSS) is a common predictor of endurance exercise capacity and a helpful tool to guide training intensities [Haverty et al., 1988; Heck et al., 1985; Kindermann et al., 1979]. MLSS is defined as the highest exercise intensity that can be sustained over time without continuous blood lactate accumulation [Beneke et al., 2000]. Thereby, blood lactate should not increase >1mmol·l⁻¹ during the last 20min of a 30min constant load endurance test [Beneke et al., 2000; Billat et al., 2003; Jones and Doust, 1998].

To accurately determine MLSS, several exercise tests at different intensities and on different days are necessary, making this method impractical [Beneke et al., 2000]. However, there is some evidence from the literature that exercise intensity at MLSS can be determined by a single test - the so-called lactate minimum test (LMT) in patients as well as in moderately and well-trained athletes [Bacon and Kern, 1999; Knoepfli-Lenzin and Boutellier, 2011; MacIntosh et al., 2002; Tegtbur et al., 2001].

Besides the fact that the LMT would be a time-saving method (because only a single test is necessary) to determine MLSS, it also offers further advantages. Test results seem to be independent of the previous nutritional status [Tegtbur et al., 1993] or of the investigator’s experience [Knoepfli-Lenzin and Boutellier, 2011]. In combination with an ergospirometric assessment, maximal oxygen consumption can be concomitantly determined during an LMT as well [Dantas De Luca et al., 2003]. In summary, the LMT seems to be an easy and objective tool providing accurate and helpful results not only for scientific purposes but also for athletes and coaches in their daily practice [Knoepfli-Lenzin and Boutellier, 2011], at least for running and cycling exercise in able-bodied subjects.

In general, an LMT consists of two parts: During the first part, high-intensity exercise is performed to induce severe lactic acidosis. The second part corresponds to an incremental test starting at a moderate aerobic exercise intensity. Under these aerobic conditions during the early phase of the second part, blood lactate is metabolized with increasing workloads until a lactate minimum (LM) is reached. LM denotes the intensity at which an equilibrium between blood lactate appearance and elimination is established. Thereafter, blood lactate concentration begins to rise again.
Recently, our group developed a new, standardized and user-friendly heart rate (HR)-guided LMT protocol, which was found to be a highly reproducible method for cycling exercise in able-bodied subjects [Strupler et al., 2009]. However, there exists no study so far that used the LMT for exercise testing in wheelchair athletes. Additionally, it seems difficult to transfer the present knowledge from LMTs with able-bodied subjects into such a special group of disabled athletes. In this context one has to bear in mind that compared with able-bodied individuals, athletes with a spinal cord injury have unique changes in metabolic, cardiorespiratory, neuromuscular and thermoregulatory systems, which reduce their overall physical capacity [Bhambhani, 2002]. These disability-related physiological changes may lead to different test results using the HR-based LMT in wheelchair athletes. Thus, the aim of the present study was to investigate the precise relationship between HR at MLSS and that at LM in competitive wheelchair-racing athletes based on our HR-guided LMT protocol [Strupler et al., 2009].

Methods

Subjects
Eight well-trained wheelchair athletes (seven men, one woman) participated in the study. The subject characteristics and paralympic racing classification can be found in Table 1. The study was approved by the local ethics committee. Written informed consent was obtained from each subject (for the minor, consent was obtained from his parents) before the start of the study. We certify that all applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during the course of this research. Subjects were asked not to perform strenuous workouts the day before each test. Training and nutrition on the days before the test and on the test days were kept constant and recorded.

Table 1: Subject characteristics

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age [y]</th>
<th>Height [cm]</th>
<th>Body mass [kg]</th>
<th>Peak HR [bpm]</th>
<th>Lesion level/impairment</th>
<th>AIS/completeness</th>
<th>Race class</th>
<th>TPI [y]</th>
</tr>
</thead>
<tbody>
<tr>
<td>F</td>
<td>36</td>
<td>150</td>
<td>38</td>
<td>160</td>
<td>Th6</td>
<td>A/compl.</td>
<td>T53</td>
<td>20</td>
</tr>
<tr>
<td>M</td>
<td>48</td>
<td>178</td>
<td>58</td>
<td>183</td>
<td>Th4</td>
<td>A/compl.</td>
<td>T53</td>
<td>28</td>
</tr>
<tr>
<td>M</td>
<td>42</td>
<td>173</td>
<td>59</td>
<td>184</td>
<td>Th4</td>
<td>A/compl.</td>
<td>T53</td>
<td>20</td>
</tr>
<tr>
<td>M</td>
<td>29</td>
<td>188</td>
<td>67</td>
<td>186</td>
<td>Th5</td>
<td>A/compl.</td>
<td>T53</td>
<td>2</td>
</tr>
<tr>
<td>M</td>
<td>20</td>
<td>170</td>
<td>65</td>
<td>189</td>
<td>Spina bifida</td>
<td>C/incomp.</td>
<td>T54</td>
<td>20</td>
</tr>
<tr>
<td>M</td>
<td>15</td>
<td>159</td>
<td>49</td>
<td>199</td>
<td>Th10</td>
<td>D/incomp.</td>
<td>T54</td>
<td>15</td>
</tr>
<tr>
<td>M</td>
<td>26</td>
<td>165</td>
<td>72</td>
<td>194</td>
<td>Spina bifida</td>
<td>A/compl.</td>
<td>T54</td>
<td>26</td>
</tr>
<tr>
<td>M</td>
<td>46</td>
<td>165</td>
<td>65</td>
<td>182</td>
<td>Th12</td>
<td>A/compl.</td>
<td>T54</td>
<td>24</td>
</tr>
</tbody>
</table>

Mean 32.8 168.5 59.1 185 19.4
SD 12.2 11.6 11.0 12 8.1

Abbreviations: HR: heart rate; AIS: American Spinal Injury Association Impairment Scale; TPI: time post injury; F: female; M: male; compl.: complete lesion; incomp.: incomplete lesion; Th: thoracic lesion; SD: standard deviation
Testing protocol

Part A: Lactate minimum test (LMT). Subjects completed a HR-based LMT in their own racing wheelchair on a motor-driven treadmill (HP Cosmos Saturn, HP Cosmos, Traunstein, Germany) according to the following protocol described in detail by Strupler et al. [2009]. Briefly, HR was recorded after sitting in the wheelchair for 2min (HR at rest). After a 5-min warm-up period at 10km·h⁻¹, the LMT began with the protocol described by Conconi et al. [1996], starting at 12km·h⁻¹. At every 200m, treadmill speed was increased by 0.5km·h⁻¹. The incline of the treadmill was set at 2% and subjects received verbal encouragement to perform until exhaustion. Peak HR at the end of the Conconi test was defined as maximal HR.

The second part of the LMT started immediately at the end of the Conconi test and was guided by the subject's individual HR. For each stage a target HR was calculated. Target HR for the first stage was calculated from HR at rest plus 60% of HR reserve, which was determined by subtraction of HR at rest from the maximal HR, meeting the guidelines for aerobic training [ACSM, 2000]. For the following stages the increments of HR were 8% of the HR reserve, but with a maximal augmentation of 8 bpm. Treadmill speed was adjusted to reach the predicted HR within 3min and to keep it constant (±2bpm) for the last 2min of the stage. Mean HR during the last minute of each stage was used for analysis. The first stage of the second part of the LMT lasted 7min and the following stages lasted 5min each. The test was terminated when blood lactate concentration started to rise again by at least 0.2mmol·l⁻¹. On average, a HR-guided LMT lasted about 40min, whereas normally five stages during the second part of the LMT were performed until LM could be determined.

For blood lactate measurements, 20µl of blood was taken from the earlobe and analyzed immediately by an enzymatic method (Super GL Ambulance, Ruhrtal Labor Technik, Möhnesee, Germany). Blood lactate was measured at rest, at the end of the first part and at the end of each stage of the second part of the LMT while the subjects were exercising. HR was monitored continuously (Polar S610i, Polar Electro, Kempele, Finland) during the LMT, rating of perceived exertion according to Borg [1982] was acquired using the 15-point scale ranging from 6 (no exertion) to 20 (maximal exertion). Oxygen uptake (Oxycon Pro, Jaeger GmbH, Hoechberg, Germany) was determined breath by breath, whereas 15s-averaged values were used for data analysis. Treadmill speed was recorded continuously during the LMT. The test was terminated when blood lactate concentration increased by >0.2mmol·l⁻¹ compared with the previous stage. HR of the stage before blood lactate increased again (LM) was defined as HR at lactate minimum (LMHR).

Part B: Endurance tests to determine maximal lactate steady state (MLSS). Based on LMHR, the MLSS (represented by maximal constant HR) was determined by several endurance tests on different days. All endurance tests of the same subject were performed at the same time of the day in order to exclude circadian performance fluctuations. Each endurance test started with a standardized warm-up of 3min at 60% of the speed at LM (determined in the LMT described above in Part A), 3min at 80% and 4min at 100% of the speed at LM. Subsequently, treadmill speed was increased until the predetermined target HR was reached within 5min. Thereafter, target HR was kept constant (±2bpm, corresponding to a range of 4bpm) for 30min. If necessary, treadmill speed was adequately adjusted to keep HR within these narrow limits. Treadmill incline was again set at 2%.

Blood lactate samples were drawn at rest, after 3, 6 and 10min of warm-up, as well as subsequently every 5min until the end of the endurance test. Blood lactate analysis followed
immediately after blood sampling. Rating of perceived exertion was always recorded immediately before blood lactate sampling. HR as well as ventilatory and gas exchange variables were measured continuously throughout the test.

Target HR for the first endurance test was set at 8 bpm above LMHR. If blood lactate concentration did not increase by >1mmol·l⁻¹ during the last 20 min of the endurance test, HR intensity was increased by another 4 bpm (=LMHR+12 bpm) for the second endurance test (Figure 1). In case of a lactate steady state at this intensity, target HR was again adapted in each subsequent endurance test by 4 bpm until MLSS was determined.

Figure 1: Study design for the determination of maximal lactate steady state (MLSS) based on a heart rate-guided lactate minimum test (LMT).

If blood lactate increased by >1mmol·l⁻¹ during the last 20 min of the first endurance test or if a subject was not able to complete the endurance test at an intensity 8 bpm above LMHR, target HR for the next endurance test was reduced by 4 bpm (Figure 1). This study design allowed us to determine the MLSS for each subject after 2–3 endurance tests.

Statistics
For statistical analysis a commercially available software package (SPSS 13, Chicago, IL, USA) was used. Data are presented as means±standard deviation. Mean values at LM and MLSS were compared using a paired-sample t-test. The coefficients of correlation were calculated using Pearson’s correlation matrix. The level of significance was set at P<0.05. In order to show the difference between data at LM and at MLSS, Bland and Altman plots were used [Bland and Altman, 1986] to compare the difference between MLSS and LM (bias) versus their average value.
Results

All measured parameters (HR, blood lactate concentration, rating of perceived exertion, oxygen uptake (VO2) and treadmill velocity) were significantly lower at LM compared with MLSS (Table 2). The corresponding Bland and Altman plots revealed a positive bias for each parameter mentioned, whereas all data with the exception of one data point were within the limits of agreement.

Table 2: Mean ± standard deviation of lactate concentration, oxygen consumption, rating of perceived exertion (Borg) and treadmill velocity at lactate minimum and during the last 20min of an endurance test at maximal lactate steady state

<table>
<thead>
<tr>
<th></th>
<th>Lactate minimum</th>
<th>Maximal lactate steady state</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate [bpm]</td>
<td>163 ± 6</td>
<td>172 ± 7*</td>
</tr>
<tr>
<td>Lactate concentration [mmol·l⁻¹]</td>
<td>3.0 ± 1.7</td>
<td>4.6 ± 2.2*</td>
</tr>
<tr>
<td>Oxygen uptake [ml·min⁻¹·kg⁻¹]</td>
<td>30.0 ± 4.7</td>
<td>35.7 ± 6.6*</td>
</tr>
<tr>
<td>Rating of perceived exertion (Borg)</td>
<td>14.1 ± 1.5</td>
<td>17.0 ± 1.4*</td>
</tr>
<tr>
<td>Treadmill velocity [km·h⁻¹]</td>
<td>14.1 ± 3.0</td>
<td>16.6 ± 2.8*</td>
</tr>
</tbody>
</table>

*P<0.05 between values at lactate minimum and maximal lactate steady state

All subjects were able to complete the first endurance test without a blood lactate increase of >1mmol·l⁻¹ during the last 20min of the test. Thereafter, seven out of eight subjects were not able to complete the second endurance test at an intensity of 12bpm above LMHR and the remaining subject aborted the third endurance test at 16bpm above LMHR.

There was a significant correlation between LMHR and HR at MLSS (r=0.914; P=0.002; Figure 2), but HR at MLSS was significantly higher than LMHR (172±7 versus 163±6bpm; P<0.001). The mean difference (bias) for LMHR and HR at MLSS, and the limits of agreement (mean±2 standard deviations of the difference) are graphically presented in Figure 3 by means of a Bland and Altman plot. The corresponding bias for HR was 8±3bpm. LMHR and HR at MLSS were found to be 88±2% and 92±2% of the maximal HR, respectively.

Peak blood lactate concentration at the end of the first part of the LMT was 8.5±2.3mmol·l⁻¹, absolute peak oxygen consumption (VO2peak) 2.76±0.73l·min⁻¹, maximal treadmill speed 21.2±4.0km·h⁻¹ and the corresponding rating of perceived exertion 19±1.
Significant correlations between values at LM and MLSS were found for treadmill speed ($r=0.935, P=0.001$), blood lactate concentrations ($r=0.944; P<0.001$) and VO$_2$ ($r=0.798, P=0.018$). VO$_2$ at LM and at MLSS were $67\pm7\%$ and $76\pm9\%$ of the VO$_{2\text{peak}}$, respectively.

Figure 2: Correlation of heart rate at maximal lactate steady state (MLSS-HR) versus heart rate at lactate minimum (LMHR).

Figure 3: Bland and Altman plot comparing the difference between heart rate (HR) at maximal lactate steady state and lactate minimum (bias) versus their average value. SD: standard deviation.
Discussion

The main finding of the present study was that all measured values at LM determined by a HR-guided LMT were significantly below the values at MLSS (Table 2). These findings are in line with the results of other studies that used workload-guided LMT protocols [Knoepfli-Lenzin and Boutellier, 2011; MacIntosh et al., 2002]. However, we found a close and significant correlation between values at LM and those at MLSS for HR data (Figure 2), which allows the prediction of HR and thus exercise intensity at MLSS in our wheelchair-racing athletes. These findings support the role of HR as one of the main predictors of training intensity, especially during exercise in the field [Foster et al., 1995; Röcker et al., 2003]. This is of practical importance, as transferring laboratory-based absolute data such as speed or workload often is difficult [Vobejda et al., 2006], even more in wheelchair racing. Further, the application of HR as a feasible tool for guidance of endurance training was also confirmed by results of a recently published study [Vobejda et al., 2006] showing a high and reproducible correlation between maximal constant HR and MLSS. This result is in line with observations of sports practice, where athletes perform at a constant HR during competitions (e.g. cycling time trial performance) [Hoogeveen et al., 1997; Palmer et al., 1999] rather than at constant blood lactate concentrations or VO$_2$. Results of a study by Janssen et al. [2001] investigating male handcycle users seem to support these findings also for subjects with spinal cord injury. Keeping this in mind, HR seems to be a useful and easy-to-use parameter to guide training intensity even in wheelchair racing athletes with intact sympathetic heart innervation. However, to determine different HR intensity zones for a systematic guidance of the endurance training, the knowledge of HR at and related to MLSS seems to be crucial. Based on the present findings, it seems possible to gain this information for a group of highly trained wheelchair-racing athletes of the paralympic racing categories T53 and T54 based on a single LMT, which helps to facilitate the daily business of athletes, coaches and scientists.

Additionally, the use of a LMT seems to offer further advantages compared with other common exercise tests applied. LMT results seem to be independent of the previous nutritional status [Tegtbur et al., 1993] and of the investigator’s experience, as test results do not depend on a subjective estimation of, for example, ‘thresholds’ [Knoepfli-Lenzin and Boutellier, 2011]. Further, in combination with an ergospirometric assessment, maximal oxygen consumption can be concomitantly determined during the first part of an LMT as well [Dantas De Luca, 2003]. In summary, the LMT seems to be an easy and objective test providing accurate and helpful test results not only for scientific purposes but also for the daily work of athletes and coaches [Knoepfli-Lenzin and Boutellier, 2011].

However, although the above-mentioned advantages seem to be valid for the LMT in general, one has to keep in mind that at present several different LMT protocols are applied. This circumstance might lead to slightly different outcomes depending on the test protocol used (e.g. workload-guided versus HR-guided protocols). Several previous studies [Bacon and Kern, 1999; MacIntosh et al., 2002; Tegtbur et al., 1993; Tegtbur et al., 2001] have found that power output at LM corresponded to the definition of MLSS when performing a constant workload test, whereas a slightly higher power output resulted in increasing blood lactate concentrations throughout the test. This observation stands in contrast to the results presented in our study. However, all the above-mentioned studies used a workload-guided protocol, whereas our study was based on a HR-guided one [Strupler et al., 2009]. This means that with our test protocol the workload during the second part of the LMT is
continuously adjusted to reach the predetermined HR and to maintain it within narrow limits. This stands in contrast to workload-guided protocols, where a constant workload for each stage is kept, which may lead to a HR drift.

Moreover, in the present study, subjects transitioned immediately into the incremental phase after the high-intensity loading phase during the first part of the LMT. In contrast, most of the other studies included a rest or recovery period of several minutes before starting with the incremental phase [Bacon and Kern, 1999; Jones and Doust, 1998; Knoepfli-Lenzin and Boutellier, 2011; MacIntosh et al., 2002; Tegtbuer et al., 1993; Tegtbuer et al., 2001]. This difference in methodology might have led to different lactate kinetics resulting in significantly lower values at LM compared with MLSS (Table 2) in the present study. Further, the increments of 8bpm for each stage during the second part of the LMT might also have resulted in a certain inaccuracy, as such an increment might be overspending. In this context, a modification of the LMT test protocol towards smaller and/or shorter increments during the second part of the LMT might be helpful, but first has to be validated by further investigations. However, if successful, such an adaptation should result in the outcome that values at LM ideally correspond to values at MLSS. Such a finding would further improve the quality and accuracy of the HR-guided LMT.

Finally, one has to bear in mind that our study investigated wheelchair athletes performing upper-body exercise, whereas the results of former studies were based on cycling or running data in able-bodied subjects, where a higher total muscle mass is involved during exercise. One could hypothesize that the amount of muscle mass involved might influence the relation between HR at LM compared with MLSS. However, further studies are needed to prove this hypothesis.

Conclusions

There exists a close relationship between LMHR and HR at MLSS in wheelchair-racing athletes, but also for other parameters such as treadmill speed and blood lactate concentration. Consequently, the prediction of MLSS based on a single HR-based LMT in this special group of athletes can be obtained. If training is guided based on HR recommendations during the daily training routine, HR at MLSS can be assumed to be 8 to 9bpm above LMHR in wheelchair-racing athletes.
2.5 Comparison of blood lactate elimination in paraplegic and able-bodied subjects during active recovery from exhaustive exercise

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Affiliations: ¹Institute of Human Movement Sciences and Sport, ETH Zurich, Switzerland  
²Institute for Clinical Research, Swiss Paraplegic Research, Nottwil, Switzerland


Introduction

Strenuous exercise leads to lactate and hydrogen ion production in the exercising muscle and a concomitant decrease in intracellular pH, which compromises muscle contraction and glycolytic enzyme activity and thus, exercise performance [Hermansen, 1979]. In order to regain optimal performance as soon as possible, fast lactic acid elimination is crucial for athletes. Lactate oxidation takes place primarily in type-I fibers of working skeletal muscle, in the liver and the heart [Brooks, 1991]. However, there is some evidence that inactive skeletal muscle also plays a role in lactate metabolism. It was shown in able-bodied people that inactive skeletal muscle can store lactate [Lindinger et al., 1990; Poortmans et al., 1978] and retain 24% of the total lactate produced 25 minutes after cessation of strenuous exercise [Lindinger et al., 1990]. Furthermore, 5% of the lactate produced during maximal exercise is metabolized in inactive skeletal muscle [Poortmans et al., 1978].

Adaptations after spinal cord injury (SCI) include a loss of muscle mass [Lotta et al., 1991; Spungen et al., 2000] in the paralyzed limbs. Olive et al. [2003] found a 38% reduction of muscle volume in paralyzed legs 10 years after an accident leading to SCI. Another adaptation to SCI is reduced oxidative capacity of paralyzed limbs, because no more type-I fibers can be found in the leg muscles of people with chronic paraplegia, although the proportion of these fibers in able-bodied people is up to 40% [Burnham et al., 1997]. As a consequence and in comparison with able-bodied individuals, the paralyzed leg muscles of individuals with paraplegia might have a reduced capacity of taking up and oxidizing lactate when performing upper body exercise.

Because the opportunities for people with paraplegia to participate in sports have increased markedly in recent years, it seems important to know whether training practices of able-bodied athletes can be adopted without any modifications. If one would observe differences in lactate removal between paralyzed and able-bodied athletes, then training designs such as recovery duration between maximal bouts of interval training ought to be adapted. This seems to be of relevance in professional sport, in which peak performance is dependent on optimization of these kinds of details.

The aim of the present study was therefore to investigate whether paralyzed (P) participants and able-bodied (AB) participants would show significant differences in blood lactate elimination after exhaustive arm exercise. Based on the above-mentioned differences
between P and AB participants concerning leg muscle mass and fiber-type composition, we expected a slower lactate elimination rate for P individuals compared with AB individuals.

**Methods**

**Study participants**

Eight P and eight AB men participated in this study, which was approved by the local ethics committee. All participants gave their written informed consent and completed a detailed questionnaire about their health histories and dietary practices; training and especially arm-training status was assessed using a questionnaire about each person’s sportive activities. P participants were mainly engaged in sports like hand biking or wheelchair basketball, whereas AB participants had performed sports like swimming, rowing or cross-country skiing for several years. Activities of daily living were not considered arm-training exercise in P participants.

The P and AB groups did not differ in age, height, weight, and weekly arm-training volume (Table 1). In the P group, all participants had complete SCI (ASIA A). Those in the P group had been paralyzed for 17±7 years (range: 9–28 years). Lesion levels ranged from T4 to T12.

**Table 1: Study participants’ characteristics**

<table>
<thead>
<tr>
<th></th>
<th>Able-bodied</th>
<th>Paraplegic</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age [y]</td>
<td>33.5 ± 10.7</td>
<td>37.8 ± 9.8</td>
<td>0.420</td>
</tr>
<tr>
<td>Height [cm]</td>
<td>177.5 ± 7.1</td>
<td>179.5 ± 7.5</td>
<td>0.593</td>
</tr>
<tr>
<td>Weight [kg]</td>
<td>77.8 ± 8.0</td>
<td>76.0 ± 11.0</td>
<td>0.721</td>
</tr>
<tr>
<td>Arm training [h·wk⁻¹]</td>
<td>2.8 ± 0.8</td>
<td>4.1 ± 1.6</td>
<td>0.061</td>
</tr>
</tbody>
</table>

**Materials**

All tests were performed by using an arm-cranking ergometer (Ergometrics 800 SH, Ergoline, Bitz, Germany). Test participants were placed on a chair that was connected to the ergometer. Before each test, the pedal axis was aligned with the participant’s shoulder, and participants were positioned such that their elbows were slightly flexed at maximal reach. Their feet were placed on the floor such that the knees were bent at an angle of approximately 90°. Mixed-capillary blood was taken at the earlobe to determine lactate concentrations as measured by an enzymatic lactate analyzer (Super GL Ambulance, Ruhrtal Labor Technik, Mönneese, Germany). For heart rate determination, a monitor watch (Polar S610, Polar, Kempele, Finland) was used. Oxygen consumption was determined by using an ergospirometric device (Oxycon Pro, Jaeger, Würzburg, Germany), which was calibrated immediately before each test according to the manufacturer’s recommendations.
Exercise protocol
The test consisted of a maximal intensity-graded exercise test with a subsequent 30-minute active recovery period. The graded exercise test started at 20W. Thereafter, the workload was increased 5W every 20s until the participant’s volitional exhaustion. The load was then reduced to one third of the maximally achieved power output (Pmax) for active recovery. Immediately before starting the active recovery period, study participants rated their overall perceived exertion by means of the Borg Scale [Borg, 1982], with a rating of 6 indicating “no” and 20 “maximal” exhaustion. Further, they were asked to indicate the reason for exhaustion. Lactate was sampled at rest, immediately after cessation of the intensity-graded exercise test, as well as every minute up to the 10th minute of the active recovery period. For the remaining 20min of recovery, blood sampling was performed every 4min.

Data evaluation and statistical analysis
Data points of the measured lactate concentrations were fitted to the following biexponential curve, as described in detail elsewhere [Freund and Zouloumian, 1981]:

$$La(t) = La(0) + A_1 \cdot (1 - e^{-\gamma_1 t}) + A_2 \cdot (1 - e^{-\gamma_2 t})$$

La(t) denotes the time-dependent lactate concentration, with La(0) being the lactate concentration at the start of recovery. This equation suggests that the lactate kinetics during recovery can be described by two main processes, one with a high velocity constant (\(\gamma_1\)) describing the appearance (\(A_1 > 0\)) of lactate in the bloodstream and the other with a low velocity constant (\(\gamma_2\)) describing its disappearance (\(A_2 < 0\)).

The parameters were calculated using SYSTAT (Version 10, SPSS Inc, Richmond, CA) with the regression method of least meansquares. The maximal lactate concentration (La\(_{max}\)) was defined as the peak value of the fitted curve. All statistical analysis was conducted using SYSTAT. Variables were compared with an unpaired two-tailed t test for data with separate variances. Statistical significance was set at P < 0.05.

Results
The velocity constant for lactate elimination (\(\gamma_2\)) showed no significant difference between P and AB participants. However, the velocity constant for lactate accumulation (\(\gamma_1\)) was significantly higher in P participants (Table 2).

The time course of average blood lactate and corresponding biexponential correlation curves for P and AB participants are presented in Figure 1. The correlation coefficients of the individual curves ranged from 0.98 to 0.99. Significantly lower values for P participants compared with AB participants were found in P\(_{max}\), La\(_{max}\), and peak oxygen consumption (VO\(_{2peak}\)), whereas no statistical difference was found in maximal heart rate (HR\(_{max}\)). Corresponding data are presented in Table 2. All participants indicated exhaustion of the arm muscles as the exercise-limiting factor. Rating of overall perceived exertion ranged from 18 to 20 and showed no significant difference between groups (Table 2).
Table 2: Results of comparisons of able-bodied men vs. men with paraplegia

<table>
<thead>
<tr>
<th></th>
<th>Able-bodied</th>
<th>Paraplegic</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\gamma_1$ [min$^{-1}$]</td>
<td>0.257 ± 0.064</td>
<td>0.419 ± 0.133</td>
<td>0.008</td>
</tr>
<tr>
<td>$\gamma_2$ [min$^{-1}$]</td>
<td>0.054 ± 0.030</td>
<td>0.062 ± 0.036</td>
<td>0.632</td>
</tr>
<tr>
<td>$P_{\text{max}}$ [W]</td>
<td>158.8 ± 15.1</td>
<td>136.3 ± 17.3</td>
<td>0.015</td>
</tr>
<tr>
<td>Lac$_{\text{max}}$ [mmol·l$^{-1}$]</td>
<td>9.7 ± 1.1</td>
<td>8.6 ± 0.4</td>
<td>0.014</td>
</tr>
<tr>
<td>VO$_{2\text{peak}}$ [ml·kg$^{-1}$·min$^{-1}$]</td>
<td>36.6 ± 2.9</td>
<td>29.5 ± 4.4</td>
<td>0.002</td>
</tr>
<tr>
<td>HR$_{\text{max}}$ [min$^{-1}$]</td>
<td>180.0 ± 12.9</td>
<td>174.8 ± 13.4</td>
<td>0.437</td>
</tr>
<tr>
<td>Borg value</td>
<td>19.8 ± 0.5</td>
<td>19.5 ± 0.9</td>
<td>0.506</td>
</tr>
</tbody>
</table>

$\gamma_1$: velocity constant for lactate accumulation; $\gamma_2$: velocity constant for lactate elimination; $P_{\text{max}}$: maximal power output; Lac$_{\text{max}}$: maximal lactate concentration; VO$_{2\text{peak}}$: peak oxygen uptake; HR$_{\text{max}}$: maximal heart rate

Figure 1: Comparison of blood lactate elimination between P and AB participants during active recovery after exhaustive arm exercise (means and standard deviations). The point of origin of the time scale indicates the beginning of active recovery.
Discussion

Blood lactate elimination
The expected difference in \((\gamma_2)\) between P and AB participants was not confirmed by our findings. With regard to the wide statistical spread of \((\gamma_2)\) in our groups (P: 0.008-0.114min\(^{-1}\), AB: 0.011-0.095min\(^{-1}\)), the individual fitness level and genetic predisposition might have a much higher influence on blood lactate elimination than being either P or AB status. A wide range of lactate elimination constants (expressed as half-life period: 9.2-18.2min) had already been found in cyclists after exhaustive exercise [Francaux et al., 1989], which supports the hypothesis of interindividual differences in lactate kinetics. Moreover, it has to be taken into account that the mechanisms for production and clearance of lactate may differ between P and AB individuals and that the specific source of lactate elimination can not be determined in the present study. Thus, it remains unknown whether lactate is cleared by muscle above or below the level of lesion in P individuals. Further studies are needed to clarify this issue.

However, considering the similar range of \((\gamma_2)\) in our groups, P participants seem not to be disadvantaged compared with AB participants concerning the trainability of lactate removal: a well-directed training program probably leads to improved blood lactate elimination in both groups. However, the trained P individuals in our study do not represent the general population with thoracic SCI. Our findings may be applicable to paralyzed athletes but not to the large segment of the paralyzed population that is not performing sports regularly. Further investigations are needed to elucidate this issue.

Blood lactate accumulation participants
\(\gamma_1\) was significantly higher in the paralyzed men and thus, the slope of the P group’s curve rises more rapidly in the early stage of recovery than that of the AB group. Although it is not definitely known, one could speculate about a negligible potential for lactate oxidation [Olive et al., 2003] and a reduced potential for lactate storage [Lotta et al., 1991; Spungen et al., 2000] in the leg muscles of our P participants, because all these men were paralyzed for more than 9 years. Reduced oxidative and lactate storage capacities in our P participants’ paralyzed limb muscles could have contributed to the finding of faster blood lactate accumulation in the present study. However, follow-up confirmation is needed to prove our assumptions.

In the late stage of exercise and in the early stage of recovery, AB participants were able to eliminate more lactate from the bloodstream than P participants, resulting in a slower increase of the lactate concentration after cessation of exhaustive arm exercise. The lower gradient of lactate accumulation in AB participants compared with P participants makes the regression lines of the lactate curves converge (Figure 1). However, it is important to note that our P participants’ lactate concentrations are lower throughout the whole recovery period. The difference in \(\gamma_1\) is therefore not big enough to evoke higher Lac\(_{\text{max}}\) in P participants. If this were the case, one could argue that even though \(\gamma_2\) is not significantly different between groups, P participants have to handle higher lactate concentrations and are therefore disadvantaged when compared with AB participants. Regarding the lower Lac\(_{\text{max}}\) in P participants, we conclude that they are not disadvantaged concerning lactate elimination, despite a higher \(\gamma_1\).
Peak values
Compared with AB participants, P participants showed significantly lower values for $VO_{2peak}$, $P_{max}$, and $Lac_{max}$, whereas no differences were found in $HR_{max}$ between groups (Table 2). Previous studies found similar results [Hopman et al., 1993; Jehl et al., 1991], with reduced $VO_{2peak}$ values and lower $Lac_{max}$ in paralyzed individuals compared with able-bodied individuals [Taylor et al., 1986]. However, Borg values at the end of the intensity-graded exercise test indicate a high level of physical exhaustion, which was not different between groups (Table 2). Thus, the lower values for $VO_{2peak}$, $P_{max}$, and $Lac_{max}$ in paralyzed individuals may not be explained by incomplete exhaustion of this group, particularly because no differences in Borg values and $HR_{max}$ were found between groups.

Van Loan et al. [1987] observed reduced cardiorespiratory capacity in paralyzed individuals with higher-level lesions. Because the lesion level was limited in our P participants to those lower than T4 and no differences in heart rate or rating of perceived exertion were found between the P and the AB group, neither the respiratory nor the cardiovascular system seemed to be an exercise limiting factor in the present study, and may not explain the differences in performance between the groups.

Because all of our test participants ate food rich in carbohydrates prior to the exercise test and abstained from exercise for at least 24 hours before the test, the smaller $Lac_{max}$ in P participants cannot be explained by incompletely filled glycogen stores. However, some trunk and leg muscle contraction in AB participants cannot be absolutely excluded. Thus, the resulting larger volume of muscle mass eventually recruited in our AB participants could have led to higher $VO_{2peak}$, $P_{max}$, and $Lac_{max}$. This speculation contrasts with the results of Theisen et al. [2001], who found no differences in $P_{max}$ between paralyzed and able-bodied individuals.

Active recovery
Moderate active recovery was performed to enhance lactate extraction of inactive muscles [Ahlborg et al., 1986], which otherwise stops soon after the cessation of exercise [Lindinger et al., 1990; Poortmans et al., 1978; Kowalchuk et al., 1988]. Moreover, active recovery is a widespread technique in sports and more applicable than passive recovery.

Study participant recruitment
In the present study, participants’ characteristics with respect to age, height, weight, and arm-training status did not differ between groups (Table 1). This is of importance because age-related differences in muscle fiber composition and enzyme activity exist, with younger people having more type-II fibers and increased anaerobic enzyme capacity [Houmard et al., 1998; Larsson et al., 1978; Scott et al., 2001]. Furthermore, muscle fiber composition is plastic and trainable: endurance training leads to an increased ability to lower blood lactate concentrations and an increased proportion of type-I fibers and aerobic enzymes [Schantz et al., 1997; Gladden, 2000], and resistance training is followed by hypertrophy of type-II fibers and enhanced muscle buffer capacity [McComas, 1994; Parkhouse and McKenzie, 1984]. People with SCI are known to have a higher proportion of arm muscle type-I fibers compared with untrained able-bodied individuals [Schantz et al., 1997]. This was an important point to consider during participant recruitment. Thus, all our AB participants had to perform sports in which the arms are used, such as rowing, swimming or cross-country skiing. Activities of
daily living of P participants were not considered training because intensities of such activities are markedly lower than intensities experienced during sport activities and are not known to improve physical fitness [Janssen et al., 1994]. Although no muscle biopsies were performed in the present study, no or only small differences in arm muscle fiber composition were expected between the groups due to careful study participant recruitment.

Conclusions

Participants with paraplegia do not seem to be disadvantaged compared with AB participants concerning blood lactate elimination, even though lactate concentrations after cessation of heavy exercise rose faster in P participants. For both P and AB test participants, the individual fitness level and genetic predisposition might be of greater importance for lactate elimination than their group status. Thus, the time of recovery after maximal bouts of physical arm activity does not have to be prolonged in trained paralyzed individuals to reach lactate recovery levels comparable with those of able-bodied individuals.
Respiratory muscle training in persons with spinal cord injury

Respiratory complications are still the leading cause of death in individuals with SCI [Garshick et al., 2005; van den Berg et al., 2010]. Compared to able-bodied persons mortality based on respiratory problems is three to four times higher in subjects with SCI [Kelley et al., 2003] whereas the reduction in respiratory function seems to depend on the level and completeness of the lesion [Kelley et al., 2003]. The most common complications include dyspnoea, sleep-disordered breathing and pneumonia [Brown et al., 2006]. In this context Haisma and colleagues [2007] stated that the number of pulmonary infections increases with a higher neurological level and completeness of the SCI. Such complications do not only lead to prolonged hospitalization times during the rehabilitation process [Winslow et al., 2002] but also mean a severe loss in quality of life [Tator et al., 1993]. Thus, it seems obvious that one goal of the rehabilitation process in subjects with SCI should be to preserve an optimal functioning of the respiratory system. A promising approach to reach this aim might be a well-directed respiratory muscle training. However, according to recently published review articles [Brooks et al., 2005; van Houtte et al., 2006] the number of randomized controlled trials investigating respiratory muscle training methods in subjects with SCI is limited. As a consequence, van Houtte and co-workers [2006] were not able to perform a meaningful meta-analysis due to the limited number of randomized controlled trials and thus evidence for an overall effect of respiratory muscle training in subjects with SCI could not be confirmed. However, some promising tendencies towards an increased maximal expiratory pressure and forced vital capacity as well as a reduction in residual volume were found after respiratory muscle training programs, whereas no conclusions could be drawn concerning effects on e.g. respiratory complications quality of life, exercise performance or respiratory muscle endurance [van Houtte et al., 2006]. Interestingly, the possibility of a respiratory muscle endurance training by means of normocapnic hyperpnoea was not mentioned in these reviews [Brooks et al., 2005; Van Houtte et al., 2006].

Several studies with able-bodied subjects confirmed positive effects of a normocapnic hyperpnoea training on respiratory muscle endurance as well as on physical exercise performance in trained and untrained subjects [e.g. Boutellier et al., 1992; Boutellier and Piwko, 1992; Spengler and Boutellier, 2000]. However, although normocapnic hyperpnoea training might also be beneficial for individuals with SCI, duration and intensity of such a training regime has to be adapted to meet the needs of this special population. Thus, the first study of this chapter aims to determine the optimal intensity for respiratory muscle endurance training in patients with para- and tetraplegia [Mueller et al., 2006]. In a second step, it seems also interesting to see, if this respiratory training method has some potential in enhancing exercise performance in wheelchair athletes by increasing respiratory and physical endurance exercise capacity [Mueller et al., 2008] or by enhancing the recovery process due to an accelerated blood lactate elimination after exhaustive upper body exercise [Perret and Mueller, 2007]. Finally, the effects of inspiratory muscle training on quality of life, respiratory function and repetitive propulsive sprint performance in wheelchair basketball players was investigated in a further study [Goosey-Tolfrey et al., 2010] of this chapter as well.
3.1 Optimal intensity for respiratory muscle endurance training in patients with spinal cord injury

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Published in: J Rehabil Med 2006; 38: 381-386.

Introduction

Complete spinal cord injury (SCI) results in diminished pulmonary function, due to paralysis of the respiratory muscles, depending on the lesion level [Kelley et al., 2003]. While persons with paraplegia lack abdominal muscle function and also lesion-dependent intercostal muscle function, persons with tetraplegia lack most of the expiratory and even some of the auxiliary inspiratory muscles [De Troyer and Estenne, 1988]. This may lead to more rapid fatigue of the respiratory pump in subjects with SCI during physical activity, as well as to a restricted pulmonary capacity [Silva et al., 1998]. Furthermore, persons with SCI are generally at higher risk for progressive respiratory insufficiency compared with able-bodied people [Lanig and Peterson, 2000]. Pulmonary complications are a major cause of death, in particular for persons with tetraplegia surviving the first 6 months after the trauma [DeVivo et al., 1993]. The fact that patients with SCI activate their remaining respiratory muscles in daily life less than able-bodied persons, due to the lack of whole-body physical activity, may also explain the weakened respiratory system [Monroe et al., 1998], particularly for persons with tetraplegia. Therefore, special attention should be given to the functioning and improvement of their respiratory pump.

Leith and Bradley [1976] showed that respiratory muscle strength and endurance can be specifically increased by appropriate respiratory muscle training in able-bodied subjects. So far, studies showing an improvement of respiratory muscle function in patients with SCI focussed on respiratory resistance or resistance endurance training [Gross et al., 1980; Huldtgren et al., 1980; Rutchik et al., 1998; Wang et al., 2002]. However, for these patients, respiration causes major problems during physical activity [Wien et al., 1999] and for patients with tetraplegia also during the night (e.g. sleep apnoea) [Burns et al., 2001; Burns et al., 2000; McEvoy et al., 1995; Saikov et al., 1998]. Therefore, respiratory endurance seems to be more critical than respiratory muscle strength. Sedentary as well as trained able-bodied subjects [Spengler and Boutellier, 2000] significantly increased the endurance of respiratory muscles as well as whole-body exercise endurance by means of normocapnic hyperpnoea training (NHT) at 60-70% of their individual maximal voluntary ventilation (MVV). In view of these effects in able-bodied subjects, NHT is expected to be even more beneficial for persons with weak respiratory muscles, such as people with SCI, but data is missing for this group.
Therefore, the first step to adapt NHT for persons with SCI is to test respiratory endurance in this group of subjects at different minute ventilations, i.e. different intensities. We hypothesize that, due to the partly lacking respiratory muscle mass, the minute ventilation that can be sustained for 10-20 minutes, an appropriate duration for NHT in this group of patients, will be higher for patients with paraplegia than for patients with tetraplegia. To test this hypothesis, patients with paraplegia as well as tetraplegia performed respiratory endurance tests (RETs) by means of exhaustive normocapnic hyperpnoea at three different intensities.

Methods

Subjects
Thirty-three patients, 22 with paraplegia and 11 with tetraplegia, who were hospitalized in an SCI rehabilitation centre for first rehabilitation between September 2001 and July 2004, met the inclusion criteria (see below). Of these, 3 did not want to participate and 16 were excluded according to the exclusion criteria (see below). Finally, 8 patients with paraplegia and 6 patients with tetraplegia were enrolled in the study. Characteristics of these subjects are shown in Table 1.

Table 1: Characteristics of patients with paraplegia and tetraplegia

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age [years]</th>
<th>Height [cm]</th>
<th>Weight [kg]</th>
<th>TPI [mt]</th>
<th>Lesion level</th>
<th>Smoker</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paraplegia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>m</td>
<td>30</td>
<td>180</td>
<td>75</td>
<td>6</td>
<td>Th4</td>
<td>no</td>
</tr>
<tr>
<td>m</td>
<td>22</td>
<td>178</td>
<td>62</td>
<td>4</td>
<td>Th5</td>
<td>no</td>
</tr>
<tr>
<td>f</td>
<td>26</td>
<td>168</td>
<td>53</td>
<td>4</td>
<td>Th5/6</td>
<td>no</td>
</tr>
<tr>
<td>m</td>
<td>23</td>
<td>198</td>
<td>83</td>
<td>4</td>
<td>Th6</td>
<td>yes</td>
</tr>
<tr>
<td>m</td>
<td>39</td>
<td>170</td>
<td>80</td>
<td>5</td>
<td>Th10</td>
<td>no</td>
</tr>
<tr>
<td>m</td>
<td>26</td>
<td>182</td>
<td>72</td>
<td>4</td>
<td>Th11</td>
<td>no</td>
</tr>
<tr>
<td>m</td>
<td>32</td>
<td>185</td>
<td>70</td>
<td>4</td>
<td>Th11</td>
<td>no</td>
</tr>
<tr>
<td>m</td>
<td>45</td>
<td>178</td>
<td>73</td>
<td>4</td>
<td>L1</td>
<td>no</td>
</tr>
<tr>
<td>Mean±SD</td>
<td>30.8 ± 7.9</td>
<td>179.9 ± 9.3</td>
<td>71 ± 9.7</td>
<td>4.4 ± 0.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tetraplegia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>f</td>
<td>22</td>
<td>173</td>
<td>52</td>
<td>7</td>
<td>C4</td>
<td>no</td>
</tr>
<tr>
<td>f</td>
<td>39</td>
<td>153</td>
<td>41</td>
<td>6</td>
<td>C5</td>
<td>no</td>
</tr>
<tr>
<td>m</td>
<td>21</td>
<td>187</td>
<td>70</td>
<td>6</td>
<td>C6</td>
<td>no</td>
</tr>
<tr>
<td>m</td>
<td>21</td>
<td>183</td>
<td>59</td>
<td>7</td>
<td>C7</td>
<td>yes</td>
</tr>
<tr>
<td>m</td>
<td>22</td>
<td>175</td>
<td>69</td>
<td>7</td>
<td>C7</td>
<td>no</td>
</tr>
<tr>
<td>m</td>
<td>22</td>
<td>172</td>
<td>58</td>
<td>6</td>
<td>C7</td>
<td>no</td>
</tr>
<tr>
<td>Mean±SD</td>
<td>24.5 ± 7.1</td>
<td>173.8 ± 11.8</td>
<td>58.2 ± 10.9</td>
<td>6.5 ± 0.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

TPI: time post injury; m: male; f: female; C: cervical lesion; Th: thoracic lesion; L: lumbar lesion ; SD: standard deviation
Inclusion criteria were: age between 18 and 45 years with an acute traumatic, motor complete lesion (ASIA A or B). Exclusion criteria were: asthma, chronic obstructive pulmonary disease, pneumonia, tracheotomy, bronchial or urinary infections, irregular medication, peanut or latex allergy and epilepsy. In order to reach stable ventilatory function after injury, we enrolled patients with paraplegia 4-6 months after injury and patients with tetraplegia 6-8 months after injury [Anke et al., 1993; Liaw et al., 2000]. Prior to the start of the study, all patients were informed in detail about the study and gave their written informed consent. The ethics committee of the canton Lucerne, Switzerland approved the study.

Equipment
Vital capacity, peak inspiratory and expiratory flows, forced in- and expiratory volumes in 1s (FIV₁, FEV₁) and MVV were measured by a metabolic chart (Oxycon α, Jaeger, Höchberg, Germany), using a turbine for volume measurements and fast-responding gas analysers. The system had been calibrated with a 3-litre syringe and certified mixed calibration gas before each test. Maximal inspiratory and expiratory pressures (Piₘₐₓ, Peₘₐₓ) were measured by a hand-held mouth pressure meter (Micro MPM, Micro Medical Ltd, Chatham Kent, UK) including a small air leak to prevent glottis closure.

RETs, i.e. normocapnic hyperpnoea to exhaustion, were performed using a special partial re-breathing device (SpiroTiger®, Fehraltorf, Switzerland) providing target respiratory frequency and tidal volume. During RETs, end-tidal CO₂ partial pressure and ventilatory variables were monitored and recorded breath by breath (Oxycon α) to verify normocapnia and target ventilation. Heart rate (HR) was measured by oximetry (OxyTip, Datex-Ohmeda 3900, Louisville, USA) on the right middle finger and blood pressure (BP) by plethysmography (Finapress BP Monitor, Ohmeda 2300, BOC Healthcare, Englewood, Colorado, USA) on the left middle finger, placing the left forearm on heart level (see Fig. 1 for experimental setup).

Figure 1: Experimental setup.
Blood samples (20ml) were drawn from an earlobe and analysed for blood lactate concentration enzymatically (Super GL Ambulance, Ruhrtal Labor Technik, Mönchengladbach, Germany). Perception of breathlessness and respiratory effort were indicated by the subjects at rest and at the end of each test on a visual analogue scale (VAS) ranging from 0 to 10.

Protocol

Every patient reported 6 times to the laboratory, with an interval between visits of at least 72 hours. During the first 3 sessions, lung function as well as $P_{\text{Imax}}$ (from residual volume) and $P_{\text{Emax}}$ (from total lung capacity), both measures of respiratory muscle strength, were assessed. Each measurement was repeated 3-6 times until values did not differ more than ±5%. The best effort within these limits was recorded. All respiratory measurements were conducted according to the spirometry testing standards in SCI [Kelley et al., 2003] in an upright sitting position in the patient’s own wheelchair. After lung function and respiratory muscle strength measurements, the NHT device was adjusted to 1 of the 3 intensities corresponding to 20%, 40% or 60% of each patient’s individual MVV. Subjects were then familiarized with the technique to perform normocapnic hyperpnoea and they were also trained to use the VAS.

During the last 3 sessions, subjects performed 1 RET each day at either 20%, 40% or 60% of their MVV, in random order. During RET sessions, they were verbally encouraged to keep the target minute ventilation ($V_E$) of the corresponding intensity. The test was stopped either by the patient due to exhaustion or by the experimenter, if $V_E$ was more than 5l·min$^{-1}$ lower than the target value for more than 30 seconds or if the test duration reached 60 minutes. Patients had to abstain from caffeine for 24 hours before each test. Information about nutrition, sleep, physical activity and medication 24 hours prior to each test were given by a questionnaire.

HR and BP were registered at rest, every 2 minutes during and at the end of each test. Blood samples for blood lactate analyses were drawn at the same time-points. BP measurements were performed in patients with paraplegia only, as sympathetic activity regulating BP is absent in patients with tetraplegia and thus BP is influenced by other factors (e.g. micturition) causing random fluctuations [Teasell et al., 2000].

Statistics

Between-group differences in respiratory muscle endurance, absolute $V_E$ during RETs, breathlessness, respiratory effort, HR, BP and blood lactate concentration, were tested using Wilcoxon’s rank sum tests. Systolic vs. diastolic increases in BP between rest and test break off were also tested with Wilcoxon’s rank sum tests. The Friedman two-way analysis of variance was used to assess within-group differences of RET variables between tests at the three different intensities. For HR, BP, breathlessness and respiratory effort, differences between rest and test break off were calculated for further analyses. Significance was accepted at p<0.05. Statistical analyses were performed with a computer software package (Systat, Version 10.2; Systat Software Inc.; Point Richmond, CA, USA). Values are presented as mean (SD).
Results

Spirometry and respiratory muscle strength
Lung function and respiratory muscle strength of patients with paraplegia and tetraplegia are presented in Table 2. All variables were significantly higher in patients with paraplegia compared with tetraplegia.

RET: duration and respiratory assessments
RET duration at 60% MVV was significantly higher in patients with paraplegia compared with patients with tetraplegia being 12.2±9.0 vs 4.2±3.4 minutes (Fig. 2). Durations of RETs at 40% MVV (38.8 vs 18.9 minutes; p=0.053) and 20% MVV (51.8 vs. 46.0 minutes; p=0.391) did not differ significantly between groups. Within groups, RET durations were significantly different between intensities (p=0.006 for both groups).

Table 2: Lung function and respiratory muscle strength (% predicted) for patients with paraplegia and tetraplegia presented as mean ± standard deviation

<table>
<thead>
<tr>
<th></th>
<th>Patients with paraplegia (n = 8)</th>
<th>Patients with tetraplegia (n = 6)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>VC</td>
<td>100.5 ± 22.4</td>
<td>59.3 ± 21.9</td>
<td>0.007</td>
</tr>
<tr>
<td>FEV\textsubscript{1}</td>
<td>100.9 ± 22.6</td>
<td>58.2 ± 18.6</td>
<td>0.007</td>
</tr>
<tr>
<td>FIV\textsubscript{1}</td>
<td>104.8 ± 26.3</td>
<td>60.8 ± 21.5</td>
<td>0.012</td>
</tr>
<tr>
<td>PEF</td>
<td>106.5 ± 23.5</td>
<td>57.0 ± 12.0</td>
<td>0.002</td>
</tr>
<tr>
<td>MVV</td>
<td>127.4 ± 32.7</td>
<td>68.0 ± 19.0</td>
<td>0.003</td>
</tr>
<tr>
<td>P\textsubscript{max}</td>
<td>81.4 ± 14.6</td>
<td>59.0 ± 14.4</td>
<td>0.010</td>
</tr>
<tr>
<td>P\textsubscript{e max}</td>
<td>42.3 ± 18.1</td>
<td>20.7 ± 5.5</td>
<td>0.004</td>
</tr>
</tbody>
</table>

VC: vital capacity; FEV\textsubscript{1}: forced expiratory volume in 1 s; FIV\textsubscript{1}: forced inspiratory volume in 1 s; PEF: peak expiratory flow; MVV: maximal voluntary ventilation; P\textsubscript{max}: maximal inspiratory pressure; P\textsubscript{e max}: maximal expiratory pressure

At 20% MVV, 6 patients with paraplegia and 2 with tetraplegia were stopped by the experimenter as they reached 60 minutes. Due to the higher absolute MVV of patients with paraplegia, their average V\textsubscript{E} during RETs was significantly higher at 20%, 40% and 60% MVV compared with patients with tetraplegia (Fig. 3).

Within both groups, V\textsubscript{E} was significantly different between intensities (p<0.001 for patients with paraplegia and p=0.007 for patients with tetraplegia). Perception of breathlessness did not differ between intensities for patients with paraplegia and tetraplegia (p=0.159 and p= 0.449). In patients with paraplegia, perception of respiratory effort was significantly higher at the end of RETs at 60% (p=0.005) and 40% MVV (p=0.034) than at the end of the RET at 20% MVV while it did not differ between intensities in patients with tetraplegia (p=0.368) (Table 3).
Figure 2: Median (lines inside boxes) and lower and upper quartiles (lower and upper edge of boxes) and range (whiskers) for duration of respiratory endurance tests at different intensities for patients with paraplegia (PP) and for patients with tetraplegia (TP). MVV= maximal voluntary ventilation, o= outlier.

Figure 3: Median (lines inside boxes) and lower and upper quartiles (lower and upper edge of boxes) and range (whiskers) of minute ventilation during respiratory endurance tests at different intensities for patients with paraplegia (PP) and for patients with tetraplegia (TP). MVV= maximal voluntary ventilation.
Table 3: Differences (Δ) between rest and end of respiratory endurance tests at different intensities for patients with paraplegia and tetraplegia presented as mean ± standard deviation.

<table>
<thead>
<tr>
<th>Intensity (% MVV)</th>
<th>Patients with paraplegia (n = 8)</th>
<th>Patients with tetraplegia (n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>20%</td>
<td>40%</td>
</tr>
<tr>
<td>Δ heart rate [bpm]</td>
<td>11±11</td>
<td>30±14</td>
</tr>
<tr>
<td>Δ systolic blood pressure [mmHg]</td>
<td>11.6±24.5</td>
<td>24.1±24.3</td>
</tr>
<tr>
<td>Δ diastolic blood pressure [mmHg]</td>
<td>14.1±10.5</td>
<td>16.9±9.7</td>
</tr>
<tr>
<td>Δ breathlessness [points]</td>
<td>5.01±3.31</td>
<td>4.39±3.20</td>
</tr>
<tr>
<td>Δ respiratory effort [points]</td>
<td>4.06 ±2.47</td>
<td>7.17±3.06</td>
</tr>
</tbody>
</table>

Symbols: *Significant difference to 20% MVV; † Significant difference to 40% MVV; Note that there were no significant differences between groups of para- and tetraplegic patients.

RET: cardiovascular measurements
At rest, mean HR was 91±12 bpm in patients with paraplegia and 74±14 bpm in patients with tetraplegia. While BP was not assessed in patients with tetraplegia (for details please see methods), in patients with paraplegia resting systolic BP was 134±28 mmHg and diastolic BP was 74±11 mmHg. Increases in systolic BP between intensities were not significantly different (p=0.159). Diastolic BP increased significantly more during RETs at 60% compared with 20% MVV (p=0.005) and during 60% compared with 40% MVV (p=0.034). Increases in diastolic BP between 20% and 40% MVV were not significantly different (Table 3). Furthermore, at 60% MVV the increase in systolic BP, was significantly larger than the increase in diastolic BP (p=0.012) while increases in systolic vs. diastolic BP did not differ at 40% (p=0.483) and 20% MVV (p=0.726).

RET: blood lactate concentration
Blood lactate concentrations of the 3 tests are shown in Fig. 4A for patients with paraplegia and Fig. 4B for patients with tetraplegia. In patients with paraplegia, blood lactate concentrations during as well as at the end of RETs decreased significantly compared with resting levels at 20% MVV (p=0.005) as well as at 40% MVV (p=0.034). At 60% MVV, blood lactate concentration did not change throughout the test (Fig. 4A). In patients with tetraplegia, blood lactate concentrations decreased significantly compared with resting values during as well as at the end of the RET at 20% MVV only (p=0.014). At 40% and 60% MVV, blood lactate concentration did not change throughout the test (Fig. 4B). Blood lactate concentrations did not differ between groups at identical intensities (p=0.180-0.564).
Discussion

Respiratory muscle endurance and optimal training intensity

This study confirms our hypothesis that respiratory muscle endurance is reduced in patients with SCI compared with able-bodied subjects, more so with higher lesion levels. While able-bodied subjects can breathe for 30 minutes at >60% of their individual MVV [Boutellier et al., 1992; Boutellier and Piwko, 1992; Spengler et al., 1999], patients with paraplegia only sustained this intensity for an average of 12.2 minutes while patients with tetraplegia only reached 4.2 minutes on average. Thus, when aiming for NHT durations of 10-20 minutes (as proven feasible for respiratory training in this group of patients) [Derrickson et al., 1992; Hultgren et al., 1980; Loveridge et al., 1999; Rutchik et al., 1998; Uijl et al., 1999; Wang et al., 2002], $V_E$ can be set around 60% MVV for patients with paraplegia, but needs to be reduced to around 40% MVV for patients with tetraplegia. Although the perception of breathlessness was quite similar at the end of all tests in both groups, motivation to perform RETs may also have influenced performance in these patients with SCI, shown by rather large inter-individual differences of RET durations at given intensities. The above-mentioned guidelines should therefore be taken as starting levels in order to adjust the individual optimal training intensity.

It is noteworthy that respiratory endurance of patients with SCI is decreased compared with able-bodied subjects, although the $V_E$ values were based on individual subjects’ MVV [McMahon et al., 2002]. For patients with tetraplegia, MVV was significantly below that of able-bodied subjects. The decreased endurance may reflect a certain degree of muscle atrophy, occurring due to the immobilization of the patients during the first weeks after injury [Bunham et al., 1997]. Furthermore, in patients with SCI, breathing and thus respiratory muscles are stimulated only little during daily activities due to the smaller muscle mass available compared with able-bodied persons that are walking, running and lifting objects.
[Monroe et al., 1998]. In addition, the potential of shifting between the use of different respiratory muscles allowing some recovery at submaximal ventilations is reduced.

Lung function and respiratory muscle strength
While lung function of patients with paraplegia was similar to able-bodied subjects, respiratory muscle strength was lower than normal with $P_{l_{\text{max}}}$ being around 20% and $P_{e_{\text{max}}}$ around 60% lower than predicted from values assessed in able-bodied persons. In patients with tetraplegia, however, lung function and inspiratory muscle strength were reduced to about 60% of predicted while expiratory muscle strength was only around 20% predicted. As hyperpnoea consists of repeated forced inspirations and expirations, maximal strength of these muscles (and the loss of it in the course of the task) is likely a factor affecting endurance as well. This assumption is supported by the fact that respiratory muscle endurance was reduced in subjects with paraplegia in the presence of reduced muscle strength and despite normal lung function, including $F_{I_{\text{V}}}$, $F_{E_{\text{V}}}$, and MVV.

Cardiovascular responses to RETs
While BP was not assessed in patients with tetraplegia due to the sympathetic autonomic impairment at this lesion level [Figoni, 1993], both systolic and diastolic BP increased significantly in patients with paraplegia during RETs with a tendency to larger changes with increasing $V_{E}$. Also, systolic BP increased to a larger extent than diastolic BP at 60% MVV, suggesting that stroke volume increased during this task. This cardiovascular challenge, in particular during breathing at 60% MVV, strongly suggests that respiratory endurance training at this intensity will result in significant training effects.

Blood lactate metabolism
Interestingly, blood lactate concentrations decreased during RETs at 20% and 40% MVV for patients with paraplegia and at 20% MVV for patients with tetraplegia, probably meaning that working muscles increasingly consumed blood lactate as an energy source. This shift in the balance of lactate production vs. consumption relative to resting conditions may result from the increased activity of respiratory muscles also using blood lactate as a fuel and/or the increased activity of the heart muscle, one of the major blood lactate consumers. The fact that blood lactate did not change during breathing at 60% MVV in patients with paraplegia and at 40% and 60% in patients with tetraplegia shows, in turn, that anaerobic metabolism was increasingly needed to achieve the work, also showing that patients with tetraplegia reached this limit “earlier”, i.e. at a lower intensity than patients with paraplegia. This, in turn, supports our previous suggestion that the higher the lesion, the smaller the ability of the still innervated respiratory muscles to share the work and to recover in-between. The continuous work then likely results in a faster development of fatigue and the necessity to recruit fast twitch fibres running on anaerobic metabolism.

These metabolic changes during RETs, together with the cardiovascular changes observed, suggest that respiratory muscle endurance training - if performed in form of NHT at 60% MVV (patients with paraplegia) and 40% (patients with tetraplegia) - will result in significant training effects, e.g. increased respiratory muscle endurance probably accompanied by increased aerobic metabolism. Increased respiratory muscle endurance will, in turn, not only reduce the sensation of breathlessness during tasks involving heavy breathing, e.g. wheeling
uphill, but also the shift towards increased aerobic metabolism (as observed in able-bodied people after this kind of training [Spengler et al., 1999]) might help to reduce early arm muscle fatigue resulting from intramuscular acidosis. In addition, lung function of patients with tetraplegia is likely to improve, which would then result in improved airway clearance and reduction of pulmonary complications.

Conclusions

In conclusion, this study showed that respiratory muscle endurance of patients with tetraplegia is reduced compared with patients with paraplegia. The optimal intensity for patients with SCI to perform NHT for 10-20 minutes should be set at $V_E$ around 60% MVV for patients with paraplegia and around 40% MVV for patients with tetraplegia. Due to inter-individual differences, however, this intensity should then be adapted individually using the above-mentioned values as guidelines.
3.2 Effects of respiratory muscle endurance training on wheelchair racing performance in athletes with paraplegia

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²Institute of Sports Medicine, Swiss Paraplegic Centre, Nottwil, Switzerland  
³Medical Centre, Radboud University, Nijmegen, The Netherlands


Introduction

Thirty minutes of respiratory muscle endurance training (RMET), 5 times a week for 4 consecutive weeks, increased respiratory muscle endurance more than 450% and leg cycling exercise time at the anaerobic threshold by 38% in trained able-bodied subjects [Boutellier et al., 1992]. The effects of RMET on upper body exercise performance have not yet been evaluated. Wheelchair racing athletes with paraplegia, using muscles of the upper extremities for breathing and locomotion concurrently, seem to be an ideal group to study the effects of RMET on upper body endurance exercise performance. We hypothesized that RMET would improve ventilatory muscle endurance and upper body exercise performance on a 10km time-trial in athletes with paraplegia. If this is the case RMET might also offer an interesting option for the rehabilitation of subjects with spinal cord injury.

Methods

Twelve competitive wheelchair racing athletes with paraplegia participated in the study. Subjects were divided as matched pairs with respect to their lesion level and allocated randomly either to the training group or to the control group by toss of a coin (Table 1). The local ethics committee approved the study and subjects provided written informed consent.

At the study’s inception, all subjects reported to the laboratory on four separate occasions for baseline testing, which included lung function measurements, a VO₂peak test, a 10km time trial, and a respiratory muscle endurance test. Thereafter, training group subjects performed RMET by means of normocapnic hyperpnoea for 30 training sessions of 30min each (5 sessions per week for 6 consecutive weeks), while control group subjects performed no respiratory muscle training. Otherwise, both groups followed their usual wheelchair training throughout the study. After 6 weeks, the 4 baseline testing sessions were repeated in the same order.
Table 1: Anthropometric and training data

<table>
<thead>
<tr>
<th>Group</th>
<th>Sex</th>
<th>Height [cm]</th>
<th>Weight [kg]</th>
<th>Age [y]</th>
<th>Lesion level</th>
<th>ASIA score</th>
<th>Lesion since [y]</th>
<th>Training [h·wk⁻¹]</th>
<th>Training since [y]</th>
</tr>
</thead>
<tbody>
<tr>
<td>T</td>
<td>m</td>
<td>178</td>
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<td>24</td>
</tr>
<tr>
<td>T</td>
<td>w</td>
<td>150</td>
<td>40</td>
<td>34</td>
<td>T6</td>
<td>A</td>
<td>24</td>
<td>5</td>
<td>12</td>
</tr>
<tr>
<td>T</td>
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<td>67</td>
<td>29</td>
<td>T6</td>
<td>A</td>
<td>1.5</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>T</td>
<td>w</td>
<td>170</td>
<td>46</td>
<td>18</td>
<td>L1</td>
<td>C</td>
<td>4</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>T</td>
<td>m</td>
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<td>31</td>
<td>L1</td>
<td>C</td>
<td>10</td>
<td>7.5</td>
<td>7</td>
</tr>
<tr>
<td>T</td>
<td>m</td>
<td>173</td>
<td>60</td>
<td>18</td>
<td>L3</td>
<td>C</td>
<td>18</td>
<td>8.5</td>
<td>6</td>
</tr>
<tr>
<td>C</td>
<td>m</td>
<td>173</td>
<td>60</td>
<td>40</td>
<td>T5</td>
<td>A</td>
<td>18</td>
<td>8.5</td>
<td>16</td>
</tr>
<tr>
<td>C</td>
<td>m</td>
<td>165</td>
<td>65</td>
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<td>T6</td>
<td>A</td>
<td>13</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>C</td>
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<td>27</td>
<td>L3</td>
<td>C</td>
<td>27</td>
<td>6.5</td>
<td>13</td>
</tr>
</tbody>
</table>

Mean±SD  170±13  54±10  29±10   14±10  7.5±3.6  9±9

Mean±SD  166±6  60±7  28±10   17±7  6.3±2.1  9±5

m: man; w: woman; L: lumbar lesion; T: thoracic lesion; ASIA: American Spinal Injury Association; SD: standard deviation. Note that there were no significant differences between groups.

All athletes completed the VO₂peak test and the 10km time-trial using their own racing wheelchair. They performed the VO₂peak test on a treadmill (Saturn HP Cosmos, Munich, Germany) and started at a speed of 10 km/hour and an inclination of 0.7%. Every 3min, speed increased by 2km·h⁻¹ until volitional exhaustion.

The athletes performed the 10km time trial on a nearly frictionless training roller (Spinner, New Hall’s Wheels, Cambridge, MA). No information was given to the athletes during the test other than notification of each accomplished kilometer. Subjects simply had to complete 10km as fast as possible.

Subjects performed the respiratory muscle endurance test with the RMET device (SpiroTiger Medical, Idiag AG, Fehraltorf, Switzerland). Breathing frequency was adjusted to reach target minute ventilation (Vₑ), which could be maintained for 5 to 10min during pretests, corresponding to an intensity of 65% to 75% of the individual maximal voluntary ventilation value. Either the experimenter aborted the test if target Vₑ could not be maintained for more than 30s after indication or the athlete himself stopped the test due to exhaustion.

We used Wilcoxon’s rank-sum tests to evaluate within groups differences between pre- and postintervention values and Mann-Whitney-U tests to calculate between-group differences on the pre-post change scores. Significance was set at P<0.05. Statistical analyses were performed with a computer software package (Version 10.2; Systat Software; Point Richmond, CA).
Results

Respiratory muscle endurance increased significantly by 332% in the training group (P = 0.028) after RMET, while the control group did not show any change in muscle endurance. A significant difference existed between groups (P = 0.016; Table 2).

Table 2: Values during respiratory muscle endurance tests (RMET)

<table>
<thead>
<tr>
<th></th>
<th>Training group (n=6)</th>
<th>Control group (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>Time of RMET [min]</td>
<td>9.1 ± 7.2</td>
<td>39.9 ± 17.8*</td>
</tr>
<tr>
<td>Mean HR [bpm]</td>
<td>124 ± 13</td>
<td>122 ± 11</td>
</tr>
<tr>
<td>Mean $V_e$ [l·min$^{-1}$]</td>
<td>91 ± 28</td>
<td>91 ± 28</td>
</tr>
<tr>
<td>Mean tidal volume [l]</td>
<td>2.52 ± 0.90</td>
<td>2.58 ± 0.90</td>
</tr>
<tr>
<td>Mean BF [min$^{-1}$]</td>
<td>37 ± 4</td>
<td>36 ± 5</td>
</tr>
<tr>
<td>Mean end tidal $CO_2$ [kPa]</td>
<td>3.9 ± 0.7</td>
<td>3.7 ± 1.3</td>
</tr>
</tbody>
</table>

Data are means ± standard deviation. Note that mean values are calculated over the whole test duration. HR: heart rate; $V_e$: minute ventilation; BF: breathing frequency; *Significant between-group difference; †Significant within-group difference.

Table 3: Lung function measurements by body plethysmography

<table>
<thead>
<tr>
<th></th>
<th>Training group (n=6)</th>
<th>Control group (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>TLC [%]</td>
<td>89.2 ± 14.9*</td>
<td>90.7 ± 21.8</td>
</tr>
<tr>
<td>RV [%]</td>
<td>171.6 ± 31.0</td>
<td>176.5 ± 35.7</td>
</tr>
<tr>
<td>FVC [%]</td>
<td>85.3 ± 29.0</td>
<td>88.5 ± 31.3</td>
</tr>
<tr>
<td>FEV$_1$ [%]</td>
<td>84.5 ± 26.6</td>
<td>87.4 ± 28.4</td>
</tr>
<tr>
<td>FEV$_1$ / FVC [%]</td>
<td>102.1 ± 10.8*</td>
<td>102.7 ± 11.2</td>
</tr>
<tr>
<td>PEF [%]</td>
<td>76.8 ± 20.6*</td>
<td>83.3 ± 28.1</td>
</tr>
<tr>
<td>MVV [%]</td>
<td>124.8 ± 61.9</td>
<td>134.4 ± 66.7</td>
</tr>
<tr>
<td>P$_{i\max}$ [%]</td>
<td>90.8 ± 29.3</td>
<td>102.3 ± 17.7</td>
</tr>
<tr>
<td>P$_{e\max}$ [%]</td>
<td>46.4 ± 11.3</td>
<td>59.8 ± 11.2†</td>
</tr>
</tbody>
</table>

Data are mean ± standard deviation of percents predicted for able-bodied persons. Note that there were no significant differences in changes from pre to post between groups.
TLC: total lung capacity; RV: residual volume; FVC: forced vital capacity; FEV$_1$: forced expiratory volume in 1s; PEF: peak expiratory flow; MVV: maximal voluntary ventilation; P$_{i\max}$: maximal inspiratory muscle strength; P$_{e\max}$: maximal expiratory muscle strength; *Significant between-group difference (baseline testing); †Significant within-group difference.
Lung function values and $P_{\text{max}}$ did not change between pre- and post-tests within or between groups. $P_{\text{e,max}}$ increased significantly by 25% within the training group ($P = 0.028$) but showed no significant differences within the control group nor between groups (Table 3).

The 10km time-trial performance showed significant within-group differences for the training group ($P = 0.046$) but not for the control group (Table 4). Even if between groups comparisons were not significantly different ($P = 0.150$), the effect size of 10km time-trial performance was high ($d = 0.87$).

Table 4: 10km time-trial data

<table>
<thead>
<tr>
<th></th>
<th>Training group (n=6)</th>
<th>Control group (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>Time of 10km time trial</td>
<td>27.1 ± 9.0</td>
<td>24.1 ± 6.6†</td>
</tr>
<tr>
<td>Mean $\text{VO}<em>2$ as % of $\text{VO}</em>{2\text{peak}}$</td>
<td>71.5 ± 9.9</td>
<td>77.1 ± 12.2</td>
</tr>
<tr>
<td>Mean heart rate [bpm]</td>
<td>161 ± 13</td>
<td>168 ± 15</td>
</tr>
<tr>
<td>Mean minute ventilation</td>
<td>52.6 ± 18.6*</td>
<td>64.6 ± 27.9</td>
</tr>
<tr>
<td>Mean tidal volume [l]</td>
<td>1.28 ± 0.33</td>
<td>1.47 ± 0.51†</td>
</tr>
<tr>
<td>Respiratory equivalent for $\text{O}_2$</td>
<td>30.6 ± 2.3*</td>
<td>28.5 ± 3.9</td>
</tr>
<tr>
<td>Mean breathing frequency</td>
<td>41.5 ± 11.6</td>
<td>44.8 ± 13.2</td>
</tr>
<tr>
<td>Mean end tidal $\text{CO}_2$ [kPa]</td>
<td>3.3 ± 0.2</td>
<td>3.3 ± 0.3</td>
</tr>
</tbody>
</table>

Data are means ± standard deviation. Note that mean values are calculated over the whole test duration.

$\text{VO}_2$: oxygen uptake; *Significant between-group difference; †Significant within-group difference

Peak oxygen consumption (Table 5) showed no significant differences before versus after intervention for within- or between-group comparisons.

Table 5: $\text{VO}_{2\text{peak}}$-test values

<table>
<thead>
<tr>
<th></th>
<th>Training group (n=6)</th>
<th>Control group (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>$\text{VO}_{2\text{peak}}$ [ml·min$^{-1}$·kg$^{-1}$]</td>
<td>41.2 ± 10.6</td>
<td>40.3 ± 6.8</td>
</tr>
<tr>
<td>$\text{HR}_{\text{peak}}$ [bpm]</td>
<td>185 ± 16</td>
<td>184 ± 14</td>
</tr>
<tr>
<td>$V_{E\text{peak}}$ [l·min$^{-1}$]</td>
<td>96 ± 31</td>
<td>94 ± 30</td>
</tr>
<tr>
<td>Peak tidal volume [l]</td>
<td>1.97 ± 0.61</td>
<td>1.96 ± 0.56</td>
</tr>
<tr>
<td>Peak breathing frequency</td>
<td>60 ± 13</td>
<td>62 ± 14</td>
</tr>
</tbody>
</table>

Note that there were no significant differences in changes from pre to post between groups.

$\text{VO}_{2\text{peak}}$: peak oxygen uptake; $\text{HR}$: heart rate; $V_{E}$: minute ventilation
Discussion

This study shows that RMET has a positive effect on respiratory muscle endurance, while there is no effect of RMET on maximal exercise performance ($VO_{2peak}$) in wheelchair racing athletes. Our results provide first evidence that RMET increases upper extremity exercise performance, showing significant within training group decreases in exercise time on a 10km wheelchair racing time trial. Results of the 10km time trial should be considered with care because between-group difference was not statistically significant due to large interindividual differences and the small size of the groups tested.

A spinal cord injury (SCI) causes lesion dependent functional loss of respiratory muscles [Haas et al., 1965; Ohry et al., 1975]. Nevertheless, lung function in our SCI subjects was normal, but $P_{max}$ was severely decreased (Table 3). Interestingly, $P_{max}$ significantly increased within the training group after RMET. Thus, RMET seems to offer an interesting option for the rehabilitation of subjects with SCI to improve expiratory muscle strength. Our results further show that RMET improves respiratory muscle endurance in athletes with paraplegia to a similar extent as in able-bodied individuals [Boutellier et al., 1992; Boutellier and Piwko, 1992].

Training group subjects showed a significant within-group difference of an 11% mean decrease in time over a 10km time trial. This result is of high practical relevance for wheelchair racing competitions.

During upper body exercise, similar muscles are innervated for movement and breathing. Therefore, these muscles are concurrently used. It has been shown that the ventilatory pattern changes during upper-body exercise and exercise endurance is decreased compared to leg exercise [Celli et al., 1988]. Consequently, improvements in respiratory muscle endurance may have a positive influence on upper-body exercise performance. The lesion dependent differences in the amount of innervated upper-body muscle mass differ among athletes and may therefore provide a source of variation in exercise performance and in potential to increase upper-body endurance performance.

Conclusions

This study shows that 6 weeks of RMET increases respiratory muscle endurance in wheelchair racing athletes. Because there was a large observed effect size in the 10km time trial of $d = 0.87$, there is evidence that 6 weeks of RMET may also improve upper-body exercise performance.
3.3 Effects of inspiratory muscle training on respiratory function and repetitive sprint performance in wheelchair basketball players

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²Institute of Exercise & Sport Science, Manchester Metropolitan University, Cheshire, Alsager, UK
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⁴Institute for Biomedical Research into Human Movement and Health, Manchester Metropolitan University, Cheshire, UK


Introduction

It has been found that inspiratory muscle training (IMT) among able-bodied athletes increases respiratory muscle strength and delays respiratory muscle fatigue and the onset of breathlessness [Holm et al., 2004; Inbar et al., 2000; Lomax and McConnell, 2003; Romer et al., 2002a; Romer et al., 2002b; Wells et al., 2005; Williams et al., 2002]. Respiratory muscle training has proven to be beneficial for respiratory function in patients with chronic diseases [Mancini et al., 1995; Sanchez Riera et al., 2001; Van Houtte et al., 2006; Weiner et al., 1992] and patients during the early rehabilitation stages of spinal cord injury (SCI) [Liaw et al., 2000].

To date, in terms of disability athletic populations, only one study using a slightly different training method to IMT investigated the benefits of respiratory muscle endurance training in athletes with paraplegia [Mueller et al., 2008]. In that study of Mueller and colleagues [2008] significant improvements were found in respiratory muscle endurance after 6 weeks of respiratory muscle endurance training but not in 10km performance. No research has investigated whether these noted benefits are transferable to wheelchair game players, using methods similar to the work of Romer and coworkers [2002a] who found improvements in the recovery time of able-bodied athletes during high-intensity and intermittent exercise. Interestingly, along with those wheelchair sports of a repetitive sprint nature, this type of exercise seems to correspond much better to daily life activities of the general wheelchair user.

Wheelchair basketball is a popular team sport for wheelchair users with paraplegic conditions that include SCI, spina bifida and poliomyelitis. These disabilities generally affect the lower limbs of the body but, depending on the severity of the disorder and the level of the lesion, can also affect the upper limbs and the respiratory system. Because normal respiratory function involves the respiratory musculature of chest, back and abdominal area, this could account for the feelings of dyspnoea in wheelchair athletes compared with able-bodied counterparts during the same duration and type of exercise [Wells and Hooker, 1990].
Accordingly, the purpose of this study was to examine the effects of IMT training on the quality of life/respiratory function and repetitive propulsive sprint performance in wheelchair basketball players.

**Materials and Methods**

Participants
This study was approved by the local university ethics committee, and participants’ informed consent was obtained before data collection. Sixteen competitive wheelchair basketball players participated in the study. The disabilities of the participants included SCI, postpolio and spina bifida; the minimum time of onset of disability was 5 years. The descriptive characteristics of the participant groups are presented in table 1. All participants were highly trained and competed regularly in wheelchair basketball competitions at a national level or higher for at least 4 years. In addition to basketball-specific training, other sporting activities included swimming, weight training and tennis with a minimum weekly activity of 10h. All but one participant were non-smokers (self-reported).

<table>
<thead>
<tr>
<th>Table 1: Characteristics of participants in inspiratory muscle training (IMT) and sham-IMT groups (mean ± standard deviation)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>IMT</strong></td>
</tr>
<tr>
<td>Age [y]</td>
</tr>
<tr>
<td>Sex</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>Female</td>
</tr>
<tr>
<td>Level of play</td>
</tr>
<tr>
<td>International</td>
</tr>
<tr>
<td>National (club)</td>
</tr>
<tr>
<td>Self reported smoker</td>
</tr>
<tr>
<td>Disability</td>
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<tr>
<td>Spinal cord injury</td>
</tr>
<tr>
<td>Polio</td>
</tr>
<tr>
<td>Spina bifida</td>
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<tr>
<td>IWBF basketball classification</td>
</tr>
<tr>
<td>1/1.5</td>
</tr>
<tr>
<td>2/2.5</td>
</tr>
<tr>
<td>3</td>
</tr>
</tbody>
</table>

IWBF: International Wheelchair Basketball Federation

General design and procedures
Participants were divided by sex, ranked according to, first, their baseline maximum inspiratory muscle strength (maximum inspiratory pressure [MIP]) and, second, to the International Wheelchair Basketball Federation classification and then assigned to either an
experimental (IMT) or a placebo (sham-IMT) group using an ABBA grouping technique to ensure that no difference in MIP at baseline between the two groups existed.

Testing took place during the closed season. However, all participants were still completing their normal training programme. To prevent possible bias related to circadian rhythms, each test was scheduled at the same time of day, at the same regular training session. Participants were advised not to take part in vigorous exercise during the 2 days before the tests and did not exercise on the day of the tests. All lung function tests were completed in the participants’ “everyday” wheelchair, whereas sprints were completed in their “basketball” wheelchair. To complete the lung function tests, all participants sat up straight, without straps, to prevent bias related to differences in posture and the disposition of the abdomen.

Pulmonary function
Forced vital capacity, forced expiratory volume in 1s and the maximum voluntary ventilation during 12s were measured using a portable pneumotachograph spirometer (Vitalograph Gold Standard 2150, Buckingham, UK). Peak expiratory flow was measured using a handheld peak-flow meter (Mini-Wright, Harlow, UK). Measurements were made according to recommendations of the European Respiratory Society [Quanjer et al., 1993].

Respiratory muscle strength
Respiratory muscle strength was measured using a portable handheld pressure meter (Micro Medical, Rochester, Kent, UK). Each participant was assessed for maximum expiratory pressure (MEP) and MIP. Ten maximal efforts were performed at 30s intervals from residual volume for MIP or total lung capacity for MEP. In line with previous methodologies, the highest value was chosen to represent MIP and MEP [Romer et al., 2002a].

Repetitive wheelchair sprint performance
The experimental design was similar to that previously reported by Romer and colleagues [2002a], which consisted of fifteen 20m sprints with the performance criteria being to maintain maximal sprint performance whilst taking as little rest as possible between sprints. To familiarise the participants, the warm-up consisted of 5x20m at a self-selected speed followed by three sprints with 30s of rest in between. After this warm-up, each participant completed fifteen 20m sprints. Sprints were automatically timed to the nearest 0.01s by an electronic timing system (MMU-Cheshire, UK). Total test time was recorded manually to the nearest 0.01s with a stopwatch. Total sprint time (sprint 1 time plus sprint 2 time, etc) and total recovery time (total test time minus total sprint time) were calculated for all sprints and each set of five sprints.

Heart rate was recorded using radiotelemetry throughout the sprint test (Polar Sport Tester, Kempele, Finland). Immediately after the sprint protocol, a capillary blood sample was taken from the earlobe for subsequent determination of blood lactate concentration (Lactate Pro, blood lactate test meter, Arkray, Kyoto, Japan).
Inspiratory muscle training
The IMT was completed with a POWERbreathe device (IMT Technologies, Warwickshire, UK). After 1 week of IMT at level 1 of the device, the protocol for the following 5 weeks was given to each participant, and the technique was watched for accuracy. The experimental group (IMT) performed 30 dynamic inspiratory efforts twice a day against a pressure-threshold load equivalent to 50% MIP, a pressure known to improve performance in healthy people [Caine and McConnell, 2000]. Participants in this group were instructed to incrementally increase the load (quarter turn on the POWERbreathe) once 30 breaths became easy to complete. The placebo group (sham-IMT) trained with 60 slow breaths once a day equivalent to 15% MIP, a pressure known to induce only minimal changes, if any, in inspiratory muscle function in able-bodied people [Romer et al., 2002a]. Groups were told they were completing a study to look at the differences between endurance (sham-IMT) and strength (IMT) training of the respiratory muscles and were therefore blinded to the real purpose of the study. Instructions were given to cease IMT 48h before the post-test and to return the completed physical activity and IMT diary. This diary enabled participants to write down their daily activities and their adherence to the training programme. Furthermore, a questionnaire was given to each participant on completion of all the tests to identify the personal feelings of using POWERbreathe.

Data analysis
The SPSS V.15.0 statistical package (SPSS, Chicago, Illinois, USA) was used for all the statistical analyses. Means and SDs were computed for all variables. A two-way analysis of variance with repeated measurements was used, with training (pre and post) as within-subject variable and group (IMT vs sham-IMT) as between-subject variable. Significance was assumed at $p \leq 0.05$. A Bonferroni post hoc test was applied to determine the location of any significant main effects.

Results
The participants for this study were representative of wheelchair basketball players who trained regularly yet had never used a POWERbreathe respiratory training device before. An important feature of the two groups was that they did have similar baseline MIP and MEP ($p>0.05$).

IMT compliance
The self-reported diary sheets showed a 63% (13%) adherence in the experimental group and 79% (19%) adherence in the sham-IMT group. The participants were asked for their experiences of using POWERbreathe (table 2). Of the 18 who completed the training, 75% said they would now buy a POWERbreathe and 83% said they felt that it would decrease the number and length of respiratory infections in the long term. “Less feelings of breathlessness” and “less tightness in the chest during the training” were familiar comments made in response to questions, indicating an improvement in the quality of life.
Table 2: Personal experience of using POWERbreathe

<table>
<thead>
<tr>
<th>Question</th>
<th>IMT</th>
<th>Sham-IMT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Did you think it helped in everyday life?</td>
<td>67 (33)</td>
<td>83 (17)</td>
</tr>
<tr>
<td>2. Do you think it helped on the basketball court or while doing any other physical activity?</td>
<td>83 (17)</td>
<td>83 (17)</td>
</tr>
<tr>
<td>3. Do you think it relieved some of the symptoms of being out of breath? Less tightness in the chest? Less heavy breathing?</td>
<td>83 (17)</td>
<td>67 (33)</td>
</tr>
<tr>
<td>4. Would you buy a POWERbreathe?</td>
<td>83 (17)</td>
<td>83 (17)</td>
</tr>
<tr>
<td>5. Do you think it could help long term in sports or everyday life?</td>
<td>83 (17)</td>
<td>83 (17)</td>
</tr>
</tbody>
</table>

Pulmonary and respiratory muscle function
For both groups, none of the pulmonary function measures were significantly changed after the training period (table 3).

Table 3: Inspiratory muscle training (IMT, n=8) and sham-IMT (n=8)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>IMT</th>
<th>Sham-IMT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-IMT</td>
<td>Post-IMT</td>
</tr>
<tr>
<td>FVC [L]</td>
<td>3.77 ± 0.60</td>
<td>3.77 ± 0.45</td>
</tr>
<tr>
<td>FEV₁ [L]</td>
<td>3.36 ± 0.70</td>
<td>3.39 ± 0.60</td>
</tr>
<tr>
<td>PEF [L·min⁻¹]</td>
<td>492 ± 46</td>
<td>488 ± 39</td>
</tr>
<tr>
<td>MVV [L·min⁻¹]</td>
<td>142 ± 41</td>
<td>158 ± 59</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± standard deviation
FEV₁: forced expiratory volume in 1s; FVC: forced vital capacity; PEF: peak expiratory flow; MVV: maximum voluntary ventilation

However, significant improvements in MIP (75.4±33.3 to 91.9±25.5 cm H₂O) and MEP (69.4±21.0 to 84.5±21.2 cm H₂O), which corresponded to a change of 17% and 23%, respectively, were noted for the IMT group. Interestingly, similar improvements were noted for the sham-IMT group with 23% and 33% improvements, respectively, from baseline for MIP (74.5±27.3 to 87.5±30.7 cm H₂O) and MEP (60.4±18.3 to 79.6±28.5 cm H₂O) (fig. 1).
There were main effects for time, indicating a change from pre to post (MIP, p=0.028 and MEP, p=0.003). The non-significant group by time interaction (MIP, p=0.776 and MEP, p=0.667) showed that the changes were similar for the two groups.

Repetitive sprint performance
Table 4 shows that neither IMT nor sham-IMT training resulted in changes in total test, sprint or recovery time (p>0.05), or post-blood lactate concentration (p=0.183). Also, the peak heart rate during the sprints was similar before and after training (p=0.521), irrespective of the type of IMT training.

Table 4: Means ± standard deviation of performance tests pre an post-IMT or sham-IMT training

<table>
<thead>
<tr>
<th>Question</th>
<th>IMT</th>
<th>Sham-IMT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total test time [s]</td>
<td>132 ± 10</td>
<td>148 ± 25</td>
</tr>
<tr>
<td>Total recovery time [s]</td>
<td>35 ± 12</td>
<td>46 ± 27</td>
</tr>
<tr>
<td>Peak heart rate [bpm]</td>
<td>181 ± 6</td>
<td>172 ± 21</td>
</tr>
<tr>
<td>Post blood lactate concentration [mmol·L⁻¹]</td>
<td>9.0 ± 3.1</td>
<td>6.9 ± 2.9</td>
</tr>
</tbody>
</table>
Discussion

The primary finding of this study is that a 6-week period of IMT improves respiratory function and quality of life in wheelchair athletes. Interestingly, the improvements in respiratory function occurred even after mild IMT training. This indicates that even minimal-intensity IMT is sufficient to improve respiratory muscle function and quality of life in all disability groups tested.

Maximum respiratory pressures are temporarily reduced in people with high-lesion spinal injuries but recover to some extent during rehabilitation [Van Houtte et al., 2006; Liaw et al., 2000]. Given that participants used a wheelchair for daily ambulation for several years, it was considered that they all had a stable lung function. Because IMT predominantly concerns inspiration, most previous studies have focused on MIP rather than on MEP. Nevertheless, significant increases have been found in MEP [Liaw et al., 2000; Mueller et al., 2008]. For SCI, the increased MEP may be an important factor contributing to the improved quality of life, as the increased ability to generate pressure will aid with coughing and normal respiration. Grandas et al. [2005] noted that dyspnoea in chronic SCI patients may be because of impaired ventilatory muscle function and that adaptive strategies are used to alleviate the problem.

The similar improvements in respiratory function in the IMT and sham-IMT groups may be related to the better adherence to the training programme shown in the sham-IMT group. Possibly, the effect of IMT training, whether strength or endurance focused, was not because of stronger respiratory muscles but to a better technique/mechanical coordination or efficiency/learning effect of respiratory muscles. This might explain why both groups improved MIP and MEP by about the same amount. In this context, although not expected, the reason for an increase in MEP could be a mechanical advantage of respiratory muscles. If, because of IMT, subjects are able to inflate their lungs better (or different from a mechanical point of view), elastic recoil of the chest might be increased and thus also lead to a higher MEP value. These observations also highlight the need to closely control the adherence to training programmes in these groups.

We have shown that even minimal IMT would reduce symptoms of breathlessness during everyday activities and exercise. In addition, in both groups, the participants felt the training would reduce the number and length of respiratory infections. Thus, overall IMT improved the quality of day to day life. Therefore, even mild IMT may be used successfully during the rehabilitation process of individuals with SCI to avoid respiratory infections and complications. However, to prove this assumption, further studies are needed [Van Houtte et al., 2006].

In line with previous observations in able-bodied subjects [Williams et al., 2002; Morgan et al., 1987] we observed no significant improvement in sprint performance after IMT. It is quite possible that the large interindividual variances in performance and the small sample size attributed to these non-significant findings. Moreover, it has been found that prefatigued respiratory muscles limit a subsequent exercise performance [Mador and Acevedo, 1991]. Hence, we could speculate that the time between the last respiratory training session and the sprint performance session (48h) was too short, and the respiratory muscles were still fatigued, which may have influenced test results. This area warrants further investigation.
The main limitation of this study is the mixed disabilities in the present study population. Indeed, van der Woude et al. [2002] noted that aerobic capacity and respiratory function are highly variable upon disability and lesion level. It is thus advisable that future studies focus on a single disability and have the level of lesion in people with SCI as close as possible. The study is, however, indicative of the paraplegics within wheelchair basketball.

Conclusions

Our data indicate that the IMT, even at low intensity, had beneficial effects on respiratory function and quality of life. Future studies may address the question whether long-term respiratory muscle training would have lasting advantageous effects on respiratory muscle function and help decrease the severity of re-occurring chest and throat infections - a common illness of people with SCI.
3.4 Impact of low intensity isocapnic hyperpnoea on blood lactate disappearance after exhaustive arm exercise

Authors: Claudio Perret¹ and Gabi Mueller²

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²Institute of Sports Medicine, Swiss Paraplegic Centre, Nottwil, Switzerland


Introduction

Exhaustive, anaerobic exercise leads to a large production of lactate and hydrogen ions in the exercising muscles, causing a concomitant decrease in intracellular pH [Astrand et al., 1986; Hermansen, 1979]. This acidosis impairs the ability for muscle contraction and glycolytic enzyme activity, leading to reduced exercise performance [Hermansen, 1979]. To regain optimal performance as soon as possible, fast lactic acid elimination is crucial for athletes, particularly if there are repeated, high-intensity bouts of exercise within a short period of time, such as competitions in swimming, athletics or wheelchair racing [McMaster et al., 1989; Monedero and Donne, 2000].

In general, blood lactate elimination is accelerated by active recovery of the previously loaded muscle group at moderate intensities [Ahmaidi et al., 1996; Bond et al., 1991; Gupta et al., 1996; Hermansen and Stensvold, 1972; Monedero and Donne, 2000; Stambrook et al., 1981; Weltman et al., 1977]. In practice, owing to restricted infrastructural possibilities or a densely packed schedule during a competition, active recovery is not always possible. Moreover, active recovery using the previously loaded muscle group may impair subsequent exercise performance as energy stores become additionally depleted [Dupont et al., 2003], early resynthesis of muscle glycogen is compromised, and time for an optimal refilling of glycogen stores is shortened [Choi et al., 1994]. This might have negative consequences for subsequent exercise performance, especially in sports or situations where it is not possible to use another muscle group for active recovery purposes (e.g. leg exercise after upper body exercise) as is the case in athletes with an injured spinal cord.

Type I fibres can metabolise lactate [Brooks and Gaesser, 1980; Juel, 2001; McLoughlin et al., 1991], and as respiratory muscles mainly consist of type I fibres [Johnson et al., 1973; Lieberman et al., 1973; NHLBI workshop summary, 1990], we hypothesised that respiratory muscles may have the potential to enhance blood lactate elimination. If this is the case, respiratory muscles might be used as an easy to handle tool to accelerate blood lactate elimination without affecting glycogen resynthesis in previously exhausted limb muscles. This study aimed at investigating the impact of low-intensity isocapnic hyperpnoea (IH) on blood lactate disappearance after exhaustive arm exercise and at comparing these data with results of conventional active and passive recovery protocols. An acceleration of blood lactate elimination by active recovery strategies (moderate arm cranking exercise and IH) was expected compared with that of passive recovery.
Subjects and Methods

Subjects
Eighteen trained, male, non-smoking subjects participated in the study. Their average age was 30±5 years, height 178±7cm, weight 72±9kg, weekly physical training volume 5.6±3.3h and peak oxygen uptake (VO₂peak) for arm cranking 42±7ml·min⁻¹·kg⁻¹. Subjects were asked to perform no strenuous exercise and eat food rich in carbohydrates the day before testing and to abstain from caffeine intake on the test days. The study was approved by the ethics committee of Canton Luzern, Switzerland. Written informed consent of the subjects was obtained before the start of the study.

Experimental procedure
Preliminary testing
Two preliminary sessions preceded the three main test trials. During the first preliminary session, standard spirometric data, including forced vital capacity (FVC), forced expiratory volume in 1s (FEV₁), peak expiratory flow rate (PEF) and maximal voluntary ventilation (MVV) over 12s were obtained by an ergospirometric device (Oxycon Alpha, Jaeger, Hoechberg, Germany). Afterwards, a familiarisation trial for IH by partial rebreathing from a bag was completed (Spirotiger, Idiag, Volketswil, Switzerland). Bag size corresponded to about one third of subjects’ FVC. Subjects were breathing at a preset respiratory frequency to reach a target minute ventilation (see below).

The second preliminary session consisted of 30min continuous IH at a minute ventilation corresponding to 30% MVV and ensured that subjects could sustain this load over 30min. To assure the predetermined target ventilation, tidal volume, breathing frequency and end tidal partial pressure for carbon dioxide were monitored by means of the above-mentioned metabolic cart. If necessary, subjects were guided to keep preset tidal volume and breathing frequency to achieve the target ventilation.

Main experiments
All three main tests started with a 2min resting period for baseline data determination. Subjects were seated in a chair connected to an arm cranking (AC) ergometer (Ergometrics 800 SH, Ergoline, Bitz, Germany). After this resting period, subjects started AC at 20W. Then, the workload was increased 5W very 20s until subjects’ volitional exhaustion. Respiratory variables at rest and during the exercise tests were sampled breath by breath through a face mask by an Oxycon Pro (Jaeger, Würzburg, Germany). This device was calibrated for gas and volume according to the manufacturer’s recommendations immediately before each test. The three main tests were at least 48h apart.

During AC, the pedal axis of the ergometer was aligned with the shoulder joint axis and subjects were positioned such that the elbow was slightly flexed at maximal reach. Five minutes after the end of the test, subjects followed one of three recovery strategies in a randomised order. These strategies were passive recovery (PR), active recovery by AC or ventilatory recovery (VR) by means of IH for 30min. While subjects sat relaxing on a chair for PR, they were either arm cranking at 30% of the previously reached maximal workload (AC) or breathing at 30% MVV with the Spirotiger device (VR) as an active recovery strategy.
Capillary blood for enzymatic lactate analysis (Super GL Ambulance, RuhrタルLabor Technik, Möhnesee, Germany) was sampled from an earlobe at rest, immediately after cessation of the exercise test and every 2 min until the end of the recovery phase. Heart rate (HR) was determined by an HR monitor (Polar S610, Polar, Kempele, Finland) throughout the whole test and recovery period. Immediately after cessation of the AC exercise test, subjects were asked to rate their perceived exertion by means of a Borg scale, with 6 indicating “no” and 20 “maximal” exhaustion [Borg, 1982].

Statistics
Data points of the measured lactate concentrations of the three different recovery strategies were fitted to the following biexponential curve, as described in detail elsewhere [Freund and Zouloumian, 1981]:

\[ La(t) = La(0) + A_1 \cdot (1 - e^{-\gamma_1 t}) + A_2 \cdot (1 - e^{-\gamma_2 t}) \]

La(t) denotes the time-dependent lactate concentration, with La(0) being the lactate concentration at the start of recovery. The form of this equation suggests that the lactate kinetics during recovery can be described by two main processes, one with a high velocity constant (\( \gamma_1 \)) describing the appearance (\( A_1 > 0 \)) of lactate in the bloodstream and the other with a low velocity constant (\( \gamma_2 \)) describing its disappearance (\( A_2 < 0 \)).

The variables were calculated using a commercially available computer software package (SYSTAT, version 10, SPSS Inc, Richmond, California, USA) with the regression method of least mean squares.

An analysis of variance for repeated measures was applied to compare \( \gamma_1 \) and \( \gamma_2 \) between the different intervention strategies (PR, AC, VR). In addition, VO\(_{\text{2peak}}\), peak power, rating of perceived exertion as well as maximal and average recovery HRs were analysed in the same way. If significance was found, a post hoc test with Bonferroni correction was used to locate significant differences. Results are given as mean ± standard deviation. Values were considered to be significantly different if \( p<0.05 \).

The ventilatory threshold (VT) of the exercise test preceding AC was calculated from carbon dioxide output (V\( \text{CO}_2 \)) and VO\(_2\) values according to the V-slope method [Beaver et al., 1986].

Results
The mean of the subjects’ FVC was 5.8±1.1 l (115% predicted), FEV\(_1\) 4.6±0.7 l (109% predicted), PEF 11.4±1.4 l·s\(^{-1}\) (119% predicted) and MVV 206.8±30 l·min\(^{-1}\) (140% predicted). Peak power output, VO\(_{\text{2peak}}\), maximal HR and rating of perceived exertion did not differ significantly between tests (table 1).
Table 1: Values measured at the end of exhaustive arm cranking tests before starting different recovery strategies

<table>
<thead>
<tr>
<th>Values measured</th>
<th>Passive</th>
<th>Arm cranking</th>
<th>Isocapnic hyperpnoea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak power [W]</td>
<td>148.1 ± 24.7</td>
<td>147.8 ± 22.7</td>
<td>146.9 ± 23.0</td>
</tr>
<tr>
<td>Peak oxygen uptake [mL·min(^{-1})·kg(^{-1})]</td>
<td>41.7 ± 7.3</td>
<td>40.6 ± 4.8</td>
<td>41.7 ± 8.0</td>
</tr>
<tr>
<td>Maximal heart rate [bpm]</td>
<td>175.9 ± 11.7</td>
<td>176.2 ± 9.8</td>
<td>176.2 ± 10.5</td>
</tr>
<tr>
<td>Rate of perceived exertion (Borg)</td>
<td>18.3 ± 1.4</td>
<td>18.3 ± 1.4</td>
<td>18.2 ± 1.2</td>
</tr>
</tbody>
</table>

Note that there were no significant differences between tests.

Figure 1 shows blood lactate curves. The low-velocity constants (\(\gamma_2\)) describing blood lactate disappearance were 0.052±0.031min\(^{-1}\), 0.072±0.035min\(^{-1}\) and 0.053±0.032min\(^{-1}\) for PR, AC and VR respectively (p=0.138), indicating that blood lactate elimination rate constants after exhaustive arm ergometry were not significantly different among the three different recovery strategies. Average recovery HRs during AC (115.9±9.2 bpm) and VR (111.3±16.8 bpm) were significantly higher than the average HR during PR (92.8±10.5 bpm).

Figure 1 Mean blood lactate elimination curves for different recovery strategies after an exhaustive arm cranking exercise test (AET).
Average workload during AC was 44.2±7.3W, corresponding to 29.9±1.0% of peak power output. This workload was clearly below the VT at 55.8±14.5% of VO\textsubscript{2peak}, which corresponded to an absolute workload of 88.1±30.0W.

During VR subjects were ventilating 61.6±9.3l/min (29.8±0.9% of MVV) at an average breathing frequency of 26.2±3.7min\textsuperscript{-1}. Average end tidal partial pressure for carbon dioxide was 32.8±5.1mmHg.

Discussion

The main finding of this study is that there were no significant differences in blood lactate disappearance after exhaustive arm cranking exercise due to the different recovery strategies. This was surprising, as a superior impact of active recovery strategies (AC and VR) on blood lactate elimination was expected compared with PR. Different reasons may explain this observation and are discussed below.

AC for active recovery

Active recovery at moderate intensities by means of the previously loaded muscle group is known to accelerate blood lactate elimination compared with PR [Ahmaidi et al., 1996; Bond et al., 1991; Gupta et al., 1996; Hermansen and Stensvold, 1972; Monedero and Donne, 2000; Stanford et al., 1981; Weltman et al., 1977]. Although AC showed a nearly 40% faster low-velocity constant (γ\textsubscript{2}) than PR and VR, no significant differences (p=0.138) were found between the different recovery strategies. This might be owing to the relatively large interindividual differences between subjects (fig. 1) but may be caused for other reasons discussed below.

In contrast to the above mentioned studies where leg exercise was applied, we used an arm cranking exercise for recovery. Slower blood lactate kinetics for arm exercise compared with leg exercise were also reported by Thiriet et al. [1993]. Obviously, the magnitude of the affected muscle mass is critical for effective active recovery. Arm muscle mass in a group of 29-year-old men was found to be only 40% (7.1kg vs. 17.7kg) of leg muscle mass [Nindl et al., 2002]. Taking into account the higher proportion of type I fibres [Johnson et al., 1973; Susheela and Walton, 1969], combined with the bigger muscle mass involved during leg exercise, it seems not surprising that lactate elimination from arm muscles would be different from that of leg muscles. The large proportion of type II fibres in arm muscles [Johnson et al., 1973; Susheela and Walton, 1969] limits their ability to oxidise lactate itself. In combination with the relatively small muscle mass of the arms, lactate elimination is additionally compromised compared with elimination from leg muscles.

Moreover, if the chosen intensity for AC had been too high, lactate elimination would have decelerated. However, considering the measured mean recovery HR of 116bpm during AC compared with other active recovery HRs reported in the literature [Gupta et al., 1996; Monedero and Donne, 2002; Thieret et al., 1993], it seems very unlikely that our recovery intensity of 30% of maximal power output was too high. Moreover, a recovery load of 30% of maximal power output was suggested to be within the optimal range for active recovery and has been successfully applied in the past [Dodd et al., 1984; McLellan and Skinner, 1984;]
Thieret et al., 1993]. Further, the chosen recovery workload was far below the VT, which supports the assumption of an optimal active recovery intensity.

However, the present active recovery design by means of AC (instead of using previously unloaded leg muscles) was chosen, as there are situations where it is not possible to use another muscle group for active recovery purposes as is the case, for example, in athletes with an injured spinal cord.

VR for active recovery
With regard to the type of muscle fibre in the arm versus respiratory muscles, one can hypothesise that VR is better than AC and PR in accelerating blood lactate disappearance. However, this assumption was not supported by the present findings. Although respiratory muscles mainly contain type I fibres [Johnson et al., 1973; Lieberman et al., 1973; NHLBI workshop summary, 1990], the total muscle mass affected in VR is quite small and thus oxidative capacity is limited. In fact, diaphragmatic muscle mass was found to be only 262g [Arora and Rochester, 1982] compared with the above-mentioned 7kg of arm muscle mass [Nindl et al., 2002]. Even if during VR the auxiliary respiratory muscles worked actively, this seems to be a very small active muscle mass to engage in lactate oxidation, which might be the main reason why the VR strategy failed to enhance blood lactate elimination.

A further reason might be that the chosen intensity of IH at 30% MVV was too low for a sufficient effect on lactate metabolisation over 30min. On the other hand, a higher intensity of IH might have led to respiratory muscle fatigue, which compromises subsequent exercise performance [Mador and Acevedo, 1991; Martin et al., 1982]. In fact, Martin and coworkers [1982] showed that intense IH at 66% of the mean MVV reduces subsequent running performance.

Additionally, the possibility has to be taken into account that during upper body exercise, some of the respiratory muscles of the rib cage have to partake in non-ventilatory functions [Celli et al., 1988]. Thus, a recovery strategy like IH, integrating all respiratory muscles, should be performed at a moderate intensity, to avoid the development of rib cage muscle fatigue, which otherwise might compromise subsequent AC performance. The fact that average recovery HR during VR was close to the HR values during AC and significantly higher than during PR (table 1) suggests that the chosen intensity of IH at 30% MVV was adequate and comparable to the intensity during AC. Hence, it is questionable if a higher intensity of IH (e.g. 40–50% MVV) would provide faster lactate removal without causing respiratory muscle fatigue.

Perhaps specifically trained respiratory muscles would have provided faster blood lactate elimination. This assumption is supported by the finding of Spengler et al. [1999], who demonstrated lower blood lactate concentrations at the end of an exhaustive cycling exercise after 4 weeks of IH training. It was suggested by the authors that the decrease in blood lactate concentration was probably caused by enhanced lactate uptake of the trained respiratory muscles. In contrast, others observed no changes of blood lactate concentrations after respiratory muscle training [Sonetti et al., 2001]. As the respiratory muscles of our study subjects were not specifically trained, it would be interesting to see if there were differences in the potential for lactate elimination after a respiratory training period. Further investigations are needed to answer this question.
Preliminary exercise and lactate elimination

It has been shown that the intensity of preliminary exercise influences the kinetics of subsequent lactate elimination [Freund et al., 1986] and thus might falsify results. Data of the three arm cranking tests to exhaustion preceding the three recovery interventions refute this assumption for the present study. No differences were found in peak power, \( \text{VO}_{2}\text{peak} \), maximal HR or peak lactate concentration between tests (table 1 and fig. 1), which implies that the preceding exhaustive exercise was comparable for each testing session.

Conclusions

Low-intensity IH seems not to enhance blood lactate disappearance after exhaustive arm exercise compared with passive or active recovery with the previously loaded muscle group. The magnitude of the affected muscle mass seems to be critical to an effective active recovery as shown by the fact that AC also failed to enhance blood lactate elimination significantly.
4 Functional electrical stimulated (FES) cycling in persons with spinal cord injury

The possibilities for physical activities in individuals with a spinal cord injury (SCI) are obviously restricted due to a loss of motor function in the paralysed limbs. However, reduced fitness levels are responsible for co-morbidities such as hyperlipidaemia, diabetes, cardiovascular diseases or obesity [Myers et al., 2007]. It is an evident fact, that the prevalence of the latter is 40-66% in the population with SCI [Anson and Shepherd, 1996; Chen et al., 2006; Liang et al., 2007]. Thus, there is no doubt that regular physical activity is a must and plays a key role in reducing health-related risk factors and complications in individuals with SCI [Mohr et al., 1997a]. However, at the same time overuse symptoms of the upper extremities, in particular of the shoulder, are very common [Sie et al., 1992; Silfverskiold and Waters, 1991] and have to be taken into account when exercising with persons with SCI. Additionally, only a small muscle mass is involved, when physical activities are restricted to the upper body. Keeping all these circumstances in mind, it seems to be a good option to involve the lower extremities of patients with SCI in a regular physical activity program. In this context, the use of functional electrical stimulated (FES) cycling might offer a perfect possibility to involve a large muscle group, and concurrently to preserve the upper extremities from additional exercise-induced loads.

The studies of the following chapter focus on different aspects of FES cycling, mainly related to cardiorespiratory and musculoskeletal adaptations after a high-volume, home-based 12-month FES cycle training program. The first step to accurately monitor and modify a FES exercise training program was the implementation of a meaningful exercise testing setup to allow a stringent characterization of physiological performance parameters of FES cycle ergometry [Perret et al., 2009]. A follow-up study aimed to investigate the power and the cardiorespiratory adaptations of a high-volume one year FES cycle training program using the above mentioned testing method [Berry et al., 2008]. Further, efficiency of FES cycling exercise was characterised [Hunt et al., 2007] and the energetic demand of this special mode of exercise was assessed [Perret et al., 2010].

Beside cardiorespiratory adaptations, the question arised, what the impact of a high-volume FES cycle training program over 12 months on musculoskeletal parameters such as bone mineral density or muscle mass will be [Frotzler et al., 2008b] and if possible adaptations will be preserved when training intensity is reduced or training is totally stopped [Frotzler et al., 2009].

Finally, the last study of this chapter [Kakebeeke et al., 2008] investigated, if a high-volume FES cycle training program over several months is also feasible for subjects with a complete tetraplegia.
4.1 Determination and possible application of the gas exchange threshold in aerobically untrained paraplegic subjects based on stimulated cycle ergometry

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Published in: Disabil Rehabil 2009; 31: 1432-1436.

Introduction

Several beneficial, functional and therapeutic effects in spinal cord injured (SCI) subjects have been reported after functional electrical stimulation (FES) exercise programmes [Andrews and Wheeler, 1995; Janssen et al., 1998]. Health benefits include improved cardiopulmonary fitness [Hooker et al., 1992], enhanced insulin sensitivity [Mohr et al., 2001] and positive effects on bone density [Chen et al., 2005; Mohr et al., 1997b]. Moreover, the use of lower extremity exercise enables SCI subjects to be engaged in physical activity without stressing the upper body thus avoiding the possibility of overuse syndrome of the upper extremities, which might otherwise lead to major problems [Burnham et al., 1993].

Exercise testing is useful in SCI subjects as it allows changes in physical fitness to be monitored and provides guidance on FES exercise training programme modifications. Several studies have investigated the effects of FES-cycling exercise on cardiopulmonary fitness using stationary FES ergometers [e.g. Hooker et al., 1995; Krauss et al., 1993; Mohr et al., 1997b; Mutton et al., 1997] but the very low work capacity of SCI subjects has until now resulted in limitations in the application of exercise tests in this population. The smallest work rate increment of commonly used ergometers is mostly limited to 6W, which, for many SCI subjects, will be a substantial fraction of their maximal exercise capacity and thus limits the sensitivity of test results [Wasserman et al., 2004]. Moreover, it has to be considered that untrained SCI subjects may not be able to generate sufficient power to overcome the frictional losses associated with turning their legs to move the pedals and initiate unloaded cycling.

Fortunately, recent progress in biomedical engineering now allows stringent characterisation of physiological performance parameters of electrically stimulated cycle ergometry even in aerobically untrained paraplegic subjects [Hunt et al., 2004]. In a previous pilot study, utilising this new testbed, it was demonstrated that it is in principle possible to detect an aerobic gas exchange threshold (GET) in a trained SCI subject performing FES-cycling [Ferrario et al.,
2007]. However, there is a lack of data on cardiopulmonary performance parameters in this special group of patients and a consensus on how FES-cycle training should be guided best is lacking. Thus, the aim of the present study was to characterise cardiopulmonary baseline performance in aerobically untrained paraplegic subjects and to derive possible training recommendations based on these measurements.

**Methods**

**Subjects**

Twelve motor complete paraplegic subjects (10 males/2 females) participated in the study. Detailed information of their anthropometrical data and impairment are presented in Table 1.

**Table 1: Subject characteristics**

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age [years]</th>
<th>Height [cm]</th>
<th>Weight [kg]</th>
<th>Time post injury [years]</th>
<th>Lesion level</th>
<th>AIS</th>
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<tr>
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<td>184</td>
<td>70</td>
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<td>T7</td>
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<td>173</td>
<td>59</td>
<td>3.5</td>
<td>T6</td>
<td>A</td>
</tr>
</tbody>
</table>

**Mean ± SD**

41.7 ± 9.1 175.6 ± 7.3 75.7 ± 15.1 10.1 ± 7.0

SD: standard deviation; AIS: American Spinal Injury Association Impairment Scale; T: thoracic lesion; M: male; F: female

The study was approved by the local ethical committees and subjects gave their written informed consent before participation.

The subjects had not previously performed stimulated leg-cycling exercise and completed an incremental exercise test (IET) (see below) to determine baseline values of cardiopulmonary performance parameters.

**Equipment and experimental procedure**

Exercise tests were carried out in three centres Swiss Paraplegic Research, Nottwil, Switzerland; Queen Elizabeth National Spinal Injuries Unit, Southern General Hospital, Glasgow, UK; King’s College London. All procedures were standardised.
Exercise testing was performed by means of a work rate and cadence controlled IET until maximal work rate was reached as described in detail elsewhere [Hunt et al., 2004]. Briefly, a recumbent tricycle (Inspired Cycle Engineering, Cornwall, UK) adapted for paraplegic subjects was used. The tricycle was equipped with an auxiliary electric motor and an integrated feedback system for control of exercise work rate and cadence.

Subjects sat on the tricycle to perform the IET. After a rest period of 3min, and a 4min period of motor-assisted passive cycling, the IET was performed. Work rate increments of 1W·min⁻¹ were used until stimulation intensity reached its preset maximum (150mA; 500µs). Stimulation of Mm. glutei, quadriceps and hamstrings was provided via surface electrodes by a multi-channel stimulator (Stanmore Stimulator; for details see [Phillips et al., 1993]) operating at a constant frequency (20Hz). These stimulation parameters were selected based on data from a previous study [Hunt et al., 2004] and a smooth cycling motion is obtained with them.

Work rate resulting from stimulation of the leg muscles was measured by a sensor fitted to the crankshaft (SRM Powermeter, Schoberer Rad Messtechnik GmbH, Jülich, Germany), independently of the motor’s contribution to total external work. During passive cycling (i.e. cycling with stimulation switched off), the legs were turned by the motor alone, resulting in measurement of a negative work rate at the crankshaft [Hunt et al., 2007]. This corresponds to the rate of work required just to rotate the legs.

During the whole IET, gas exchange and ventilatory variables were measured breath-by-breath (MetaMax 3B, Cortex Biophysik GmbH, Leipzig, Germany). The ergospirometric device was calibrated for gas and volume immediately before each test according to the manufacturer’s guidelines. Heart rate (HR) was recorded continuously (OxyTip, Datex-Ohmeda 3900, Louisville) during the whole testing period.

Statistics

Data are presented as mean±standard deviation. Respiratory data were calculated based on 15s average values derived from the breath-by-breath data. The highest 15 average of oxygen uptake (VO₂), minute ventilation (Vₑ), tidal volume (VT), respiratory frequency (fR) and respiratory exchange ratio (RER) reached during the test were taken as VO₂peak, Vₑpeak, VTpeak, fRpeak and RERpeak, respectively.

Aerobic GET was calculated from carbon dioxide output (VCO₂) and VO₂ values according to the V-slope method [Beaver et al., 1986] by means of a computerized linear regression model. The highest HR measured was determined as HR peak and the highest work rate reached as peak power (Ppeak).
Results

Mean values for resting as well as peak VO$_2$, HR, V$_E$, VT, RER are presented in Table 2.

Table 2: Resting and peak values of the incremental exercise test (mean±standard deviation)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Resting values</th>
<th>Peak values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen uptake (absolute) [ml·min$^{-1}$]</td>
<td>278 ± 58</td>
<td>671 ± 192</td>
</tr>
<tr>
<td>Oxygen uptake (relative) [ml·min$^{-1}$·kg$^{-1}$]</td>
<td>4.0 ± 0.9</td>
<td>9.1 ± 3.4</td>
</tr>
<tr>
<td>Heart rate [bpm]</td>
<td>68.7 ± 9.3</td>
<td>90.0 ± 12.4*</td>
</tr>
<tr>
<td>Minute ventilation [l·min$^{-1}$]</td>
<td>8.1 ± 2.3</td>
<td>23.6 ± 7.5</td>
</tr>
<tr>
<td>Tidal volume [l]</td>
<td>0.49 ± 0.13</td>
<td>1.32 ± 0.37</td>
</tr>
<tr>
<td>Respiratory frequency [min$^{-1}$]</td>
<td>16.1 ± 2.7</td>
<td>25.2 ± 4.1</td>
</tr>
<tr>
<td>Respiratory exchange ratio</td>
<td>0.71 ± 0.07</td>
<td>1.26 ± 0.21</td>
</tr>
</tbody>
</table>

*Data of one subject missing because of technical problems.

Figure 1. Typical results of an incremental exercise test with one subject. Note that the subject reached a peak oxygen uptake (VO$_{2peak}$) of 0.584l·min$^{-1}$, a peak heart rate (HR$_{peak}$) of 89 beats·min$^{-1}$, a gas exchange threshold (GET) of 0.350l·min$^{-1}$ VO$_2$ and a net peak power (P$_{peak}$) of 12.5W. Note that the subject did not reach an absolute work rate of 0W, which is a prerequisite to turn the pedals to initiate unloaded cycling.
Mean net $P_{\text{peak}}$ was $8.4\pm3.3\text{W}$ and average VO$_2$ at GET corresponded to $345\pm81\text{ml}\cdot\text{min}^{-1}$. Mean GET was found to be at $51.4\%\pm9.6\%$ of VO$_2\text{peak}$ and corresponded to $41.3\%\pm13.0\%$ of $P_{\text{peak}}$ (absolute work rate $3.1\pm3.6\text{W}$). Typical results of a test with one subject are shown in Figure 1.

**Discussion**

The present data underline the very low aerobic fitness level of untrained SCI subjects in the context of FES-cycling exercise. The average net peak work rate of $8\text{W}$ is minimal, and the corresponding absolute cycling work rate is therefore less than $0\text{W}$ in some subjects. This means that untrained SCI subjects may not be able to produce sufficient power even to overcome the frictional losses associated with turning their legs to move the pedals. This observation reflects the striking leg muscle atrophy after complete SCI [Olive et al., 2003]. Nevertheless, because of the use of the motor-assisted exercise protocol with very low work rate increments as described earlier [Hunt et al., 2004], it was possible for the first time to adequately determine cardiopulmonary performance parameters during stimulated cycling even in a group of untrained paraplegic subjects. In the past, sensitivity of test results was limited, as the smallest work rate increment of FES-cycling ergometers was mostly restricted to $6\text{W}$ [e.g. Hooker et al., 1995; Krauss et al., 1993; Mohr et al., 1997b; Mutton et al., 1997], which is a substantial fraction of the maximal work rate in untrained subjects. Therefore, a stringent characterisation of cardiopulmonary performance parameters in untrained paraplegic subjects was previously not possible.

Comparison of our data with results from trained FES-cyclists reveal the high potential of a FES-cycle training programme to improve cardiopulmonary fitness in SCI subjects. In fact, VO$_2\text{peak}$, HR$_{\text{peak}}$ and V$_{\text{Epeak}}$ values of up to $1430\text{ml}\cdot\text{min}^{-1}$, $132\text{beats}\cdot\text{min}^{-1}$ and $59\text{l}\cdot\text{min}^{-1}$, respectively, have been reported [e.g. Barstow et al., 2000; Hooker et al., 1995; Mutton et al., 1997]. This corresponds to values of $213\%$ in VO$_2\text{peak}$, $147\%$ in HR$_{\text{peak}}$ and $250\%$ in V$_{\text{Epeak}}$ compared to our baseline data of untrained subjects (Table 2). Further, one has to take into account that FES-cycling not only promotes an increased cardiopulmonary fitness but has also the potential to enhance the health status of SCI patients in a number of areas outlined in the introduction.

In general, endurance exercise has gained importance for rehabilitation. However, in order to successfully guide the training process, the assessment of adequate training intensities becomes important [Meyer et al., 2005]. For this purpose, the non-invasive determination of GET based on the V-slope method [Beaver et al., 1986] seems to offer some attractive possibilities. Training at an intensity above GET is necessary to ensure a training effect [Meyer et al., 2005]. However, no values for GET during FES-cycling exercise have been published so far, other than our single case study with a pre-trained subject [Ferrario et al., 2007]. This can be attributed to the limited precision and reliability of exercise testing previously available in this field, as discussed earlier.

In our subjects GET was found to be $51\pm10\%$ of VO$_2\text{peak}$ representing a relatively low fitness level at about the same relative level as the average $55\%$ VO$_2\text{max}$ reported for sedentary able-bodied subjects [Meyer et al., 2005]. In paraplegic subjects, GET during a wheelchair IET occurred at $56\%$ [Vinet et al., 1997] whereas GET was detected at $70$–$75\%$ VO$_2\text{max}$ in
well trained able-bodied cyclists [Lucia et al., 2001]. Given that GET can now be determined in SCI subjects, it is suggested that a high-volume FES-cycle training programme (normally associated with SCI subjects) above GET will lead to gains in cardiopulmonary fitness over time. Further studies are needed to investigate if and to what extent this is the case, and how the training process can best be guided based on GET measurements. However, the value of 51% VO$_{2\text{peak}}$ at the GET in our SCI subjects is very close to the 55% VO$_{2\text{max}}$ reported for untrained able-bodied subjects [Meyer et al., 2005] and reveals that the generally accepted principles for training prescription and guidance may also be applied for FES-cycle training. This insight might be useful in further improving FES-cycling performance and its health-related benefits during the rehabilitation process of subjects with complete SCI.

Conclusions

In conclusion, a cadence and work rate controlled exercise test allows the stringent characterisation of cardiopulmonary parameters during stimulated cycle ergometry even in aerobically untrained paraplegic subjects, who have a very low work capacity. The precise determination of GET allows an appropriate exercise intensity to be prescribed and thus provides a suitable method for exercise intensity calculation in the SCI population in the future.
4.2 Cardiorespiratory and power adaptations to stimulated cycle training in paraplegia

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Introduction

People with spinal cord injury (SCI) often become very sedentary, which leads to low cardiorespiratory fitness levels and many of the comorbidity associated with inactivity, including obesity, type 2 diabetes, and cardiovascular disease [Myers et al., 2007]. Physical activity has been found to have a preventative and therapeutic role for these conditions, with the greatest improvements in health being gained by the least fit because they become physically active. Thirty minutes of moderate daily activity, performed at an oxygen uptake (VO₂) of around 1000-1500ml·min⁻¹ for men or 700-1100ml·min⁻¹ for women, is reported to be the minimum requirement for minimizing health risks [Warburton et al., 2006a]. However, the peak oxygen uptake (VO₂peak) of untrained SCI wheelchair users rarely meets the minimum VO₂ required to be sustained for this duration of activity [Janssen et al., 2002].

The relatively small muscle mass used during upper-body exercise, the risk of shoulder pain from overuse [Burnham et al., 1993], and the deleterious effects of possible injury on activities of daily living have led to the research and development of electrically stimulated (ES) lower-limb exercise systems for SCI individuals [Heesterbeck et al., 2005; Hunt et al., 2007; Petrofsky et al., 1983; Petrofsky and Smith, 1992]. These systems allow temporary restoration of function to the paralyzed lower-limb muscles where stationary or mobile exercise training can then be performed.

In addition to the many diverse physiological benefits gained by complete-lesion SCI subjects after periods of ES cycling (for a review see Jansen et al. [1998]), significant, but variable, improvements in VO₂peak, peak power output (PO_peak), and endurance have been reported. Studies to date have investigated the responses of individuals with SCI during clinic-based ES cycle training studies of between 6 weeks and 12 months’ duration [Arnold et al., 1992; Barstow et al., 1996; Figoni et al., 1990; Goss et al., 1992; Hooker et al., 1992; Krauss et al., 1993; Mohr et al., 1997b; Pollack et al., 1989; Ragnarsson et al., 1988]. Training regimes have varied in work rate (0-42W), duration (5-30min), frequency (two to three times per
week), and test protocol (continuous or discontinuous), with some studies including a preparatory muscle-conditioning period before or after baseline testing.

The lack of consistency in methodology, test protocol, data treatment, and analysis across studies has made the direct comparison of results difficult if not invalid. Additionally, the relatively large power increments (~6W) used in all previous tests have resulted in a lack of measurement sensitivity and therefore the ability to detect small but perhaps clinically important changes in power and \( V_{O2peak} \) over time.

As an advance on the pedaling drive torque measurement test bed developed by Gföhler et al. [2001], an integrated feedback system was used for exercise testing that allowed simultaneous feedback control of power via automatic adjustment of stimulation and electric motor control of cadence. This permitted the application of arbitrarily small work rate increments and accurate quantification of power output even during unloaded cycling [Ferrario et al., 2007; Hunt et al., 2004]. To optimize the individual’s time management and to maximize their training within the prescribed high-volume limits, a home-based training program was designed.

The aim of the present multicenter study, therefore, was to investigate the power and the cardiorespiratory adaptations to a progressive, high-volume, home-based 12-month ES cycle training program using a novel, sensitive test bed that permits high-resolution power output analyses to be performed for the first time in ES cycling. The feasibility and the viability of home-based ES cycle training for improving and maintaining cardiorespiratory fitness by individuals with paraplegia were also examined.

**Methods**

Subjects
Twelve SCI individuals (2 females and 10 males), all motor and sensory complete T3 to T12 lesion (grade A on the American Spinal Injuries Association impairment scale), and with no previous experience of ES cycling, were recruited via the Queen Elizabeth National Spinal Injuries Unit, Glasgow (GLA), the Swiss Paraplegic Research, Nottwil (NOT), and the King’s College, London (LON; for full subject details see Table 1). Subjects gave their written informed consent to participate in the study, which was approved by their respective center’s ethics committee: the ethics committees of the Southern General Hospital and the Faculty of Biomedical and Life Sciences at the University of Glasgow (GLA), the ethics commission of Kanton Luzern (NOT), and the research ethics committee of King’s College Hospital (LON). All subjects were given a full physical assessment before taking part and were required to have distal tibia and femur trabecular bone densities greater than 40mg·cm\(^{-3}\) measured by peripheral quantitative computed tomography. One subject (GLA) dropped out of the study after baseline testing due to an adverse autonomic response to stimulation, and his data are therefore not included.
### Table 1: Subject characteristics

<table>
<thead>
<tr>
<th>Subject</th>
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<th>Years post injury</th>
<th>Height [cm]</th>
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</table>

Mean ± SD  
41.8 ± 7.6  
T5.5 ± 2.5  
10.7 ± 7.0  
175.6 ± 7.6  
73.6 ± 14.0

All subjects were grade A on the American Spinal Injuries Association impairment scale.
F: female; M: male; GLA: Glasgow; LON: London; NOT: Nottwil; T: thoracic spine; SD: standard deviation

### Muscle conditioning

To strengthen and to increase the fatigue resistance of the leg muscles before cycle training, subjects completed a minimum of 6wk (mean±SD, 14.2±6.9wk) of progressive (up to 60min per session with 1kg ankle weights added as required and tolerated thereafter), concentric muscle conditioning at home on 5d of the week. Pairs of self-adhesive surface electrodes (PALS Platinum; Nidd Valley Medical Ltd., Harrogate, North Yorkshire, UK) were placed proximally and distally to the motor point of the knee flexor and extensor muscle groups to be used during cycle training. A Salisbury Odstock four-channel stimulator (GLA) or an eight-channel electronic Stanmore stimulator (Salisbury, UK; LON and NOT) was used with stimulation parameters preset individually for each subject using a pulse frequency of either 20 or 50Hz and pulse duration between 300 and 400µs. Intensity was controlled by altering the current amplitude between 80 and 150mA. Stimulation was applied simultaneously to the flexors of one leg and the extensors of the other with a 1:1 duty cycle set at 6s on or off before stimulating the opposite muscle groups of each leg. Muscle conditioning was performed until subjects were able to pedal on the tricycle (detailed below) for at least 10min with no external load applied to allow the initial tests to be completed.

### Training equipment.

All training was performed at home on a commercially available mobile recumbent tricycle (Inspired Cycle Engineering Ltd., Falmouth, Cornwall, UK) adapted for ES use. This was mounted on an electronically braked cycle trainer (Flow Ergotrainer Tacx, Wassenaar, The Netherlands), which supplied resistance to the rear wheel. Feet were secured firmly to the pedals by rigid ankle orthoses to prevent movement around the ankle and to constrain limb movement to the sagittal plane. Due to a combination of relatively short limb length and low
bone density, one subject (NOT) used an adapted orthosis in conjunction with calf muscle stimulation.

A throttle was attached to the left-hand grip and interfaced with the stimulator software to allow the user to manually control stimulation intensity. A shaft encoder mounted on the crankshaft relayed feedback of crank arm position to the stimulator software to permit angle-specific muscle stimulation. A handheld computerized interface was used to control the trainer resistance and to display cycle cadence (revolutions per minute).

An eight-channel electronic stimulator (Stanmore stimulator) was used to stimulate the quadriceps, hamstrings, and glutei muscle groups of each leg via surface electrodes (detailed earlier). The triceps surae (calf) muscle groups were also stimulated in five LON subjects, because this group considered that this might augment knee flexion and increase power output, and in one NOT subject as noted previously. The stimulator was programmed to deliver charge to each muscle group, and the current was individually predetermined for each subject within the range of 0–150mA at a frequency of 50Hz. Stimulation intensity was controlled by adjusting the pulse duration, via the throttle, within the range of 0–510µs.

Cycle training protocol
From weeks 0 to 8 of cycle training, subjects were required to train three times per week. This was increased to four times from weeks 9 to 16 and then up to five times per week thereafter to a total of 236 sessions over 52wk. Training started with no external trainer resistance applied, pedaling at a cadence of 50 rpm for as long as possible up to 60min or until cadence dropped to about 30–35 rpm, when cyclists were then permitted to assist the legs using their hands to complete the session. When subjects were able to complete three 60min sessions of unloaded, unassisted cycling, trainer resistance was applied to the back wheel from the start of the next session.

The electronically braked cycle trainer did not give an accurate measure of the training work rate in Watts (unlike the motorized cycle used in testing); nonetheless, the trainer was set to the highest resistance level (HRL) that the subjects could pedal against at 50 rpm. Resistance was removed when cadence dropped to about 30–35 rpm to complete the session. Once subjects were able to complete 10min of continuous pedaling against their HRL on three consecutive sessions, a new HRL was introduced at the start of the following session by increasing the trainer resistance one increment. The subjects were exposed to progressive resistance in subsequent training sessions in this manner to ensure that maximum training stimulus was applied for each session.

Testing equipment
Subjects were tested in the laboratory on a bike similar to that used in training (Inspired Cycle Engineering Ltd.) but with a motor and a crankshaft mounted power sensor (SRM Powermeter, Schoberer Rad Messtechnik GmbH, Julich, Germany) fitted. The motor and the power sensor were integrated with control software run on a laptop PC (Toshiba) to allow accurate control of cycling cadence and quantification of leg power output, including the internal work required to rotate the legs. Power output was feedback controlled via automatic adjustment of stimulation intensity (this system is fully described in Hunt et al. [2004]).
Breath-by-breath and intrabreath metabolic gas exchange measures were recorded using a low dead-space portable system in GLA and LON (Metamax II, CORTEX Biophysik GmbH, Leipzig, Germany) and a stationary system in NOT (Oxycon alpha; Jaeger, Hoechberg, Germany). Before each test, the analyzer was calibrated to a known volume and to a certified calibration gas and an ambient air according to the manufacturer’s instructions. HR and oxygen saturation were monitored continuously and recorded every minute using a fingertip sensor linked to a Datex-Ohmeda 3000 pulse oximeter system (Datex-Ohmeda Inc., Madison, WI).

Physiological testing
An incremental work rate test (IWRT) to stimulation saturation (SS) point (100% of each individual’s min–max stimulation charge range) [Ferrario et al., 2007] was performed in the week before commencing cycle training and then after 3, 6, 9, and 12 months where the main outcome measures (detailed later) were estimated. All subjects were familiarized with each test at least 1wk before the baseline tests. Subjects reported for testing rested and in good health. They were instructed to refrain from strenuous exercise or alcohol consumption in the preceding 24h and from consuming food or caffeine in the preceding 2 and 4h, respectively.

The IWRT was conducted at a cadence of 50 rpm. A warm-up of 7min of cycling at the lowest stimulated work rate was followed by a rest period of a minimum of 10min where metabolic gases were required to stabilize with a respiratory quotient between 0.75 and 0.9. Rest was continued until these criteria were met. This was followed by a minimum of 4min of passive motor-controlled cycling where variables were required to stabilize as for the rest period. Stimulation was then applied to allow the power output to increase at an individually predetermined rate of 1 or 2W·min⁻¹ to SS point, chosen to allow the test to be completed within 8-12min [Buchfuhrer et al., 1983]. The test ended with a minimum recovery period of 6min, pedaling at the lowest stimulated work rate.

Outcome measures and analysis
Using a custommade graphical user interface programmed in Matlab (MathWorks Inc., Natick, MA), raw breath-by-breath data were edited to remove mistriggered breaths to minimize the influence of nonmetabolic fluctuations in gas exchange [Röecker et al., 2005]. Peak values were determined over a 60s rolling average, and steady state values were taken as the average of the last 2min of the rest and the passive phases. A relatively large 60-s averaging window was chosen to reduce the distortion caused by data noise [Röecker et al., 2005]. Peak values were measured at SS point to allow valid test-to-test comparisons to be made because tests ended at arbitrarily differing time points beyond this point and often before VO₂ had reached a plateau. The VO₂ of stimulated work only (net VO₂) was calculated by subtracting mean passive exercise VO₂ from VO₂peak. Peak oxygen pulse (O₂ pulse) was calculated by dividing VO₂peak by peak heart rate (HRpeak).

Power data were filtered with a non–phase-shifting low-pass filter with a bandwidth of 25/60Hz (half of the pedal cadence frequency) to ensure that any noise or disturbances occurring more regularly than this frequency were ignored.
$P_{\text{O}_{\text{peak}}}$ was measured as the highest filtered value reached, which always occurred either before or at the SS point after which time the power often dropped in level (Fig. 1).

Figure 1: The power and $\text{VO}_2$ response for one subject from the application of stimulation to stimulation saturation (SS) point and during recovery. Vertical dotted lines indicate SS point (100% of its min–max range), and vertical dashed line indicates the time at which stimulation was reduced to the lowest stimulated work rate. A. The reference power (black) and the actual power response to stimulation (gray). Note the slight dip in power as stimulation reaches 100% saturation. B. The stimulation pulse-width as a percentage of its min–max range. C. The $\text{VO}_2$ response to the incrementing load (a nine-breath average is used here for clarity).
Statistical analysis
Using Minitab 13 software (Minitab Inc., State College, PA), all data and model residuals were examined for normality of variance and distribution (Anderson–Darling test) before analysis and were found not to be different from normal (P > 0.05). Paired t-tests (two-tailed) were then performed between consecutive tests and between each test and baseline values. Where differences reached significance (P ≤ 0.05), the delta values were further analyzed using a summary approach to preserve independence of data [Grafen, 2002]. Multiple Pearson product-moment correlations were run between significant delta values and subject age, weight, height, years post-injury, lesion level, and training duration to examine possible sources of variance. A regression analysis was then performed where these associations were found to be significant. A general linear model was also carried out with sex and stimulation protocol as factors. Differences are expressed in mean absolute terms with the standard deviation (mean±SD) and mean relative to baseline (mean %) where appropriate.

Results

Training
Total training duration was 189±36 h, which comprised a total of 197±34 training sessions of 57.6±5.0min, 3.7±0.6 times per week for 53.3±3.9wk over a period of 57.3±6.2wk. Training frequency compliance (sessions per week) was at its highest at 91% during the first 3 months of training and declined to 85%, 78%, and 75% during the last three quarterly training periods. Total training duration compliance (hours completed) for each training period was 85%, 95%, 78%, and 78%. Continuous cycling capacity increased from 10 to 60min of pedaling over the course of the study for all subjects.

Peak power output
The greatest increase in PO_{peak} occurred within the first 3 months of training (P = 0.02) with a further increase measured between 3 and 6 months (P = 0.009; Fig. 2A). Changes after this time were not significant, resulting in a significant mean relative increase of 132% (P = 0.001) after 12 months. Individual responses ranged from a loss of power of 0.7W to an increase of 25.8W, with final values ranging from 6.7 to 35.6W (for mean values see Table 2).

The increases in PO_{peak} between 0 and 6 months of between 0.77 and 20.82W were significantly related to total training hours completed during this time, which ranged from 59 to 114h. This relationship (r^2 = 0.84, P < 0.001; Fig. 3A) was not found thereafter (r^2 = 0.03, P = 0.60). Additional calf muscle stimulation did not affect PO_{peak} differences between tests or overall (P = 0.36). Variance in pre-training PO_{peak} was explained by sex (P = 0.043), where female values were lower, but this did not account for any of the variance in the magnitude of change (P = 0.46) or absolute values after training (P = 0.14).
Figure 2: Incremental work rate test (IWRT) results showing the delta (Δ) values between each consecutive test for (A) peak power output (PO\textsubscript{peak}), (B) oxygen uptake net of passive oxygen uptake (net VO\textsubscript{2peak}), (C) peak heart rate (HR), and (D) peak oxygen uptake per minute per heartbeat (peak O\textsubscript{2} pulse). Horizontal dashed line represents no change. Data are presented as mean±SEM. Peak values were taken at SS point (100% of stimulation min–max range). Two-tailed paired t-tests were performed between each consecutive test. *Significantly different where P<0.05; **significantly different where P<0.001.
Table 2: Summary of incremental work rate’s test outcome measures

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>0 months</th>
<th>N</th>
<th>3 months</th>
<th>N</th>
<th>6 months</th>
<th>N</th>
<th>9 months</th>
<th>N</th>
<th>12 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>$PO_{\text{peak}}$ [W]</td>
<td>11</td>
<td>8.4 ± 3.2</td>
<td>11</td>
<td>13.4 ± 7.4*</td>
<td>11</td>
<td>17.8 ± 8.6**</td>
<td>11</td>
<td>18.8 ± 7.3**</td>
<td>11</td>
<td>18.4 ± 8.7**</td>
</tr>
<tr>
<td>$VO_{\text{2peak}}$ [ml·min$^{-1}$]</td>
<td>11</td>
<td>543 ± 148</td>
<td>11</td>
<td>651 ± 271</td>
<td>11</td>
<td>819 ± 267**</td>
<td>11</td>
<td>802 ± 277**</td>
<td>11</td>
<td>820 ± 226**</td>
</tr>
<tr>
<td>$\text{net VO}_2$ [ml·min$^{-1}$]</td>
<td>11</td>
<td>247 ± 125</td>
<td>11</td>
<td>374 ± 240*</td>
<td>11</td>
<td>510 ± 241**</td>
<td>11</td>
<td>530 ± 243**</td>
<td>11</td>
<td>524 ± 217**</td>
</tr>
<tr>
<td>$HR_{\text{peak}}$ [bpm]</td>
<td>9</td>
<td>82 ± 8</td>
<td>11</td>
<td>88 ± 12*</td>
<td>11</td>
<td>91 ± 13*</td>
<td>11</td>
<td>94 ± 15*</td>
<td>9</td>
<td>92 ± 16</td>
</tr>
<tr>
<td>$O_2$ pulse [ml·beat$^{-1}$]</td>
<td>9</td>
<td>6.7 ± 1.7</td>
<td>11</td>
<td>7.2 ± 2.2*</td>
<td>11</td>
<td>8.8 ± 2.0*</td>
<td>11</td>
<td>8.5 ± 2.4*</td>
<td>9</td>
<td>9.4 ± 1.1*</td>
</tr>
</tbody>
</table>

Data are means ± standard deviation. All measures were taken at stimulation saturation point.

$PO_{\text{peak}}$: Peak power output; $VO_{\text{2peak}}$: peak oxygen uptake; $\text{net VO}_2$: difference between $VO_{\text{2peak}}$ and passive oxygen uptake; $HR_{\text{peak}}$: peak heart rate; $O_2$ pulse: oxygen uptake per heartbeat

Paired t-tests (two tailed) were performed between each test and baseline values. Significantly different from baseline, where *$P<0.05$ or **$P<0.001$. 

Cardiorespiratory adaptations.
All significant change in VO$_{2peak}$ occurred between 3 and 6 months (P = 0.003). However, when expressed net of passive VO$_2$, net VO$_{2peak}$ had significantly increased between 0 and 3 months (P = 0.023; Fig. 2B) with a mean relative increase of 168% overall (P < 0.001; for mean values see Table 2). The changes over the first 6 months were significantly related to total training hours completed ($r^2 = 0.52$, $P = 0.012$; Fig. 3B).

![Figure 3: Regression plots showing the relationship between total training duration in hours and changes in (A) peak power output (POpeak) between 0 and 6 months where $r^2 = 0.84$, $P < 0.001$; and (B) changes in net peak oxygen uptake (VO$_{2peak}$) between 0 and 6 months where $r^2 = 0.52$, $P = 0.012$. The equations for each regression line are shown on each plot. Data are for 11 subjects.](image)

HR$_{peak}$ (Fig. 2C) increased by 13% after 6 months ($P = 0.008$), but by 12 months, the increase just failed to reach significance ($P = 0.057$; Table 2). Peak O$_2$ pulse had increased ($P = 0.002$) by 6 months, with no significant change thereafter ($P = 0.85$; Fig. 2D), leading to a mean relative increase of 35% after 12 months ($P = 0.002$; Table 2). None of the variance in any of the outcome variables was significantly explained by stimulation protocol, years post injury, lesion level, or age.
Discussion

The aim of this study was to examine the extent to which progressive, high-volume, home-based ES cycle training could improve cardiorespiratory fitness and cycling power in paraplegic individuals. Due to substantial muscle disuse atrophy after SCI, internal work may represent a relatively large proportion of total work done, and in some cases subjects may not even be capable of producing sufficient internal work to overcome frictional losses [Hunt et al., 2007]. This is the first longitudinal study to measure and to account for this work when quantifying total power output during ES cycling tests. Furthermore, work rate was able to be increased by arbitrarily small increments (here 1 or 2W) during the IWRT, unlike the 1/8kp or approximately the 6.1W increments (assuming a cadence of 50 rpm) used during all previous ES cycling studies. This permitted a sensitive training dose–response analysis to be made for the first time in ES exercise. This type of protocol is also particularly important for examining cardiorespiratory responses such as gas exchange thresholds that require analysis with a high temporal resolution. Although the absolute magnitude of any change in power of less than 6.1W is small, the relative change is substantial and may reflect clinically significant adaptations.

The highest individual PO\textsubscript{peak} value of 35.6W (internal plus external work rate) achieved in this study after 12 months of training is similar to that measured by the same technique in a case study subject with tetraplegia after a similar training program [Kakebeeke et al., 2008]. Nonetheless, there seem to have been no greater gains in power by training more frequently or for longer durations than that in previous studies: The PO\textsubscript{peak} value here is also similar to the highest work rate (external work measured only) achieved by Ragnarsson et al. [1988] during training after only 36 sessions over 12wk. They found that their subjects (N = 19) could cycle within a range of 0 to 36W for 15min, most of whom (n = 17) cycled at between 0 and 12W. After only twenty-four 30min sessions of exercise (about 8wk), Barstow et al. [1996] reported a mean PO\textsubscript{peak} of 14.5±5.6W similar to the PO\textsubscript{peak} value of 13.5±10.7W found in this study after 39 60min sessions (about 12wk).

Studies have used either continuous [Barstow et al., 1996] or discontinuous [Figoni et al., 1990] test protocols using 5min work rate increments of about 6W (1/8kp at 50 rpm) to peak exercise tolerance. The protocol adopted by Figoni et al. [1990] interspersed four 5min bouts of ES cycling with 4min of passive exercise and rest. In addition to having many incomplete lesion subjects, their protocol, in contrast to that used by Barstow et al. [1996] and by this study, may have allowed for sufficient muscle recovery during the rest and the passive exercise intervals to account for the relatively high power output values recorded by their untrained subjects (15±7W). The methods or the calculations used for determining PO\textsubscript{peak} values are not detailed in these studies, where it is not clear whether PO\textsubscript{peak} values were estimated by linear extrapolation between the 6W increments during the final 5min increment or given as the final work rate tolerated. The combination of differences in subject group, test protocol, and PO\textsubscript{peak} calculation methods makes direct comparison between studies difficult, if not invalid.

There are two substantial differences between voluntary activation in able-bodied subjects and transcutaneous electrical stimulation of persons with SCI. First, the axons are recruited in a disorderly way [Gregory and Bickel, 2005], neither orderly according to the Henneman size principle (small to large) nor the reverse as if the electrodes were close to the nerve trunk. Second, most muscle fibers become Type IIx [Pette, 2005], and although some may
convert toward slower phenotypes as a result of the training, the pre-injury relationship between axon diameter and muscle type will not be restored. We should therefore expect that as the stimulation intensity increases toward SS, more and more muscle fibers will be recruited but the proportion of the types will remain constant (and probably predominantly Type IIx). The combination of stimulation application rate and muscle fiber fatigue rate will determine the momentary cross-sectional area of recruited muscle mass available for power production; during cycle training, the rapid stimulation application rates (60s to SS) and the initial HRL would result in a relatively high short-term anaerobic power output followed by fatigue to a sustainable power output. This would reflect the mean balance between fiber fatigue and recovery rates and the muscle’s oxidative capacity, notwithstanding the effects on power of possible antagonist cocontraction or muscle spasms. The power profile would be similar to that observed during volitional all-out cycling [Vanhatolo et al., 2007], and indeed, this has been observed by Theisen et al. [2002], except that they found power to recover slightly after the initial drop from the highest power output.

During the IWRT, however, stimulation and load application rates were progressive over 8 to 12min, by which time a degree of muscle fatigue is likely to have already occurred. Power values recorded at this time are then likely to be lower than those that could be produced at the start of each training session, when muscles are fresh and stimulation application rate is rapid. This could also explain some of the differences in PO_{peak} values measured across studies, where load and stimulation application rate have either varied between subjects [Mohr et al., 1997b] or have not been detailed, and the time of PO_{peak} measurement has not been given. Therefore, for future studies, the test protocol and the manner and time at which PO_{peak} is measured should be clearly stated to clarify which type of power is being measured, that is, peak explosive power, peak IWRT power, or endurance power.

This is the first study to report a significant and robust relationship between the magnitude of change in PO_{peak} and the total duration of training. However, this relationship was found only during the first 6 months. During this time, when both training resistance and volume were progressive, the greatest training duration of 114h saw the greatest improvement in PO_{peak} of 20.8W. Kakebeeke et al. [2008] and Mohr et al. [1997b] also observed that significant increases in power occurred only within the first 6 months of training. In the present study, although training was always performed against a maximally tolerated trainer load, training diaries revealed that this was not able to be increased after about 6 to 9 months of training. This limitation could be physiological in nature or perhaps due to the training protocol or the stimulation strategies used and merits further investigation.

The overall loss in power of 7% for one individual was explained by an examination of his training diary: After a successful training period between 3 and 6 months where he recorded an increase in PO_{peak}, his training became erratic and he took a 5wk holiday in the 6wk before his 9-month test and then completed only 27 of the expected final 65 training sessions. Discounting possible measurement error, this degree of reversibility in training adaptations is nonetheless quite remarkable.

The VO_{2peak} tests provide an indication of the maximal oxidative capacity of the stimulated muscle mass, not of the maximum systemic cardiorespiratory capacity (VO_{2max}) because upper body movement is minimal or absent. This is evident where volitional upper-body exercise is performed in conjunction with ES cycling, and the combined exercise elicits a higher VO_{2peak} [Heesterbeek et al., 2005; Mutton et al., 1997; Raymond et al., 1999].
Nonetheless, the $\text{VO}_{2\text{peak}}$ values here provide a valuable insight into the metabolic stress that can be achieved by ES cycling exercise alone.

The mean improvement in IWRT $\text{VO}_{2\text{peak}}$, which was strongly related to training duration over the first 6 months, equated to just over 1 metabolic equivalent (MET, about 250ml·min$^{-1}$ for men and 200ml·min$^{-1}$ for women). Considering that an increase in fitness of only 1 MET is associated with a mortality benefit of about 20% [Warburton et al., 2006a], then it would seem that it is possible for ES cycle training alone to be sufficient to reduce the health risks associated with inactivity, especially because cycling sessions were sustained for twice as long the recommended duration for this intensity of work [ACSM guidelines, 2000; Warburton et al., 2006a]. This is particularly important for previously sedentary individuals as they become active, but the plateau reached in $\text{VO}_{2\text{peak}}$ values (which are substantially lower than those that can be expected after similar periods of volitional cycling or running) illustrates the serious limitations of this type of exercise for further improvements in aerobic capacity for this subject group.

The highest $\text{VO}_{2\text{peak}}$ value of 1.17l·min$^{-1}$ found here is similar to those previously reported after training regimes of much lower frequencies and durations. The comparatively low $\text{VO}_{2\text{peak}}$ values attained in this study may be explained by the differences in data treatment and analysis found across studies rather than to a poorer exercise response; Mohr et al. [1997a] reported a mean $\text{VO}_{2\text{peak}}$ of 1.43±0.09l·min$^{-1}$ for 10 subjects and a highest individual value of 1.48l·min$^{-1}$, but it seems that, unlike this study, the data were neither edited nor averaged before analysis, with peak values given as the highest absolute values within a 2-min period. This could lead to erroneously high estimates of peak values, distorted by outlier values, especially where the noise to signal ratio is high [Röecker et al., 2005]. The mean sustainable $\text{VO}_2$ during training may provide another, more meaningful, indicator of aerobic capacity for this subject group and for this type of exercise. This is currently under investigation but is beyond the scope of this article.

The increase in cycling endurance capacity from 10 to 60min reflects improvements in muscle fatigue resistance and in oxidative capacity, with fibers likely to have transformed from fast, fatigable glycolytic toward more glycolytic–oxidative fatigue-resistant isoforms [Pette, 2005]. Further investigations, including an examination of changes in muscle mass and phenotype, $\text{VO}_2$ kinetics, and energetics during ES cycling, are needed in an attempt to understand the underlying physiological adaptations to this unique exercise modality.

Although $\text{HR}_{\text{peak}}$ increased by 13% after 6 months, it was not significantly different from pretraining levels by the end of the training program, and the posttraining HR of 92 ± 16 beats·min$^{-1}$ equated to only 53% of the mean age predicted maximum, suggesting that exercise limitations are peripheral and not central in nature.

The overall 35% increase in $\text{O}_2$ pulse indicates improvements in tissue $\text{O}_2$ extraction or to an increase in stroke volume (SV) or to both. Increased SV, which provides a more beneficial myocardial stress than an increase in HR, occurs during ES leg exercise due to the activation of the venous muscle pump [Figoni et al., 1990] and has been found to be greater after ES leg cycle training than after arm cycle training [Mutton et al., 1997]. The mean post-training $\text{O}_2$ pulse value was similar to the value observed by Barstow et al. [1996] after only 24 exercise sessions, but again, direct comparisons are difficult because $\text{VO}_2$ data treatment was not detailed.
This is the first ES cycling study where subjects were required to train for 60min per session for up to five sessions per week for 52wk. Similar to the findings of an earlier case study where a subject with tetraplegia followed a similar training program [Kakebeeke et al., 2008], training frequency and duration reached a peak between 3 and 6 months and then declined slightly thereafter. It seems that this high-frequency duration and therefore overall volume of training, higher than any other ES cycling study to date, was neither feasible nor sustainable in the long term. The time taken to prepare for and complete each training session (about 2h) represents a substantial weekly time commitment, especially for those working full time or those with family responsibilities, and requires a great deal of motivation and family support to complete. The training plateau reached by 6-9 months may have affected motivation levels.

Conclusions

In conclusion, the current training resulted in significant, training volume-dependent cardiorespiratory and cycling power output adaptations during the first 6 months of training when training frequency, duration, and load were progressive. The upper limits in load tolerance were met during this training program, and it is not known whether this is due to a physiological limitation or to limitations in the stimulation strategy and the training protocol used. Further study is merited to develop and to evaluate different stimulation, loading, and training strategies specific to ES exercise to optimize favorable training responses with lower training volumes for this subject group.
4.3  Energetics of paraplegic cycling: a new theoretical framework and efficiency characterisation for untrained subjects

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Introduction

Efficiency
To paraphrase the opening lines of Winter [1979]: in the assessment of the energetics of human movement, it is important to have an accurate measure of the total mechanical work done by the body, including both internal work and external mechanical work.

Winter [1979] went on to examine methods for estimation of internal work and efficiency of normal subjects walking or running. However, the above observation is especially true for subjects with a severe physical impairment: external work may be very small and therefore internal work may represent a large proportion of the total work done.

It may even be the case that the subject is unable to produce a sufficiently high work rate to overcome internal losses completely: some form of assistance is then required to maintain motion (e.g. motor-assist for cycling [Hunt et al., 2004], or driven-gait orthoses for walking [Colombo et al., 2000]). In such cases, a correct measure of internal work is essential.

It is therefore necessary to use modified equations for calculation of efficiency which account appropriately for internal work done. Moreover, efficiency measures depend upon the notion of “useful” work, and are therefore not unique: useful work must be defined in the specific context under study, and may in part incorporate some elements of internal work. Traditional measures of efficiency regard internal work as an unmeasurable loss which can be neglected in the calculation [Whipp and Wasserman, 1969; Gaesser and Brooks, 1975]. This may be appropriate when the subject is able to produce a large magnitude of external work. On the other hand, this will lead to large errors, or even the impossibility of performing the calculation, when subjects with severe physical impairment are under study.

Here we review traditional efficiency measures and propose modified measures which are appropriate to subjects with severe physical impairment performing cycle ergometry. In this context, internal work is associated with the energy cost of rotating the mass of the legs to
overcome frictional losses in the system; this type of work can be considered as useful muscular work. We describe cycling apparatus which allows this form of internal work to be accurately measured: it can therefore be included in the calculation of efficiency. The new methodology is employed to characterise the efficiency of a group of untrained paraplegic subjects performing cycle ergometry by means of functional electrical stimulation of the large leg-actuating muscles. The subjects have total paralysis of the lower limbs as a result of spinal cord injury.

The ability to characterise efficiency in this way, even for extremely weak, untrained subjects, may provide a useful tool for the study of technical methods for optimizing the efficacy of stimulation strategies, or for monitoring changes in efficiency within and between subjects during a training intervention.

Paraplegic cycling
Spinal cord injury (SCI) results in varying degrees of muscle paralysis, loss of sensation and disruption of autonomic nervous system function. Complete lower-limb paralysis precludes volitional leg exercise, and leads to severe muscle atrophy and physiological de-conditioning. Paralysed muscle can usually be activated by electrical stimulation. Following a period of re-training, limited but useful function can be temporarily restored.

An example of this is stimulated leg cycling exercise. It was established in the early 1980s that lower-limb cycling exercise can be achieved in complete SCI by means of controlled sequential stimulation of the leg musculature [Petrofsky et al., 1983; Petrofsky et al., 1984; Eichhorn et al., 1984; Kern et al., 1985]. A range of subsequent studies have shown that this form of exercise can lead to positive physiological adaptations and general health benefits [Janssen et al., 1998].

While many physiological studies have used stationary cycling ergometers, a number of mobile devices have also been put forward; these include two of the original contributions [Petrofsky et al., 1983; Kern et al., 1985] and a number of more recent proposals [Pons et al., 1989; Petrofsky and Smith, 1992; Göehler et al., 1998]. Our work is based upon adapted recumbent tricycles, which in their basic form are suitable for mobile cycling [Perkins et al., 2001; Perkins et al., 2002; Hunt et al., 2002], and with motor support and additional instrumentation can be used for physiological investigation [Hunt et al., 2004], as in the present work.

Using traditional measures of efficiency, it has previously been reported that the mechanical efficiency of stimulated cycling exercise is lower by a factor of ~1/3 than volitional cycling. This, together with the weakness of paralysed muscle, results in low power output [Glaser et al.; 1989; Raymond et al., 2002; Theisen et al., 2002]. It has been argued [Glaser et al., 1989] that the low efficiency of stimulated cycling is of little consequence in the context of stationary ergometry since a key objective is to elicit metabolic and cardiopulmonary responses, while the external energy output is not harnessed to do useful external work. In mobile cycling, on the other hand, maximisation of efficiency is clearly of the utmost importance because in this case effective production of external work is required for propulsion of the rider-cycle mass.
We set out to explore the nature of the inefficiency of work performed by stimulated, paralysed muscle, by developing an extended theoretical framework tailored specifically to impaired subject groups. This new framework accounts appropriately for useful internal work, no matter how small, and is therefore applicable to very weak, untrained subjects as well as to stronger subjects.

Little is presently known about the energetics of paraplegic cycling in untrained subjects following several years of paralysis. In an experimental application of the new energetics framework, we quantified the total work efficiency, oxygen cost and sustainable work rate of stimulated cycling exercise in ten untrained subjects with complete lower-limb paralysis. Despite the very small (usually negative) power outputs, we were able to make these measurements using our special exercise tricycles which have power assistance to maintain the cadence [Hunt et al., 2004], and we were able to perform the efficiency calculations using the new theoretical framework. We then compared these measures with data from the literature describing the efficiency of anaesthetised healthy cyclists [Kjaer et al., 1994], trained subjects with paraplegia [Glaser et al., 1989] and able-bodied individuals [Whipp and Wasserman, 1969; Gaesser and Brooks, 1975] in an attempt to identify ways in which more effective stimulation paradigms might be obtained.

Theory
The efficiency $\eta$ of an energy-transfer process is defined generally as the ratio of useful work accomplished to the energy expended within a given time interval:

$$\eta = \frac{\text{work done}}{\text{energy cost}} \times 100\% \quad (1)$$

Equivalently, efficiency is the ratio of average power output to average power input over the time interval. In the context of human movement, “useful work” may comprise both internal and external work, while the energy cost is related to metabolic processes. This leads to the general definition of efficiency of human movement proposed by Winter [1979]:

$$\eta = \frac{\text{internal + external mechanical work}}{\text{metabolic energy cost}} \times 100\% \quad (2)$$

A range of specific efficiency measures has evolved. These differ principally in the way in which components of internal mechanical work are accounted for in the numerator, and in the way in which corrections are employed in the denominator of the efficiency equation for baseline metabolic cost (e.g. the metabolic energy turnover at rest).

Standard measures of efficiency
In cycle ergometry, traditional efficiency measures neglect internal work (i.e. work required just to overcome frictional losses associated with rotating the mass of the legs), and
sometimes employ baseline metabolic corrections for the resting or “unloaded” conditions. Thus, if during steady-state cycle ergometry we define the external output power as $P_{\text{out}}$, the total metabolic input power as $P_{\text{in}}$, the metabolic power during “unloaded” cycling (i.e. cycling at an output power of $P_{\text{out}} = 0$W) as $P^0$, and the rate of metabolic energy consumption at rest as $P_r$, then the following efficiency measures can be established [Gaesser and Brooks, 1975; Glaser et al., 1989] (see also Fig. 1):

Figure 1: Representation of standard measures of efficiency (not to scale). $P_r$ is the rate of metabolic energy consumption at rest and $P^0$ is the metabolic power during unloaded exercise. At a given exercise operating point, $P_{\text{in}}$ represents the total metabolic input power and $P_{\text{out}}$ is the external mechanical output power. The slopes of the lines indicated are inversely proportional to the corresponding efficiency measure. For example, for net efficiency, rearrangement of Eq. 4, expressed in absolute rather than percentage terms, yields $P_{\text{in}} = \frac{1}{\eta_n} \cdot P_{\text{out}} + P_r$, which is a line having slope $1/\eta_n$ joining the points $(0, P_r)$ and $(P_{\text{out}}, P^0)$.
Here, all power values are normally taken as average values during some period of steady-state operation. The definition of delta efficiency in Eq. 6 is based upon any increment in output power and the corresponding increment in input power. Clearly, if, as in Fig. 1, the overall relationship between input and output power is linear, then work efficiency and delta efficiency are equal over the whole work rate range.

Figure 1 also reveals an advantage which the work and delta efficiency measures have over the gross and net calculations: if we consider the line joining the points \((0, P^0)\) and \((P^{out}, P^{in})\) as a continuum of steady-state-exercise operating points, then, clearly, \(\eta_w\) and \(\eta_b\) are independent of the operating point and are constant over the whole operating range. Gross and net efficiencies, on the other hand, are dependent on the operating point: the higher the exercise intensity, the higher are the calculated values of \(\eta_g\) and \(\eta_n\) (since the slopes of the dashed lines in the figure decrease).

This bias associated with calculation of \(\eta_g\) and \(\eta_n\) arises for two reasons: for \(\eta_n\), the metabolic cost above rest of unloaded exercise is accounted for in the denominator of Eq. 4 by subtracting only the resting work rate from \(P^n\), while the associated internal mechanical power required to move the legs is neglected in the numerator; for \(\eta_g\), the denominator of Eq. 3 includes the metabolic cost of both unloaded cycling and the resting metabolism, while the internal mechanical work rate associated with moving the legs is again left out of the numerator.

These difficulties relating to \(\eta_g\) and \(\eta_n\) were previously noted (Whipp and Wasserman, 1969; Gaesser and Brooks, 1975), and the superiority of work efficiency (and, by extension, delta efficiency) highlighted.

### Metabolic cost

In the discussion of exercise physiology, energy expended in doing sustainable steady-state work is obtained from the oxygen requirement of the work and the estimated energy value of the metabolic substrate. The oxygen requirement is defined for steady-state aerobic exercise as the volumetric rate of pulmonary oxygen uptake, denoted \(V_O^2\) [l·s\(^{-1}\) or ml·min\(^{-1}\)]. The approximate energy value of the substrate for every unit of oxygen consumed is taken to be in the range 19.59–21.14kJ·l\(^{-1}\), and is derived from the respiratory exchange ratio (RER). RER is defined as the ratio of the rates of pulmonary carbon dioxide output (\(V_{CO_2}\)) and oxygen uptake, i.e. \(RER=V_{CO_2}/V_O^2\). An RER of 0.7 corresponds to an energy value of 19.59, an RER of 1.0 has an energy value of 21.14, and intermediate values are obtained by linear interpolation.

Metabolic energy expenditure rates (\(P^n\), \(P^0\) and \(P^r\)) are therefore computed as the product of oxygen uptake and substrate energy value in each condition of interest (exercise, unloaded and rest). As an example, consider the following development of the net efficiency equation 4:

\[
\eta_n = \frac{P^{out}}{P^{in} - P^r} \times 100\% = \frac{P^{out}}{V_O^2 E^{in} - V_O^2 E^r} \times 100\% \tag{7}
\]
Here, VO$_2$"n and VO$_2$' represent the oxygen uptake rate at a workrate of P$_{\text{out}}$ and at rest, respectively. E"n and E' are the corresponding energy values of the metabolic substrate, per volume-unit of oxygen uptake. These are dependent on the steady-state RER in each condition, as described above. The quantities in Eq. 7 are normally taken as average values over time intervals of finite duration.

Extended measures of efficiency
The efficiency measures defined above may not always be appropriate, particularly in impaired subject groups capable of only low work rate magnitudes. It is apparent from Fig. 1 that the higher the output power (for a given input power), the closer the efficiencies of Eqs. 3–6 become, and that at high power levels, small changes in power output have only a small relative effect upon net and gross efficiency (under the assumption of linearity, work and delta efficiencies are independent of the operating point).

At low levels of power output, however, both gross and net efficiency become small, and both these measures become sensitive to changes in power output (e.g. a change in P$_{\text{out}}$ from 1 to 3W will cause an almost threefold increase in gross efficiency, if the metabolic cost is relatively unchanged).

It is also important to recognise that in impaired subjects operating characteristically in the low power output range, the power required just to keep the legs moving in the absence of an external load may be a large proportion of the total workrate range and may in fact exceed the capacity of the muscles. It is desirable, therefore, that the work done in moving the mass of the legs be considered useful work and, as such, should be accounted for in the efficiency calculations.

Technically, this can be achieved using a motorized ergometer system capable of turning the legs at the required testing cadence, even in the absence of muscular work. Also required is a torque sensor at the ergometer crank axis which can measure both positive and negative values (negative torque will be measured when muscular work is either absent or insufficient to fully turn the mass of the legs). Such a system is described in the sequel.

This situation is illustrated in the generalised power input-output relationship in Fig. 2, which accounts for the negative workrate range. When the legs are turned at the desired cadence by the motorised ergometer, and no muscular input is involved, a negative power output will be measured: this is referred to as “passive” cycling, and the measured power in this condition is denoted P–. Thus, the total workrate range, Pt, is given by Pt = P$_{\text{out}}$ – P–. The metabolic power measured during “passive” cycling is Pp. We note that, in general, the passive metabolic power Pp may be different from the resting metabolic rate Pr. (This may arise, for example, from reflex muscle activity caused by joint motion.)
Figure 2: Representation of extended measures of efficiency (not to scale). $P^r$ is the rate of metabolic energy consumption at rest, $P^p$ is the metabolic power during passive exercise, when the measured external mechanical work rate is $P^-$, and $P^0$ is the metabolic power at an external mechanical work rate of 0W. At a given exercise operating point, $P^{in}$ represents the total metabolic input power and $P^{out}$ is the external mechanical output power. The total mechanical work rate, incorporating both internal and external mechanical work, is $P^t = P^{out} - P^-$

With these definitions we can introduce an alternative gross efficiency measure (the “total” gross efficiency) as:

Total Gross Efficiency : $\eta^t_g = \frac{P^t}{P^{in}} \times 100\%$ (8)

Clearly, in general we have $\eta^1_g > \eta_g$ (cf. Eq. 3).

If net efficiency is defined, as before, as the efficiency with respect to a resting baseline correction, then we obtain the same definition as previously (Eq. 4) since the resting power output is zero. However, this concept of net efficiency has little utility in this new setting since the negative work rate range cannot be incorporated and, indeed, any operating point with $P^{out} < 0$ would give a negative efficiency value. It makes sense only to define efficiency measures which proceed from the passive power level $P^-$. 
Work efficiency is defined in a similar way as before, except that useful power output is considered to occur from the negative level $P$ onwards (rather than from 0W). We therefore incorporate total mechanical work rate $P^t$ and the passive metabolic baseline correction into the definition:

$$\text{Total Work Efficiency} : \eta_w = \frac{P^t}{P_{\text{in}} - P^p} \times 100\% \quad (9)$$

Note that for positive workrates, $P^{\text{out}} > 0$, and under the assumptions of linearity and noise-free measurement over the whole workrate range, the two definitions of work efficiency, Eqs. 5 and 9, deliver identical values (cf. Figs. 1 and 2). The advantages of the new definition, Eq. 9, are that it is valid also for negative workrates, and it is expected to be less numerically sensitive at positive, but low, power outputs to small fluctuations in its input variables (i.e. $P^t$ and $P^n - P^p$, in contrast to $P^{\text{out}}$ and $P^n - P^0$ in Eq. 5).

The definition of delta efficiency in this case does not differ from that in Eq. 6. As before, we note that when the input-output relationship is linear, then total work efficiency and delta efficiency are equal over the whole workrate range.

In summary, the definitions of gross efficiency in the two cases differ. The extended definition is expected to lead to better numerical properties. Net efficiency is only well defined in the standard case, but the potential numerical difficulties have been noted. Work efficiency has a new definition in the extended-workrate situation, with improved numerical properties and validity at negative absolute workrates. Theoretically, both definitions deliver the same result when $P^{\text{out}} > 0$ and under assumptions of linearity and noise-free measurement. Delta efficiency is the same in both cases.

In view of this discussion, the preferred efficiency measure in the context of subjects with severe physical impairment performing cycle ergometry is the Total Work Efficiency defined by Eq. 9. This quantity incorporates both internal and external mechanical work as “useful work” and subtracts the passive metabolic energy turnover as an appropriate metabolic loss associated with passive cycling.

**Experimental Methods**

**Subjects**
We studied ten subjects (nine male, one female) with complete lower-limb paralysis resulting from traumatic SCI sustained at least 2 years previously. The age of the subjects was 41.5±8.0 years (mean±standard deviation), their body mass was 78.4±14.6kg and their height was 1.76±0.07m. The work was approved by the appropriate local ethics bodies: the ethics committees of the Southern General Hospital and of the Faculty of Biomedical and Life Sciences at the University of Glasgow (Glasgow); the ethics commission of Kanton Luzern (Nottwil); and the research ethics committee of Kings College Hospital (London). Subjects provided written, informed consent prior to participation.
Exercise testing
These subjects, who had not previously performed stimulated leg-cycling exercise, sat on a recumbent tricycle (Inspired Cycle Engineering Ltd, UK) which we modified for use by subjects with paraplegia in combination with electrical stimulation ([Hunt et al., 2004], Fig. 3).

Cycling was achieved through the combination of a feedback-controlled electric drive motor and by coordinated stimulation of the main leg-actuating muscles. The motor was programmed to maintain cycling at a constant cadence. Together, the motor and the stimulated legs worked against a constant load at the tricycle’s rear drive wheel. A sensor fitted to the crankshaft (SRM Powermeter, Schoberer Rad Messtechnik GmbH, Germany) allowed measurement at the cranks of the passive work and the work resulting from stimulation of the leg muscles, independently of the motor’s contribution. During “passive cycling” (i.e., cycling with stimulation switched off) the legs were turned by the motor alone, resulting in measurement of a negative work rate $P^-$ at the crankshaft, the magnitude of which corresponds to the rate of work required just to rotate the legs to overcome frictional losses. During stimulated cycling, the torque measured at the cranks allowed determination of $P^{\text{out}}$ and thus the total exercise work rate (i.e. the rate of muscular work due to stimulation) as $P^l = P^{\text{out}} - P^-$. Timing of stimulation is arranged such that muscle activation occurs only during muscle shortening, which supports our assumption that frictional losses are the same during passive and active situations. A feedback control system maintained a constant, pre-specified work rate by automatic adjustment of the stimulation intensity [Hunt et al., 2004].

For muscle stimulation, adhesive electrodes were placed on the skin surface over the quadriceps (Q), hamstrings (H) and gluteal (G) muscles and connected to an electronic stimulator (Stanmore Stimulator, [Phillips et al., 1993]). A sensor on the tricycle continuously measured the crank angle, and this signal was processed by software which controlled the activation of each muscle group to ensure that the muscles contributed positively to cycling torque. The three muscle groups for the right side were typically active during the following crank-angle ranges: Q 55–155°; H 188–265°; G 90–180°. Here, 0° is at horizontal with the leg flexed. Stimulation ranges for the left side were phase shifted by 180°. Stimulation frequency was 50Hz, maximum current was 130mA (set on an individual basis for each subject), and maximum pulse duration was 510μs.
Following a period of rest, and a 4min period of passive cycling at 50 rpm, stimulation was initiated and cycling was performed at a constant work rate for a period of up to 20min. The constant work rate level for each subject was chosen based on a prior maximal power test (performed on a separate day). The level of constant work rate was set as a fixed percentage of maximal power to try and ensure subjects could maintain 20min without fatigue. In the constant work rate tests, nine of the ten subjects completed the 20min stimulated cycling phase. One subject achieved only 10min prior to fatigue, indicated by the measured work rate starting to fall off from the target after stimulation reached its maximum intensity. This duration is sufficient to allow the steady-state efficiency calculations to be included for this subject.

We measured the rates of oxygen uptake (VO\textsubscript{2}) and carbon dioxide output (VCO\textsubscript{2}) using a breath-by-breath respiratory monitoring system (MetaMax 3B, Cortex Biophysik GmbH, Germany). The system comprised a low dead-space mask, gas analysers for continuous measurement of respired O\textsubscript{2} and CO\textsubscript{2} concentrations, and a turbine for measurement of flow rate. The turbine was calibrated using a volumetric syringe, and gas analyser calibration was verified using ambient air and a certified calibration gas mixture immediately before each test.

Outcome measures
The oxygen cost of stimulated cycling exercise was calculated as the ratio of the increase in the rate of oxygen uptake above that during passive cycling $\Delta$VO\textsubscript{2} to the total mechanical work rate $P^t$:

$$\text{oxygen cost} = \frac{\Delta V_O}{P^t} [\text{ml min}^{-1} \text{W}^{-1}]$$

(10)

Efficiency was calculated as the total work efficiency given by Eq. 9, i.e. the ratio of the total mechanical work rate and the approximate net energetic cost of the exercise (i.e., the energetic cost during cycling minus the cost during passive cycling):

$$\eta_w = \frac{P^t}{P^t - P^p} \times 100\% = \frac{P^t}{(V_{O_2}^n E^n - V_{O_2}^p E^p)} \times 100\%$$

(11)

Here, $V_{O_2}^n$ is the average oxygen uptake rate during steady-state exercise over a given time interval and $V_{O_2}^p$ is the average oxygen uptake rate during steady-state passive cycling. $E^n$ and $E^p$ denote the energy equivalents of the oxygen in each state (exercise or passive, respectively). These are estimated as described above (Sect. “Metabolic cost”).

The denominator of the efficiency equation 11 is an approximation of the true energy cost of muscular work performed during the exercise: a small component of the oxygen consumed may be associated with processes other than muscular contraction (e.g. lactate clearance), tending to cause efficiency to be underestimated; but any anaerobic contribution to muscle
force production is neglected, thus leading to a possible overestimate of efficiency. Since transcutaneous nerve stimulation has been seen to recruit both aerobic and anaerobic motor units in a synchronous and non-selective manner [Gregory and Bickel, 2005], we would suggest that the latter is likely the more appreciable effect and therefore Eq. 11 represents an estimated upper bound on total work efficiency.

The above outcome measures, Eqs. 10 and 11, were computed during the last 5min of the constant-load exercise phase, to ensure that steady-state conditions prevailed. The oxygen uptake and carbon dioxide output variables were edited to remove outliers prior to the calculations in Eqs. 10 and 11.

Results

The estimated efficiency of the exercise (i.e. the total work efficiency $\eta_{\text{tw}}$, Eq. 11), averaged across all subjects, was 7.6±2.1% (mean±standard deviation) and the oxygen cost (Eq. 10) was 38.8±13.9ml·min$^{-1}$·W$^{-1}$. The sustained total mechanical work rate $P_t$ was 6.2±2.9W (with an absolute external power value $P_{\text{out}}$ of -1.6±3.9W), at a stimulation intensity of 68±29% of the maximum stimulation level.

Typical results of tests with two different subjects are shown in Figs. 4 and 5 (the second result is included as an example of a subject having an absolute work rate of less than zero during exercise). In the plots, power data have been low-pass filtered using a second-order Butterworth filter with bandwidth 0.2Hz. Oxygen uptake plots show 30s-averaged data. For the second example (Fig. 5), we provide a sample efficiency calculation showing the steps involved in computing the total work efficiency outcome defined by Eq. 11. In this case, the measured passive power was $P^-$ = -8.31W (taken as an average from the pre- and post-exercise passive phases) and the average external output power during the final 5min of steady-state exercise was $P_{\text{out}}$ = -3.03W, thus giving a total mechanical power $P_t = P_{\text{out}} - P^- = +5.27W$. During the final 2min of the passive phase the average RER was 0.85 and during the final 5min of the constant-load exercise phase it was 1.10. Thus, truncating the latter value to 1 and using the interpolation approach described in Sect. “Metabolic cost”, the energy equivalents during the passive and exercise phases are found to be $E^{p} = 20.35kJ\cdot l^{-1}$ and $E^{in} = 21.14kJ\cdot l^{-1}$. The average steady-state oxygen uptake during the final 2min of passive cycling and the final 5min of constant-load cycling were measured to be $VO_{2}^{p} = 0.316l\cdot min^{-1}$ and $VO_{2}^{in} = 0.486l\cdot min^{-1}$: The above values can now be substituted in the total work efficiency equation 11:

\[
\eta_{\text{tw}} = \frac{P_t \times 100\%}{(V_{O2}^{in} E^{in} - V_{O2}^{p} E^{p})} = \frac{5.27 \times 100\%}{(0.486 \times 21.14 \times 10^3 - 0.316 \times 20.35 \times 10^3) / 60} = 8.23\%
\]
Figure 4: Test result from one subject: power output from the stimulated legs (top); stimulation intensity, expressed as a percentage of the maximum pulsewidth (middle); Oxygen uptake response (bottom). The increments in exercise work rate ($P^t$) and oxygen uptake ($\Delta V_O^2$) used in the outcome calculations (see Eqs. 10 and 11) are indicated in the top and bottom plots.
Figure 5: Test result from another subject: power output from the stimulated legs (top); stimulation intensity, expressed as a percentage of the maximum pulsewidth (middle); oxygen uptake response (bottom). The increments in exercise work rate ($P_t$) and oxygen uptake ($\Delta V_{O_2}$) used in the outcome calculations (see Eqs. 10 and 11) are indicated in the top and bottom plots.

Discussion

Using the standard measure given by Eq. 5, the work efficiency of a group of trained paraplegic cyclists was previously reported to lie in the range 7–13% [Glaser et al., 1989], whereby the lower end of this range corresponded to the upper end of the subjects’ achievable power output range. Kjaer et al. [1994] studied a group of eight able-bodied individuals performing stimulated cycling exercise under complete epidural anaesthesia. Using data published in the latter reference, and employing the standard equation 5, we estimated the work efficiency of these eight subjects during cycling to be 9.9%.

The total work efficiency of 7.6% obtained here using the new definition of Eq. 11 is within the range of 7–13% for trained paraplegic cyclists [Glaser et al., 1989] and is slightly lower than the 9.9% for anaesthetised able-bodied individuals [Kjaer et al., 1994]. But each of these values compares poorly to work efficiencies of up to ~30% (standard calculation) for able-bodied subjects cycling under normal volitional control [Whipp and Wasserman, 1969; Gaesser and Brooks, 1975]. This outcome is reflected in the oxygen cost which is higher by
a factor of ~3.5 than that seen in able-bodied subjects performing cycle ergometry [Wasserman and Whipp, 1975].

As noted above (Sect. “Extended measures of efficiency”), the standard and extended definitions of work efficiency, Eqs. 5 and 9, respectively, are theoretically equivalent for positive work rates, $P_{out} > 0$, and under assumptions of linearity and noise-free measurement. Thus, it is reasonable to use Eq. 5 when analysing previous studies, where the condition $P_{out} > 0$ pertained, and to compare these values to those obtained in the present study with Eq. 9, when often $P_{out}$ was less than 0W (e.g. Fig. 5). Despite methodological differences, it is clear that, regardless of the experimental protocol and the details of calculation method employed, the efficiency of stimulated cycling exercise is very substantially lower than normal, volitional cycling exercise.

In the present study, our new definition of total work efficiency took proper account of useful internal work in impaired subjects and, for the first time, allowed characterisation of the efficiency of untrained subjects with paraplegia performing stimulated cycle ergometry, even for absolute work rates of less than 0W. It is striking that the efficiency of untrained subjects with paraplegia was found to be within the range for trained paraplegics and close to that of anaesthetised able-bodied individuals. In research on functional electrical stimulation, various general unquantified explanations are given for the poor performance shown by stimulated paralysed muscles. For the three groups of subjects under consideration, we can divide these explanations into two groups: chronic effects of spinal cord injury, and acute effects. There are several chronic effects: atrophy of the muscles; the conversion of the muscle to fast, rapid-fatiguing fibres; and the reduction of capillary density. The acute effects are: stimulation may not include all the synergistic motor units before antagonistic motor units are recruited; synchronous stimulation of all motor units in the muscle group (diminishing blood perfusion); absence of sensory feedback to regulate the motor activity; and constant stimulation frequency. It is striking that the efficiency of the three groups should be so similar because the anaesthetised subjects, being uninjured, can not have poor efficiency due to chronic physiological change. Therefore, poor efficiency must be due either to the artificial muscle activation (i.e. the crude timing and selection of motor units and their synchronous stimulation at constant frequency), or it must be due to the absence of sensory feedback. The latter might be directly related to motor control or to the regulation of blood flow.

On the other hand, the sustainable total mechanical work rate of the untrained subjects studied here was minimal (6.2W on average, at a mean stimulation intensity of 68%) and the corresponding absolute cycling work rate was, on average, less than 0W, reflecting muscle atrophy and deleterious adaptation of the physiological muscle fibretype profile: untrained paraplegic cyclists may not be able to generate sufficient power even to rotate their legs in the absence of an external load.

Despite severe initial limitations in muscle performance, it is surprising that, following a period of stimulated cycle training, paraplegics can perform to the extent that significant cardiopulmonary stress and bone loading can be induced during stimulated cycling exercise, leading to significant physiological and health benefits [Janssen et al.; 1998], and that sufficient power can be developed to propel a mobile tricycle [Hunt et al., 2004] (Fig. 3a). Glaser et al. [1989] reported that, of 20 trained SCI subjects, only one was able to sustain an absolute external work rate of 30W for 5min. On the other hand, in Kjaer et al. [1994], all eight anaesthetised able-bodied subjects performing stimulated cycling exercise were able to
sustain an external work rate of up to ~40W. These figures may provide reasonable expectations for achievable work rate following muscle conditioning.

These results suggest that attempts to increase power output during stimulated cycling in subjects with paraplegia should focus on two areas: training to increase muscle strength, and better methods of activating the muscles to increase efficiency. The greatest potential for increasing efficiency appears to lie in better timing of muscle activation, and improved methods for the activation and control of the muscles involved in the exercise (Janssen et al., 2004; Kebaetse et al., 2005).

**Conclusions**

We proposed modified measures of efficiency appropriate to subjects with severe physical impairment performing cycle ergometry. This allowed characterisation of the efficiency of untrained paraplegic subjects performing cycle ergometry by means of functional electrical stimulation. These new tools may prove useful for optimization of stimulation strategies, or for monitoring changes in efficiency.
4.4 Feasibility of functional electrical stimulated cycling in subjects with spinal cord injury: an energetic assessment

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Published in: J Rehabil Med 2010; 42: 873-875.

Introduction

Individuals with a complete spinal cord injury (SCI) exhibit reduced physical activity due to a paralysis-based loss of motor function [Jacobs and Nash, 2004; Washburn and Figoni, 1999]. As a consequence, low cardiorespiratory fitness levels are common in subjects with SCI and lead to inactivity-related co-morbidities, such as hyperlipidaemia, obesity, diabetes and cardiovascular disease [Myers et al., 2007]. In fact, during the past years cardiovascular disease has been one of the leading causes of mortality in people with chronic SCI [National Spinal Cord Injury Statistical Center, 2009]. Thus, regular physical activity appears to play a key role in reducing health-related risk factors and complications in this population [Mohr et al., 1997a].

Physical activity in people with complete SCI is limited and is restricted mainly to upper body exercise, where only a small muscle mass is involved. In addition, up to 64% of subjects with paraplegia [Sie et al., 1992; Pentland and Twomey, 1994] reported upper extremity (mainly shoulder) pain, which may impact on upper body exercise performance. Functional electrical stimulated (FES) cycling may provide a suitable alternative involving a large muscle group, and concurrently prevent additional exercise-induced loads being imposed on the upper extremities.

The effect of FES-cycling on the cardiovascular system of subjects with SCI has been investigated in several studies [e.g. Jacobs and Nash, 2004; Janssen et al., 1998; Petrofsky and Phillips, 1984; Petrofsky et al., 1984; Ragnarsson et al., 1988]. Although beneficial effects of FES-cycling on cardiopulmonary fitness are undisputable, the precise frequency, intensity and duration of FES-cycle training required to minimize health risks remains unclear, and training regimes have varied widely [Berry et al., 2008]. However, commonly accepted recommendations to reduce health risks are for at least 30min of moderate daily activity [Myers et al., 2007; Warburton et al., 2006b]. Some studies [Durstine et al., 2001; Slentz et al., 2007; Warburton et al., 2006b] recommend a minimal weekly training volume, with energy expenditures of approximately 1000–2200kcal, whereas others suggest that
more benefits may be expected with higher caloric expenditure [Durstine et al., 2001; Helmrich et al., 1991].

The aim of the present study was to determine the FES-cycling volume necessary to reach the generally recommended weekly exercise caloric expenditure of 1000–2200kcal [Durstine et al., 2001; Warburton et al., 2006b] in FES-trained subjects with paraplegia and to estimate how feasible and realistic such a training regime might be for a broader population with SCI.

**Methods**

Subjects:
Eight (7 males, 1 female) healthy FES-trained subjects with traumatic motor and sensory complete paraplegia (AIS A) of at least 3 years duration participated in the study. Detailed information about anthropometrical data and impairment are presented in Table 1. Subjects were all part of a multicentre FES-cycling study described in detail elsewhere [Berry et al., 2008] and gave their written informed consent to participate in the study, which was approved by their respective ethics committee: the ethics committees of the Southern General Hospital and the Faculty of Biomedical and Life Sciences at the University of Glasgow, UK (for subjects tested in Glasgow) and the ethics commission of Kanton Luzern, Switzerland (for subjects tested in Nottwil). Ethics approval was obtained prior to the start of the study.

Table 1: Subjects’ characteristics

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</table>

Mean ± SD 42.5 ± 6.7 176.0 ± 8.1 74.1 ± 7.3 13.4 ± 7.0

SD: standard deviation; m: male; f: female; T: thoracic; AIS: American Spinal Injury Association (ASIA) impairment scale
Equipment and experimental procedure:
At the end of a 12-month high-volume FES-cycling programme described by Berry et al. [2008] a 60-min home-training session performed by each subject was monitored. For this purpose, subjects were sitting on an individually adapted recumbent tricycle (Inspired Cycle Engineering Ltd, Cornwall, UK) mounted on a training roller (Flow Ergotrainer Tacx, Wassenaar, The Netherlands) and performed their habitual training at the highest workload they were able to sustain for 60min. In order to provide muscle contraction for cycling, quadriceps, gluteal and hamstring muscles were stimulated bilaterally via surface electrodes by means of a Stanmore Stimulator [Phillips et al., 1993].

During the whole training session oxygen consumption (VO\(_2\)) and carbon dioxide production (VCO\(_2\)) were recorded breath by breath with a portable ergospirometric system (Metamax, Cortex Biophysic GmbH, Leipzig, Germany), which was calibrated according to the manufacturers' instructions prior to each test.

Carbohydrate and fat oxidation rates were calculated based on VO\(_2\) and VCO\(_2\) data according to the formulae of Péronnet and Massicotte [1991]. Energy expenditure from fat and carbohydrates were converted to kilocalories per hour (kcal·h\(^{-1}\)) by multiplying the oxidation rate of fat by 9.75 [Péronnet and Massicotte, 1991] and the oxidation rate of carbohydrate by 4.15 [Ferrannini, 1988].

Statistics
Data are presented as means ± standard deviations.

Results
Subjects had a mean energy expenditure for 60min FES-cycling exercise of 288±104kcal·h\(^{-1}\). Seventy-one percent of the total amount of energy was delivered by carbohydrate oxidation and 29% by fat oxidation. This corresponded to oxidation rates of 49.5±35.2g·h\(^{-1}\) for carbohydrates and 8.5±8.4g·h\(^{-1}\) for fat. Thus, approximately 4 to 8h of FES-cycling are necessary to reach an energy expenditure of 1000–2200kcal.

Discussion
The results of the present study show that for FES-trained subjects with paraplegia between 4 and 8 hours of intense FES-cycling are necessary to reach the postulated minimal weekly energy expenditure of 1000–2200kcal in order to reduce health risks [Durstine et al., 2001; Slentz et al., 2007]. Interestingly, the calculated 4h per week seem to correspond closely to the 30min of moderate daily activity proposed in former studies for older able-bodied persons [Warburton et al., 2006b] as well as for subjects with SCI [Myers et al., 2007]. In other words, FES-cycling appears to be a feasible and promising training alternative to upper body exercise for subjects with a SCI. FES-cycling may help to reduce training induced overload of upper extremities and concomitant shoulder pain [Pentland and Twomey, 1994; Sie et al., 1992]. In addition, FES-cycling exercise is also possible in subjects with tetraplegia
Moreover, FES-cycling of the lower limbs involves large muscle groups and creates a higher cardiorespiratory and musculoskeletal stress compared with isolated arm exercise. Although the absolute cycling power output of FES-cycling was found to be very low in untrained [Perret et al., 2009] and trained FES cyclists [Berry et al., 2008], the metabolic cost is approximately 3.5 times higher than for cycling exercise in able-bodied persons [Hunt et al., 2007]. In terms of energy expenditure this is advantageous, although a higher level of work efficiency for mobility and recreation purposes would be desirable.

Despite the numerous potential health benefits of FES-cycling, including improved cardiopulmonary fitness [Berry et al., 2008], enhanced insulin sensitivity [Mohr et al., 2001] and positive effects on bone loss [Frotzler et al., 2008b], one should bear in mind that FES-cycling is quite time-consuming and requires some fundamental skills. Before transferring to the FES-cycle, electrodes have to be placed and connected to a power source, and after the training session have to be disconnected, which, depending on the person’s motor skills, may make the presence of a caregiver necessary. Such circumstances may negatively influence training compliance. However, in a recently published FES-cycling study [Berry et al., 2008] mean compliance was reported to range between 75% and 91% (corresponding to a mean of 3.7 training sessions per week of 58min duration each) over a one-year training period. It is of interest that these data accurately correspond to the 4h calculated in the present study and seem to cover the basic requirements to reduce health-related risk factors and complications. As more benefits may be expected with higher caloric expenditure [Durstine et al., 2001; Helmrich et al., 1991], it would be desirable for training duration and intensity to be increased further. Given that preparation (attaching and removing electrodes, etc.) is quite time-consuming it may be advantageous and less time-consuming to exercise less frequently but for longer durations (e.g. 3 times 90min instead of 6 times 45min per week). In addition, it was postulated by Kraus et al. [2002] that for positive effects on, for example, plasma lipid and lipoprotein concentrations, exercise volume rather than exercise intensity seems to be more important. Beside the practical considerations, this finding additionally supports the recommendation of fewer but more prolonged training sessions in order to achieve a higher training volume.

A promising alternative to further enhance energy expenditure and cardiorespiratory fitness in subjects with SCI might be a combination of voluntary upper body and FES-induced leg exercise, such as FES-rowing. A review [Hettinga and Andrews, 2008] reported average peak VO\(_2\) values for FES-rowing of 1.98l·min\(^{-1}\) compared with 1.05l·min\(^{-1}\) during FES-cycling, which underlines the higher physical and caloric demands of FES-rowing compared with FES-cycling. However, excessive FES-rowing exercise may lead to overuse and concomitant pain of the upper extremities (mainly the shoulder), which was found to be a major problem in subjects with SCI [Pentland et al., 1994; Sie et al., 1992].

For the future it might also be worthwhile considering a broader application of FES-cycling in patients with incomplete SCI or in subjects with tetraplegia [Kakebeeke et al., 2008] as an alternative training mode in order to enhance physical fitness and energy expenditure in this population, which is especially at risk for overweight and obesity [Weaver et al., 2007]. Moreover, beyond the scope of this paper, the application of FES also seems to be gaining a more important role in rehabilitation and regeneration after SCI [Hamid and Hayek, 2008].
Conclusions

In conclusion, FES-cycling appears to be a feasible and promising training alternative to upper body exercise for subjects with SCI. Four to 8h of FES-cycling are necessary to reach the recommended weekly exercise caloric expenditure of 1000–2200kcal, which seems to be essential to induce persistent health benefits.
Introduction

Complete spinal cord injury (SCI) leads to an extreme form of immobilisation in the paralysed limbs. As a consequence, a marked and rapid atrophy of the vascular system [Olive et al., 2003] and muscle tissue [Baldi et al., 1998; Castro et al., 1999; Crameri et al., 2000], and a loss of bone tissue in the paralysed regions [Biering-Sorensen et al., 1990; Dauty et al., 2000; de Bruin et al., 2005; Frey-Rindova et al., 2000; Jones et al., 1998] manifests itself. Within the first few years after SCI, bone mineral content (BMC) decreases by around 45% in the femur [Esar et al., 2004; Kiratli et al., 2000] and by 56% in the tibia [Esar et al., 2004]. More specifically, the most affected bone sites are the cancellous compartments of the long bones [Biering-Sorensen et al., 1990; Dauty et al., 2000; Eser et al., 2004; Kiratli et al., 2000], leading to a mean deficit of 73% in trabecular bone mineral density (BMD) in the distal tibia compared to able-bodied values [Esar et al., 2004]. Secondary to this bone loss, fractures caused by minimal trauma often occur in the paralysed lower extremities. The skeletal sites most prone to fractures are the distal femur and the proximal and distal tibia [Eser et al., 2005; Garland and Adkins, 2001; Lazo et al., 2001; Ragnarsson and Sell, 1981], with an estimated fracture incidence of twice the incidence in able-bodied people [Vestergaard et al., 1998; Zehnder et al., 2004].

It is still unknown whether unloading of the bones in the paralysed extremities is the only factor causing the rapid sublesional bone loss or if other factors such as neuronal, humoral and vascular adaptations after a spinal lesion are also involved [Stoner et al., 2006; Thijsse et al., 2006; Uebelhart et al., 1994]. Several studies have shown that there is no bone loss in the upper extremities of persons with paraplegia [Biering-Sorensen et al., 1990; Chantraine, 1978; de Bruin et al., 2005; Eser et al., 2004; Frey-Rindova et al., 2000; Nidecker et al., 1991], disproving the hypothesis that bone loss following SCI is systemic. Rather, local unloading of the bones in the paralysed extremities together with the detrimental effects of
SCI on muscle tissue and the vascular and neuronal systems are the major factors responsible for the rapid and vast bone loss after SCI, as postulated by Frost's mechanostat theory many years earlier [Frost, 1987; Frost, 1990a and 1990b; Frost, 1997].

Previous studies investigated the effect of reloading the leg bones via muscle contractions induced by functional electrical stimulation (FES) using exercise modalities such as cycling [BeDell et al., 1996; Bloomfield et al., 1996; Chen et al., 2005; Eser et al., 2003; Hangartner et al., 1994; Leeds et al., 1990; Mohr et al., 1997b; Pacy et al., 1988]. The findings of the impact of FES-cycle training on the bones of the paralysed limbs were equivocal: some found no bone adaptations [BeDell et al., 1996; Eser et al., 2003; Leeds et al., 1990; Pacy et al., 1988] while others documented a reduction in the rate of bone loss [Hangartner et al., 1994] or even a recovery of BMD [Bloomfield et al., 1996; Chen et al., 2005; Mohr et al., 1997b]. In most of these studies, training volume was set at 3 sessions per week during six [Eser et al., 2003; Hangartner et al., 1994; Leeds et al., 1990], nine [Bloomfield et al., 1996] or twelve months [Mohr et al., 1997b]. In contrast, Chen et al. [2005] investigated the effect of a more intensive FES-cycle training regime of 30min, five times per week, at a sub-maximal resistance load, over a period of six months. They found a recovery in areal BMD of 11.1% in the distal femur and of 12.9% in the proximal tibia in people with chronic complete SCI. Similarly, Mohr et al. [1997b] conducted a less intensive FES-cycle training programme (three weekly sessions) but for a longer duration (12 months), and found a comparable increase in areal BMD of 10% in the proximal tibia.

In summary, these results suggest a potentially positive and site-specific effect of FES-induced exercise on bones after SCI. Because the cited studies on FES-cycling indicate that there may be a dose-dependent effect, the present study investigated the effect of high-volume FES-induced cycle training on the leg bones in subjects with long-lasting and complete SCI. In contrast to other groups who investigated the osteogenic effect of an FES-cycle training programme by means of dual energy X-ray absorptiometry (DXA), we performed peripheral quantitative computed tomography (pQCT) scans at several sites in the femur and tibia in order to achieve the most detailed assessment to date with regard to volumetric BMD and bone geometry. In addition, to determine the time course of the osteogenic effect, bone and soft tissue parameters in the lower limbs were assessed three times: before, during and after the FES-training programme.

Methods

This study was conducted as a multi-centre trial at: 1) the Queen Elizabeth National Spinal Injuries Unit and the University of Glasgow, Glasgow, United Kingdom; 2) Division of Applied Biomedical Research, King's College London, London, United Kingdom; and 3) Swiss Paraplegic Research, Nottwil, Switzerland. The study was approved by the Ethics Committees of King's College Hospital and King's College London, the Southern General Hospital in Glasgow and the Canton of Lucerne.

Subjects

A total of 12 SCI subjects were recruited at the three centres: three at the Queen Elizabeth National Spinal Injuries Unit, five at the Division of Applied Biomedical Research, Kings
College London and four at Swiss Paraplegic Research. All subjects fulfilled the following inclusion criteria: motor-complete post-traumatic medullary lesion between T3 and T12 (grade A on the American Spinal Injuries Association (ASIA) impairment scale), at least three years post injury, and greater than 18 years of age. Exclusion criteria were severe spasticity, current or past unhealed bone fractures, diseases or medication known to affect bone metabolism, contractures in the hip and the lower extremities restricting full range of motion as well as participation in any FES programmes during the 12 months prior to the start of the study. Subjects provided written, informed consent prior to participation in the study.

Intervention
Muscle conditioning
In order to prepare the paralysed muscles for the FES-cycle training, the subjects first performed muscle conditioning training. For the muscle conditioning, we used an 8-channel-stimulator (Stanmore Stimulator) with the following stimulation parameters: pulse frequency of 50Hz, pulse width of 300–400µs, an amplitude between 80 and 150mA and a 1:1 duty cycle set at 6s on/off. The training consisted of 30 to 60min of isometric bilateral FES three to five times weekly, with surface electrodes placed proximally and distally to the motor points of the gluteus, quadriceps and hamstrings muscles. In addition, the calf muscles (triceps surae) were stimulated in the 5 subjects from the London group and in one from the Nottwil group. Subjects performed this training at home either in a sitting, reclined or standing position (using a standing frame). The muscle conditioning was performed until the subject was able to pedal on an FES-cycle system (described below) without resistance for at least 10min.

FES-cycle system
The cycle training was performed at home on a recumbent FES-tricycle, adapted with lower leg orthoses to provide optimal support during pedalling (Fig. 1). The cycle system was mounted on a trainer, so that the training load could be controlled. Similarly to the muscle conditioning phase, surface electrodes were applied bilaterally on the gluteal, quadriceps, and hamstrings muscles. Again, in five subjects from the London and in one from the Nottwil group the calf muscles (triceps surae) was sub-maximally stimulated. The same 8-channel-stimulator was used for the cycling as described above. Stimulation frequency was fixed at 50Hz and amplitude was preset according to individual needs (depending on muscle size and thickness of overlaying subcutaneous fat tissue), while the pulse width was adjusted by the individual via a throttle up to 500µs. The stimulator delivered a pre-programmed stimulation sequence that produced a smooth cycling motion [Hunt et al., 2004]. After appropriate instruction at their local centre, the subjects had an FES-cycle system installed at home where they performed their own unsupervised cycle training.
FES-cycle training
During the first three months of the high-volume FES-cycle training, the training protocol consisted of three to four sessions per week, each session lasting 10 to 60min. After this initial training phase, the subjects had to be able to cycle for 60min, five times a week, in order to progress to the next training stage. During the following ninemonths of the FES-training, subjects were expected to complete five sessions per week of 60min each. At the end of the study, the subjects had performed a total of 12 months of FES-cycling (Fig. 2). In cases where subjects had to interrupt the FES-cycle training due to holidays or illness, the training schedule only resumed once they had regained their pre-interruption cycling load. During the whole training period, subjects were encouraged to train at their individual maximal resistance settings and to cycle with a cadence of 45 to 50 revolutions per minute. Furthermore, the subjects had to fill in a training diary for each training session stating the training resistance and duration.
Measurements
Bone measurements were performed with a pQCT scanner (XCT3000, Stratec Medical, Pforzheim, Germany). Peripheral QCT has been shown to be highly precise in subjects with established paralysis and severe bone atrophy, achieving coefficients of variation for bone parameters at the femur and tibia of mostly better than 1% [Eser et al., 2004]. In the present study, measurements were performed as detailed in a previously published study [Eser et al., 2004]. The main advantages of pQCT over the gold-standard DXA are: it can distinguish trabecular and cortical bone compartments; one can derive the bone geometric parameters and volumetric BMD rather than areal BMD [Braun et al., 1998; Müller et al., 1989]; the measured bone parameters are independent of bone size; and finally, it gives the composition of the surrounding soft tissue [Bolotin, 1998; Bolotin, 2001; Pors Nielsen, 1998]. The latter is crucial in cases where the effect of muscle training on bone is to be assessed, but muscle mass increases are expected at the same time. The image processing and calculation of numerical values were performed using the manufacturer's software package (version 5.50 E).

Bone measurements were taken three times: at baseline prior to muscle conditioning (t₁); after six months (t₂); and after twelve months of the FES-cycle training (t₃) (Fig. 2). Bone measurements were performed bilaterally in the femur and tibia. Trabecular bone parameters were assessed at the distal epiphyses of the femur and tibia and at the proximal tibia at 4% of total bone length measured from the respective joint gap. Cortical bone parameters were obtained at 36% in the tibia and at 25% in the femur, as measured from the distal joint gaps. In addition, fat and muscle tissue parameters were assessed at 66% of total bone length in the tibia and at 25% in the femur, as measured from the distal joint gap.

Bone parameters
In the epiphyses, total bone cross-sectional area (CSAtot), BMC, total BMD (BMDtot) and trabecular BMD (BMDtrab) were calculated. The latter was determined as mean density of the central 45% of CSAtot. In the diaphyses, CSAtot, BMC, cortical CSA (CSAcort), cortical BMD (BMDcort) and the polar bone strength strain index (SSIpol) were measured. In addition, cortical thickness (THIcort) was estimated based on the assumption that the bone shaft is cylindrical, calculating the difference of the radius of CSAtot minus the radius of themedullary cavity (CSAmedulla=CSAtot−CSAcort). To avoid an underestimate of BMDcort due to the partial volume effect [Hangartner and Gilsanz, 1996], BMDcort was determined only for subjects with a cortical thickness (THIcort) of >1.6mm [Eser et al., 2004]. In addition, muscle CSA (CSAmuscle) and fat CSA (CSAfat) were also identified.

Peak power output
The FES-cycle systems that subjects used for training at home did not allow training power outputs to be recorded. However, subjects were encouraged to train at their individual maximal resistance settings. An indicator of progress in training power outputs is the peak power output that was measured during exercise tests carried out at three time points over the exercise intervention period (using a testbed described by Hunt et al. [2004] and an exercise testing protocol detailed in references [Ferrario et al., 2007] and [Kakebeeke et al., 2008]). The peak powers used here (based on data from Berry et al. [2008]) are from the following test time points: (i) after completion of muscle conditioning and immediately before
the start of the FES-cycle training, (ii) after six months, and (iii) after twelve months of FES cycle training

Statistical analysis

Peripheral QCT parameters
Mean values of bone, muscle and fat parameters at t₁, t₂ and t₃ were calculated by averaging the data of a subject's right and left legs. In the case that only one leg was measured because of the presence of a metal pin or plate in the other leg, values of the measured leg were used. In order to analyse the impact of FES-cycling on bone and soft tissue parameters, nonparametric Friedman's analysis of variance was used to test for significant differences between the measured pQCT parameters at t₁, t₂ and t₃. Due to the explorative nature of the present study we decided not to perform a Bonferroni correction to the level of significance. Where a significant difference was detected, Wilcoxon comparisons were performed to locate the statistically significant differences between t₁, t₂ and t₃. This nonparametric test procedure was chosen due to the small sample size. In addition, to test for the relationship between muscle CSA and bone parameters, Spearman correlation coefficients were calculated for absolute changes in bone and muscle parameters.

Peak power output
To test the relationship between changes in peak power output and changes in soft tissue, Spearman correlation coefficients were calculated over the period from t₁ to t₃. All statistical analyses were performed using SPSS software (Version 13.0). Statistical significance was set at \( p \leq 0.05 \).

Results

Study subjects
One subject had to terminate the study due to a foot fracture which occurred seven months into the study, but which was unrelated to the FES-cycle training. Subject characteristics of the remaining eleven subjects are shown in Table 1.

Muscle conditioning
To prepare muscles in the paralysed legs for FES-cycling, subjects performed on average 14±7 weeks (range 6 to 32 weeks) of muscle conditioning.

FES-training compliance
Overall, subjects completed an average of 79.3% of the scheduled FES-cycle training sessions, corresponding to 3.7±0.6 sessions per week, each session lasting for 58±5min. During the last nine months of the FES-training with a training schedule of 5 training sessions per week, compliance was 76.6%, corresponding to 3.8±0.7 sessions per week (range 2.8 to 4.8).
Table 1: Subject characteristics

<table>
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<th>Height [cm]</th>
<th>Weight [kg]</th>
<th>Time post injury [y]</th>
<th>Lesion level</th>
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<td>186</td>
<td>67</td>
<td>25.5</td>
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</tbody>
</table>

Mean ± SD  41.9 ± 7.5  175.5 ± 7.7  73.2 ± 14.2  11.0 ± 7.1

SD: standard deviation; T: thoracic spinal lesion

Peripheral QCT-measurements
The three scheduled bone measurements were performed at baseline (t₁), at 11.7±1.9 months for t₂ (range 8.5 to 15.1 months) and at 19.0±2.1 months for t₃ (range 16.0 to 23.1 months). Reasons for the wide range of t₂ were different periods of muscle conditioning (between 6 and 32 weeks), as well as training interruptions.

In nine of the eleven subjects, all femoral scans were performed bilaterally at t₁, t₂ and t₃. The right femoral scans of Subject 8 had to be excluded because a peripheral lesion of the right rectus femoris muscle was diagnosed by a neurologist six months after the start of the training. In addition, the right femoral shaft scan of Subject 9 could not be measured due to the presence of a metal pin. In eight subjects, all three tibia scans were completed bilaterally at all time points. All tibial scans of the left leg of Subject 3 had to be excluded due to an incorrectly placed reference line at t₁. The two diaphyseal scans (38% and 66%) in the right tibia of Subject 9 and all scans in the right tibia of Subject 4 were omitted due to the presence of metal pins or plates.

Bone parameters
Bone parameters of the femur changed significantly over the period of the FES-cycle training. In the distal femoral epiphysis, BMDtrab and BMDtot increased significantly between t₁ and t₃ by 14.4±21.1% and 7.0±10.8%, respectively (Wilcoxon post-hoc test (Wph): both p=0.05). Between t₂ and t₃, BMDtrab (Fig. 3) increased significantly by 3.1±3.2% (Wph: p=0.016), BMDtot by 1.3±1.7% (Wph: p=0.041), and CSAtot by 1.2±1.5% (Wph: p=0.001). In the femoral diaphysis, BMC was significantly reduced by 1.6±2.3% between t₁ and t₂ (Wph: p=0.019) and 1.8±3.0% between t₁ and t₃ (Wph: p=0.037), THIcort by 1.4±1.3% between t₁ and t₂ (Wph: p=0.010) and 1.5±2.1% between t₁ and t₃ (Wph: p=0.041) and BMDcort was
reduced by 0.4±0.4% between $t_1$ and $t_2$ (Wph: $p=0.016$) and 0.4±0.4% between $t_1$ and $t_3$ (Wph: $p=0.003$) (Table 2). None of the measured bone parameters in the distal and proximal tibial epiphyses (e.g. BMC, Fig. 4) or diaphyses were found to change significantly over the FES-cycle training period.

Figure 3: Time course of absolute trabecular bone mineral density (BMDtrab) in the distal femoral epiphysis at baseline ($t_1$), after six ($t_2$) and after twelve months of FES-cycling ($t_3$) ($n=11$).

Muscle and fat tissue parameters
Muscle CSA in the thigh was found to increase by 34.4±16.4% between $t_1$ and $t_2$ (Wph: $p=0.003$) and by 35.5±18.3% between $t_1$ and $t_3$ (Wph: $p=0.003$) (Table 2), while CSAfat decreased by 7.7±10.8% between $t_1$ and $t_3$, but missed the level of significance (Wph: $p=0.148$). In the lower leg, CSAfat showed a significant decrease of 16.7±12.3% between $t_1$ and $t_3$ (Wph: $p=0.013$) and of 11.2±11.3% between $t_2$ and $t_3$ (Wph: $p=0.026$) (Table 2), while no change in CSAmuscle in the shank was found.
Correlations between muscle and bone changes

No measured changes in the femoral bone parameters were significantly related to the changes in CSA

Muscle. With regard to the lower leg, again no significant correlations were found between changes in tibial CSA

Muscle and changes in tibial bone parameters. Correlations between peak power output and soft tissue changes

Mean peak power output was 8.4±3.2W at the end of the muscle conditioning phase, 17.8±8.5W at three months and

18.4±8.7W at twelve months of FES-cycle training (personal communication with H. Berry, Centre for Rehabilitation Engineering, Department of Mechanical Engineering, University of Glasgow). In the thigh, none of the soft tissue changes were significantly related to the changes in peak power output. In the lower leg, a significant negative correlation between the increase in peak power output and the change in the amount of fat (between t<sub>2</sub> and t<sub>3</sub>) was found (R=−0.636, p=0.035), while changes in CSA

Muscle were not significantly related to changes in peak power output.
Table 2: Bone, muscle and fat parameters (mean±standard deviation) calculated from bilateral measurements of all subjects at baseline (t\(_1\)) and after six (t\(_2\)) and twelve months (t\(_3\)) of FES-cycle training (n=11)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean (SD) at t(_1)</th>
<th>Mean (SD) at t(_2)</th>
<th>Mean (SD) at t(_3)</th>
<th>P-value</th>
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<td>6.46 (1.19)</td>
<td>6.64 (1.30)</td>
<td>n.s.</td>
</tr>
<tr>
<td>CSAtot [mm(^3)]</td>
<td>3924.94 (537.72)</td>
<td>3908.87 (560.15)</td>
<td>3958.31 (571.72)</td>
<td>*</td>
</tr>
<tr>
<td>BMDtot [mg/cm(^3)]</td>
<td>157.90 (24.17)</td>
<td>166.22 (25.14)</td>
<td>168.41 (26.51)</td>
<td>*</td>
</tr>
<tr>
<td>BMDtrab [mg/cm(^3)]</td>
<td>122.10 (25.21)</td>
<td>133.49 (23.33)</td>
<td>137.41 (25.26)</td>
<td>*</td>
</tr>
<tr>
<td>25%</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMC [g/cm]</td>
<td>3.38 (0.62)(^\dagger)</td>
<td>3.33 (0.59)(^\dagger)</td>
<td>3.31 (0.58)(^\dagger)</td>
<td>n.s.</td>
</tr>
<tr>
<td>CSAcort [mm(^3)]</td>
<td>227.42 (42.30)</td>
<td>224.40 (41.72)</td>
<td>223.89 (40.95)</td>
<td>n.s.</td>
</tr>
<tr>
<td>CSAtot [mm(^3)]</td>
<td>910.06 (137.79)</td>
<td>900.32 (136.36)</td>
<td>899.49 (138.72)</td>
<td></td>
</tr>
<tr>
<td>THlcort [mm(^3)]</td>
<td>2.31 (0.44)(^\dagger)</td>
<td>2.28 (0.43)(^\dagger)</td>
<td>2.27 (0.43)(^\dagger)</td>
<td>*</td>
</tr>
<tr>
<td>BMDcort [mg/cm(^3)]</td>
<td>1117.18 (33.53)(^\dagger)</td>
<td>1097.89 (31.45)(^\dagger)</td>
<td>1097.56 (30.73)(^\dagger)</td>
<td>*</td>
</tr>
<tr>
<td>SSpol [mm(^3)]</td>
<td>2858.49 (681.14)</td>
<td>2860.24 (669.26)</td>
<td>2853.54 (653.91)</td>
<td>n.s.</td>
</tr>
<tr>
<td>CSAmuscle [mm(^3)]</td>
<td>4426.20 (1167.76)(^\dagger)</td>
<td>5873.95 (1441.36)(^\dagger)</td>
<td>5940.02 (1584.42)(^\dagger)</td>
<td>*</td>
</tr>
<tr>
<td>CSAfat [mm(^3)]</td>
<td>6179.62 (2415.95)</td>
<td>5996.92 (2572.90)</td>
<td>5783.50 (2580.16)</td>
<td>n.s.</td>
</tr>
<tr>
<td><strong>Tibia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4%</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMC [g/cm]</td>
<td>2.14 (0.84)</td>
<td>2.13 (0.82)</td>
<td>2.14 (0.84)</td>
<td>n.s.</td>
</tr>
<tr>
<td>CSAtot [mm(^3)]</td>
<td>1281.65 (177.91)</td>
<td>1283.74 (185.51)</td>
<td>1279.68 (180.07)</td>
<td>n.s.</td>
</tr>
<tr>
<td>BMDtot [mg/cm(^3)]</td>
<td>166.37 (56.98)</td>
<td>165.24 (55.82)</td>
<td>166.91 (56.85)</td>
<td>n.s.</td>
</tr>
<tr>
<td>BMDtrab [mg/cm(^3)]</td>
<td>100.52 (57.28)</td>
<td>99.71 (56.64)</td>
<td>100.73 (57.28)</td>
<td>n.s.</td>
</tr>
<tr>
<td>38%</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMC [g/cm]</td>
<td>3.51 (0.73)</td>
<td>3.52 (0.69)</td>
<td>3.51 (0.69)</td>
<td>n.s.</td>
</tr>
<tr>
<td>CSAcort [mm(^3)]</td>
<td>278.55 (65.44)</td>
<td>276.75 (63.42)</td>
<td>275.91 (63.57)</td>
<td>n.s.</td>
</tr>
<tr>
<td>CSAtot [mm(^3)]</td>
<td>454.01 (73.88)</td>
<td>455.53 (74.44)</td>
<td>454.45 (73.71)</td>
<td>n.s.</td>
</tr>
<tr>
<td>THlcort [mm(^3)]</td>
<td>4.66 (1.28)</td>
<td>4.60 (1.23)</td>
<td>4.59 (1.24)</td>
<td>n.s.</td>
</tr>
<tr>
<td>BMDcort [mg/cm(^3)]</td>
<td>1111.92 (51.97)</td>
<td>1120.09 (51.00)</td>
<td>1121.28 (50.17)</td>
<td>n.s.</td>
</tr>
<tr>
<td>SSpol [mm(^3)]</td>
<td>1769.60 (400.51)</td>
<td>1779.62 (397.29)</td>
<td>1752.88 (356.07)</td>
<td>n.s.</td>
</tr>
<tr>
<td>CSAmuscle [mm(^3)]</td>
<td>4866.84 (1245.53)</td>
<td>5154.36 (1204.23)</td>
<td>5219.65 (1540.11)</td>
<td>n.s.</td>
</tr>
<tr>
<td>CSAfat [mm(^3)]</td>
<td>2722.40 (1364.27)(^\dagger)</td>
<td>2581.58 (1312.28)(^\dagger)</td>
<td>2325.70 (1255.19)(^\dagger)</td>
<td>*</td>
</tr>
<tr>
<td><strong>Proximal Tibia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4%</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMC [g/cm]</td>
<td>3.59 (0.91)</td>
<td>3.55 (0.86)</td>
<td>3.52 (0.88)</td>
<td>n.s.</td>
</tr>
<tr>
<td>CSAtot [mm(^3)]</td>
<td>2905.32 (546.00)</td>
<td>2906.75 (592.27)</td>
<td>2900.27 (571.32)</td>
<td>n.s.</td>
</tr>
<tr>
<td>BMDtot [mg/cm(^3)]</td>
<td>124.44 (27.98)</td>
<td>123.62 (27.46)</td>
<td>122.50 (26.82)</td>
<td>n.s.</td>
</tr>
<tr>
<td>BMDtrab [mg/cm(^3)]</td>
<td>71.62 (25.34)</td>
<td>69.65 (23.00)</td>
<td>69.17 (23.02)</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

\(^\dagger\) n = 10; \(^\star\) P ≤ 0.05; \(^\dagger\), \(^\dagger\), \(^\dagger\): significant difference between \(^\dagger\) t\(_1\) and t\(_2\), \(^\dagger\) t\(_2\) and t\(_3\), \(^\dagger\) t\(_3\) and t\(_3\).
Discussion

This study is the first to investigate the impact of a high-volume FES-cycle training programme on bone parameters in the lower extremities of subjects with chronic complete SCI, by means of pQCT. After 19.0 ± 2.1 months FES exercise (i.e. initial muscle conditioning followed by high-volume FES-cycle training) the bone parameters of the femur had changed significantly. In the distal femur BMDtrab increased by approximately 14% and BMDtot by 7%. In contrast, decreases of less than 2% were found in the femoral shaft for BMDcort, THIcort and BMC. None of the measured bone parameters in the tibia (proximal and distal epiphyses, and shaft) were found to be affected by the FES-cycle training (Table 2). The CSAmuscle in the thigh increased significantly by 35.5% and CSAfat of the calf decreased significantly by 16.7% (Table 2).

With regard to the training volume during the last nine months, our subjects performed on average 3.8 of the 5 scheduled one-hour FES-cycle sessions per week, corresponding to a compliance of 76.6%. Since most of the subjects were working full- or part-time, we suggest that five one-hour training sessions per week may not be feasible for SCI individuals with a busy lifestyle. The compliance in the present study is similar to the findings of Bloomfield et al. [1996] who recorded a compliance of 74%. In addition, both Mohr et al. [1997b] and BeDell et al. [1996] found that their subjects only completed 2.3 and 2.0, respectively, out of 3 scheduled FES-cycle training sessions per week. With training compliance much lower than 100%, it is necessary to prescribe a higher target training volume in order to reach the desired effective number of training sessions.

We found increases in bone parameters of the distal femur similar to those of two recent studies investigating the effects of an FES-cycling training volume of five 30min sessions per week for six months [Chen et al., 2005] and of three 30min sessions per week for 12 months [Mohr et al., 1997b], both documenting a recovery of 11% in BMD in the distal femur [Chen et al., 2005] and of 10% in the proximal tibia [Mohr et al., 1997b]. Based on existing study results it seems there is a dose-dependent response, with three 30min sessions per week for at least six months being the smallest training volume that achieves increases in bone mass. However, further studies are needed to investigate the optimal FES-cycle training regime (i.e. duration of the total training period, number of weekly training sessions, duration of sessions) to achieve maximal osteogenic effect in the paralysed legs.

We suggest that strains produced by FES-cycling were sufficiently large to induce bone formation at the distal femur. According to Frost's mechanostat theory [Frost, 1997; Frost 1990a and 1990b], which postulates that bones adapt their strength to the applied mechanical forces in order to keep bone deformation within a narrow window, it seems that the forces produced by muscle contractions during FES exceeded this strain range and provoked bone formation at the distal femur. However, we speculate that factors other than mechanical loading, such as the modulation of intramedullary pressure [Zhang et al., 2007], the improvement of peak blood flow [Thijssen et al., 2006] and the release of neuropeptide [Offley et al., 2005] may also be important in enhancing bone integrity. At the distal femur, the increase in trabecular bone found in the present study may have important clinical implications, given that the distal femoral and proximal tibial epiphyses are known to be prone to fractures due to minor trauma in people with SCI [Eser et al., 2005; Garland and Atkins, 2001]. In our previously published study [Eser et al., 2005] we found the BMDtrab in the distal femur and distal tibia to be the most sensitive bone parameter to distinguish...
between SCI persons with and without fractures. In addition, fracture thresholds at a BMDtrab of 114mg/cm$^3$ in the distal femur and of 72mg/cm$^3$ in the distal tibia were defined [Eser et al., 2005]. Above these fracture thresholds, no fractures had occurred in our study population. Five of the subjects of the present study had baseline BMDtrab values in the distal femur below the mentioned fracture threshold, and in three of them these values were lifted above the fracture threshold after initial muscle conditioning and 12 months of FES-cycle training. We therefore propose that, all other factors being equal, high-volume FES-cycle training may reduce the risk of sustaining low trauma fractures at this site in people with SCI. However, it has to be considered that a fast increase in CSAmuscle following FES-cycling is associated with an increase in muscle force which may initially be beyond the capacity of bone strength. Although documentation of bone fractures during FES-interventions is scarce [Fournier et al., 1984; Hartkopp et al., 1998] (for example, during measurements of maximal isometric forces elicited by FES [Hartkopp et al., 1998]), an adequate safety protocol for FES-interventions as suggested by Hartkopp et al. [1998] should be taken into account in order to minimise the risk of fractures during FES-intervention.

During the first nine months of the study, i.e. between $t_1$ and $t_2$, six subjects showed an increase in distal femur BMDtrab of between 3.0 and 44.8mg/cm$^3$ and five had a decrease of between 0.2 and 12.2mg/cm$^3$. In contrast, between $t_2$ and $t_3$, only two subjects showed decreases of a maximum of 2.1mg/cm$^3$ while nine had increases in the range of 1.3 and 7.9mg/cm$^3$ (Fig. 3). The finding that bone parameters of the distal femur in six subjects had a lesser increase between $t_2$ and $t_3$ than between $t_1$ and $t_2$ indicates that bone cells in these subjects may have habituated to the mechanical stimulus by $t_2$ [Schriefer et al., 2005] or their new bone strength already sufficiently reduced the bone strains induced by FES-cycling. On the other hand, three other subjects showed a delay in their osteogenic effect, with bone parameters increasing only between $t_2$ and $t_3$, suggesting large inter-individual differences in the time course of bone adaptation to reloading. The initial bone loss in five of our subjects cannot be ascribed to immobilisation-induced bone loss, as this has been found to be completed after 4 years [Eser et al., 2004] in the distal femur, a condition fulfilled in all the subjects of the present study. In fact, the five subjects who showed an initial bone loss in the distal femur had a mean lesion duration of 7.6±3.1 years. The initial decrease in BMDtrab could be due to increased remodelling activity with bone resorption exceeding bone formation at first before bone formation catches up and eventually exceeds resorption, leading to a net bone gain. Thus, our results suggest that it is crucial to conduct an FES-intervention for at least nine months in order to achieve positive bone turnover and significant increases in relevant bone parameters. Additional statistical analyses revealed that neither lesion duration, age, number of FES-cycle sessions performed per week nor duration of muscle conditioning were significantly related to the bone adaptation found in the present study. The reason for the large inter-subject difference in bone adaptation to FES-cycling remains unclear and needs to be investigated in further studies.

The significant decrease of BMDcort in the femoral shaft may also reflect an increase in bone remodelling. At this cortical bone site, the 12-month FES-cycling period may not have been sufficient for bone formation to exceed bone resorption. Increased remodelling increases the porosity of cortical bone, which normally recovers when bone geometry has adapted to the new demand (increased or decreased mechanical loading). Increased porosity would explain the decrease in BMDcort and BMC found in this study. The decrease in THIcort of the femoral shaft may not be a real decrease, but rather a result of image processing based on
thresholds. At a set threshold for cortical bone (711mg/cm³), a lowered BMD_{cort} will reduce both total and CSA_{cort} even if the actual areas have not changed (due to the partial volume effect). As THI_{cort} was calculated from CSA_{tot} and CSA_{cort} data, THI_{cort} would also be reduced erroneously. However, we can not exclude the possibility that spurious effects were found due to multiple Friedman testing.

Similarly to the increased remodelling that is found in the first few years after SCI when bone adapts to greatly reduced strains – due to immobilisation by a transient period of decreased BMD_{cort} that recovers to normal values after 2–3 years [Eser et al., 2004] – the reduction in BMD_{cort} found in the present study may also be transient. A longer FES-cycling programme may be necessary to observe a normalization of BMD_{cort} in the femoral shaft at a concomitantly increased CSA_{cort}.

The present study found a mean increase in CSA_{muscle} at the thigh of 35.5%. This increase was higher than the one published by Chilibeck et al. [1999], where FES-cycle training of 30min, three days weekly for eight weeks in people at least three years post SCI resulted in a 23% increase in muscle fibre area, as determined from muscle biopsies. The higher training volume chosen in the present study is likely to lead to greater muscle hypertrophy, which in turn is likely to lead to greater increases in BMC. Muscle growth was almost complete at 9 months and reached a plateau thereafter. This is in contrast to bone parameters which do not seem to have reached a new steady-state within the time frame of the present study. It is interesting that in our study population none of the measured bone changes were related to the increase in CSA_{muscle}. However, the relationship between muscle and bone in paralysed limbs may be different than in the limbs of able-bodied people, since muscle contractions are produced by FES. Bending of the bones would usually be minimised by the co-contraction of antagonistic muscles during normal physiological movement, but this would not occur with FES-induced activation of leg muscles, thus potentially producing much higher strains in the bones in question. While bone strains generated by FES modalities have not been investigated to date, it would be unrealistic to assume that physical adaptations to FES exercise would follow the same physiological principles as volitional exercise.

In the tibia, none of the measured bone parameters changed over the duration of FES-training. This confirms the findings of previous studies regarding the effect of FES-cycling on the lower leg in people with SCI [Bloomfield et al., 1996; Clark et al., 2007; Eser et al., 2003; Hangartner et al., 1994; Pacy et al., 1988]. However, it is in contrast to other studies [Chen et al., 2005; Mohr et al., 1997b] that found a positive impact of FES-cycling in the proximal tibia. Our findings indicate that pedalling with no or inadequate electrical stimulation to the muscles of the lower leg does not have any impact on bone parameters at the tibia (Fig. 4). Hence, we conclude that compressive, rotational and tensile forces elicited by passive movement (or by a low level stimulation of calf muscles, as seen with those six subjects who had calf stimulation) in the lower limb during the cycling motion do not stimulate bone formation. Despite the fact that only a small number of subjects were included in this study, it is unlikely that the present study was under-powered as not even a trend for changes in tibial bone parameters was found. We can only speculate that if the lower leg muscles were activated adequately by FES, these muscle contractions may have produced sufficiently strong forces to result in an increase in bone substance. This notion is supported by the encouraging results of a study employing isometric plantar flexion stimulation training in patients with acute SCI. The training was found to partially prevent bone loss in the proximal tibia [Shields
et al., 2006]. As the distal tibia is known to be a common site for low trauma fractures [Eser et al., 2005; Garland and Atkins, 2001; Vestergaard et al., 1998] further investigation into the efficacy of electrical stimulation of the lower leg muscles on tibial bone strength is warranted.

Our results suggest that bone changes caused by FES-cycling in the paralysed limbs of SCI individuals are site-specific. To appropriately assess the osteogenic effect, bone measurements need to be performed at several skeletal sites, some dominated by trabecular and some dominated by cortical bone. The recognition of the exact processes requires a sufficiently long observation period. We suggest that the period of monitoring chosen in the present study was adequate to identify the effect on trabecular bone compartments, but not on cortical bone. We propose an observation period of at least 24 months to conclusively assess the effect of FES-training on density and geometric properties of the diaphysis. This notion is supported by results of studies that measured bone parameters reflecting trabecular bone in the distal femur and/or the proximal tibia following FES-cycling, and that found a recovery in BMD [Bloomfield et al., 1996; Chen et al., 2005; Mohr et al., 1997b] or a reduction of bone loss [Hangartner et al., 1994]. In contrast, those groups who analysed bone parameters of the femoral or tibial shaft [Eser et al., 2003; Leeds et al., 1990] did not find any adaptation to the FES-training.

With regard to the soft tissue in the lower leg, CSAfat at this site decreased by more than twice as much as in the thigh, i.e. 17% (Table 2). Interestingly, despite the fact that five subjects had no calf stimulation while six did, both groups showed a similar decrease in CSAfat: −17.5% in the calf-stimulated versus −15.9% in the non-calf-stimulated group. These findings indicate that FES-cycle training may have elicited heightened fat metabolism in our subjects, a finding with important clinical implications as people with a SCI are at high risk of developing obesity and diabetes [Gupta et al., 2006].

Conclusions

High-volume FES-cycling induces site-specific bone adaptation in the paralysed limbs of persons with long-lasting SCI. This is the first study that has assessed the effect of high-volume FES-cycling in this population at several measurement sites of the legs, using pQCT. The detailed study of various sites in the legs revealed that bone formation is limited to the distal femur. Bone formation may also occur in the shaft of the femur, but observation times longer than in the present study would be needed to detect it. No effects were found in the tibia, a finding that may be due to the absence (or sub-maximal application) of FES to the lower leg muscles in this study. As persons with chronic SCI suffer from severe osteoporosis and are at risk of sustaining low trauma fractures leading to comorbidities and reduction in quality of life, improvement of bone strength at the femur has considerable clinical relevance. Although several groups investigated the impact of FES-cycling on bones [BeDell et al., 1996; Bloomfield et al., 1996; Chen et al., 2005; Eser et al., 2003; Hangartner et al., 1994; Leeds et al., 1990; Mohr et al., 1997b], there is still a lack of clarity about the optimal dosage of FES-cycle training to achieve maximal bone formation in the paralysed legs. Furthermore, the effect of increased BMD at the distal femur after FES-cycling on fracture risk remains unclear and needs to be assessed in further studies.
4.6 Effect of detraining on bone and muscle tissue in subjects with chronic spinal cord injury after a period of electrically-stimulated-cycling: a small cohort study

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Introduction

Spinal cord injury (SCI) leads to a rapid and distinct reduction in bone tissue in the paralysed regions [Biering-Sorensen et al., 1990]. The most affected areas are the metaphyseal-epiphyseal regions of the distal femur, and of both the proximal and distal tibia, where bone mineral content (BMC) is reduced by approximately 50% and 70%, respectively, within the first few years post-injury [Eser et al., 2004]. The clinical significance of this bone loss and associated reduction in bone strength is that it exposes those with SCI to a high risk of low-energy fractures, with a lifetime risk of suffering a fracture in the lower limbs being twice as high as in able-bodied people [Vestergaard et al., 1998].

In order to increase bone strength in the paralysed limbs and reduce fracture risk, the application of load on bones in people with SCI was investigated. Active loading i.e. through muscle contractions by means of functional electrical stimulation (FES)-induced cycling was found to lead to a site-specific recovery in bone mineral density (BMD) in the paralysed legs up to 14% [Chen et al., 2005; Frotzler et al., 2008b; Mohr et al., 1997b]. However, despite the positive effect of FES-cycling on bones after SCI, little is known about bone behaviour in the paralysed limbs once the FES-cycling is discontinued. To our knowledge only 2 groups [Chen et al., 2005; Mohr et al., 1997b] have as yet investigated the impact of a reduced FES-cycle training programme or detraining on bone status in the paralysed limbs. Both groups found that the partially reversed bone loss after initial FES-cycling was lost within the subsequent 6 months of either reduced FES-cycling or detraining. However, whether an initial phase of high-volume FES-cycling causes similar bone adaptations during detraining or reduced FES-cycling remains unclear.

The aim of the present study was therefore to investigate the impact of detraining or reduced FES-cycling following a one-year high-volume FES-cycle training on bones and soft tissue in the paralysed limbs of people with chronic complete SCI by detailed peripheral quantitative computed tomography (pQCT) assessment. For this purpose, we performed a follow-up
study of the subjects who participated in the previously published study on the osteogenic effects of high-volume FES-cycling [Frotzler et al., 2008b].

**Materials and methods**

The study was conducted as a multi-centre design at the Queen Elizabeth National Spinal Injuries Unit and the University of Glasgow, Glasgow, UK as well as at the Swiss Paraplegic Research, Nottwil, Switzerland. The study was approved by the local ethics committees.

**Subjects**

Eleven people with a chronic SCI participated in a high-volume FES-cycling programme (up to 5 sessions per week, for one year) as published previously [Frotzler et al., 2008b]. Of those, 4 men and one woman aged 38.6±8.1 years (age range 27.7–48.4 years) who showed a significant training effect on bone parameters attended follow-up investigations after termination of the high-volume FES-cycling programme. All subjects had motor-complete post-traumatic paraplegia (grade AIS A, i.e. American Spinal Injury Association (ASIA) impairment scale (AIS) [Maynard et al., 1997]), with a lesion level between T4 and T7 and a lesion duration of 11.4±7.0 years (range 3.6–19.8 years). Exclusion criteria were current or past unhealed bone fractures, diseases known to affect bone metabolism and use of bone acting drugs. All subjects were informed about the protocol for the bone measuring procedure and were required to sign an informed consent form.

**Reduced FES-cycle training or detraining**

At the end of the 12-month high-volume FES-cycling programme (for more training details see Frotzler et al. [2008b]), subjects were free to stop or continue the FES-cycling at their own desired training volume (as determined by the number and duration of sessions per week and the training intensity, i.e. resistance). Four subjects stopped the FES-cycling programme and one subject decided to continue a reduced FES-cycle training and performed 2–3 training sessions per week, each lasting 30min.

**Measurements**

Bone and soft tissue measurements were performed with a pQCT scanner (model XCT3000, Stratec Medical, Pforzheim, Germany) with regard to volumetric BMD and bone geometry, as well as muscle and fat cross-sectional areas. Image processing and calculation of numerical values were performed using the manufacturer’s software package (version 6.0 B). Peripheral QCT scans were performed 4 times: at baseline prior to the FES-intervention (t1), at the end of the high-volume FES-cycling programme (t2), as well as after 6 (t3) and 12 months (t4) of detraining or reduced training. Bone data at t1 and t2 have been published previously [Frotzler et al., 2008b]. In the tibial and femoral epiphyses, BMC, total BMD (BMDtot) and trabecular BMD (BMDtrab) were calculated. In the diaphyses, BMC and cortical BMD (BMDcort) were calculated. In addition, muscle cross-sectional area (CSAmuscle) and fat CSA (CSAfat) both in the thigh and the shank were also identified.
Statistical analysis
Due to the small number of subjects, only descriptive statistics were performed. The mean and standard deviation of the changes in bone and soft tissue parameters between t2 and t3, and between t3 and t4 were calculated to analyse the impact of detraining or reduced FES-cycling. To describe the effect of detraining or reduced FES-cycling, changes in bone and soft tissue parameters between t1 and t2 were compared with those between t2 and t4.

Results
Impact of detraining on bone and soft tissue
Within 12 months of detraining, a mean of 73.0±13.4% of the total bone gain achieved between t1 and t2 in BMDtrab in the distal femur was still preserved at t4 (Fig. 1). At this site, 63.8±8.0% gained in BMDtot and 59.4±3.9% gained in BMC during the FES-cycling period were also preserved at t4. In the femoral shaft, BMC and BMDcort decreased by 1.8±0.8% and 3.6±2.8% between t2 and t4, which is comparable to the decreases in this site found between t1 and t2. With regard to the impact of detraining on soft tissue in the thigh, 22.1±21.0% of the total increase in CSAmuscle achieved between t1 and t2 was preserved after 12 months of detraining. The main decrease of the total gain in CSAmuscle (68.6±34.0%) occurred within the first 6 months of detraining, i.e. between t2 and t3. CSAfat in the thigh did not change considerably during the FES-cycling programme, but increased in the phase of detraining and was, on average, 7.6% higher at t4 than at t1. With regard to the impact of detraining on the tibia, bone parameters at this site only changed by between –1.3% and 1.6%. This is similar to the negligible bone changes found during the high-volume FES-cycling programme between t1 and t2. Soft tissue parameters in the lower leg also showed only minor changes between t2 and t4; both CSAmuscle and CSAfat increased on average by 3.2% and 3.5%.

Impact of reduced FES-cycling on bone and soft tissue
One subject continued a reduced FES-cycle training programme between t2 and t4 and was able to preserve 96.2% and 95.0% of the total gain in distal femoral BMDtot and distal femoral BMDtrab (Fig. 1) achieved during high-volume FES-cycling.

In the femoral shaft, BMC and BMDcort decreased by 7.3% and 5.4%, respectively, between t2 and t4. It should be noted that BMC at this site showed similar decreases during high volume FES-cycling, i.e. between t1 and t2. With regard to the impact of reduced FES-cycling on the soft tissue in the thigh, both CSAmuscle and CSAfat decreased by 1.5% and 17.0%. Thus, nearly the complete gain in muscle tissue achieved during high-volume FES-cycling was still preserved at t4. In the tibial epiphysis and diaphysis, bone parameters showed decreases of between 1.3% and 4.8% in the phase of reduced FES-cycling. Interestingly, these decreases are less pronounced than those observed in this subject during high-volume FES-cycling with decreases in tibial bone parameters of up to 18.6%.
Figure 1: Adaptive changes in trabecular bone mineral density (BMD) of the distal femur. Values are shown at baseline ($t_1$), after 12 months of high volume functional electrical stimulation (FES)-cycling ($t_2$), and after 6 ($t_3$) and 12 months ($t_4$) of detraining or reduced FES-cycle training. Note that one subject (black line) continued reduced FES-cycling between $t_2$ and $t_4$, whilst 4 participants (grey lines) stopped high-volume FES-cycling at $t_2$. The dashed line indicates the start of the detraining or reduced FES-cycling following high-volume FES-cycling.

**Discussion**

The effect of detraining or reduced FES-cycle training following 12 months of high-volume FES-cycling on bones in the lower extremities of 5 persons with chronic complete SCI was investigated. This is the first study with such a long FES intervention and follow-up period and the first to measure bone and soft-tissue parameters by pQCT. Despite the small number of subjects the present study shows a clear tendency: between 59% and 73% of the gain achieved in BMC, BMDtot and BMDtrab in the distal femoral epiphysis following high-volume FES-cycling were still preserved after 12 months of detraining. This finding is in contrast to the documentations of Chen et al. [2005], who found that after stopping FES-cycling (from 5 to zero FES-cycle training sessions per week) bone values returned to baseline levels within 6 months of detraining. According to our results, it appears to take more than 12 months of detraining to resorb the bone tissue that was gained within 12 months of high-volume FES-cycling. In addition, according to Wilmet et al. [1995] who found a decrease of BMC of approximately 4% per month in areas rich in trabecular bone during the first year of SCI, bone loss following high-volume FES-cycling in people with chronic SCI seems to be slower with an average decrease in distal femoral BMC of 0.5% per month. The
reason for this finding remains unclear, and needs to be investigated in further studies. We speculate that factors that affect bone metabolism, such as vascular atrophy after SCI, may slow down bone loss in people with chronic SCI. Regarding the muscle tissue in the paralysed legs, more than two-thirds of the muscle gain achieved during high-volume FES-cycling was lost within one year of detraining. This is comparable to the atrophy in CSA muscle found in people after acute SCI [Castro et al., 1999]. Reduced FES-cycle training (2.5 training sessions of 30min per week) seemed to preserve the increase in both the bone at the distal femur and the muscle tissue of the thigh that resulted from high-volume FES-cycling. The present results are in contrast to the findings of Mohr et al. [1997b], who found the total gain in areal BMD (+10%) in the proximal tibia following FES-cycling for 30min per day, 3 days per week for 12 months to be lost after a further 6 months with only one training session per week. It may be that there is an important difference between 1 and 2 training sessions per week, with one weekly training session turning a bone’s disuse mode “on”, thus resulting in resorption of the gained bone substance (as described by Frost [1987]). According to Frost’s mechanostat-theory [Frost, 1987], load-induced strains in bones provide the primary control signal underlying the biological responses of bone to its mechanical usage. Thus, strains above a certain threshold turn modeling “on”, resulting in an increased bone mass and strength. On the other hand, if strains are too small or absent, a disuse mode of remodelling turns “on” and bone will be resorbed until bone strength is adapted to its new mechanical usage. Consequently, one weekly training session may not be enough to preserve an adequate muscle mass necessary to induce large enough bone strains, while 2 sessions per week may preserve the previously achieved gain in muscle mass sufficiently in order to preserve the achieved gain in BMC.

Several studies documented the fact that people with SCI are at a higher risk of low-trauma fractures [Vestergaard et al., 1998], and that fractures mainly occur in the distal femur and in the distal and proximal tibia [Eser et al., 2005]. Since fractures may lead to comorbidity and a reduction in quality of life, improvement of bone parameters in the femur may have considerable clinical relevance. High-volume FES-cycling has the potential to increase bone strength of the distal femur in people with chronic and complete SCI [Frotzler et al., 2008b]. Hence, we assume that fracture risk at this site might be reduced after high-volume FES-cycle training and that this protective effect is subsequently sustained following a period of reduced FES-cycling, or even after 12 months of detraining. Indeed, the BMD trabecular values in the distal femur below the reported fracture threshold [Eser et al., 2005]. Four of our subjects had baseline BMD trabecular values in the distal femur below the reported fracture threshold [Eser et al., 2005]. However, at the end of the 30-month period of monitoring (including 12 months of high-volume FES-cycling and 12 months of detraining or reduced FES-cycling), all of our subjects, independent of whether they stopped or continued FES-cycling, remained above this fracture threshold. Thus, for a lasting improvement in bone parameters in the distal femur of people with chronic complete SCI we recommend a 2-phase FES-cycle training schedule, the first phase consisting of high-volume FES-cycle training in order to increase bone parameters, followed by the second phase consisting of reduced FES-cycle training in order to preserve bone parameters. Studies with even longer follow-up periods are needed to determine how long this second phase of reduced training volume may be.
4.7 Training and detraining of a tetraplegic subject: high-volume FES cycle training

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Published in: Am J Phys Med Rehabil 2008; 87: 56-64.

Introduction

Spinal cord injury (SCI) can often result in an imposed sedentary lifestyle. Complete motor SCI leads to lesion-dependent paralysis of muscle, which can be followed by cardiopulmonary complications and an increased risk for long bone fractures in the paralyzed extremities [Garshick et al., 2005; Powell and Blair, 1994; Zehnder et al., 2004]. To reduce such complications secondary to SCI, exercise training can play a key role [Mohr et al., 1997a].

By means of functional electrical stimulation (FES), it is possible to stimulate the paralyzed leg muscles of persons suffering from SCI. These muscles are well suited to be used for cardiopulmonary training as a large muscle mass is involved. The effect of FES cycle training has been investigated in several studies [Faghri et al., 1992; Figoni et al., 1990; Janssen et al., 1998; Mohr et al., 1997a; Mohr et al., 1997b; Raymond et al., 2002].

It has been shown that cardiopulmonary responses during FES cycling for a given power output are higher in persons with paraplegia in comparison with able-bodied persons [Raymond et al., 2002]. For an equivalent power output during cycling, a higher cardiac output was elicited in the paraplegic subjects in contrast to the able-bodied cyclists. This inefficiency of FES cycling in comparison with volitional cycling was also demonstrated in other studies [Glaser et al., 1989; Janssen et al., 1998], making FES cycling very suitable for cardiopulmonary training in persons with SCI. It is for this reason that persons with tetraplegia might benefit even more from FES cycling with respect to the cardiopulmonary system in comparison with persons with paraplegia, because they otherwise have so little muscle mass to stress their cardiopulmonary system. Moreover, the highly overused upper-extremity muscles of persons with tetraplegia can be protected from additional loads due to physical training [Burnham et al., 1993].

Most studies with FES cycling involve training sessions of 30min, three times per week, for a varying period of time [Eser et al., 2003; Faghri et al., 1992; Hooker et al., 1990; Janssen et al., 1998; Mohr et al., 1997a] As far as the cardiopulmonary system is concerned, it is not known whether high-volume FES exercise (training for more than 30min, several times per week) can improve aerobic fitness even more. It is one of the aims of this article to investigate to what extent the cardiopulmonary fitness in a tetraplegic subject can be improved.
It is generally accepted that bones adapt their structure and strength to applied loads [Frost, 1997]. Thus, the effect of loading on bones from FES-induced muscle contractions in persons with SCI has been investigated in several studies. These findings were not always in agreement: some documented a positive effect of FES-induced muscle training with an increase of 10–13% in the areal bone mineral density (BMD) of the proximal tibia [Belanger et al., 2000; Chen et al., 2005; Mohr et al., 1997b]. Belanger et al. [2000] documented an increase of areal BMD of 30% in the distal femur after a loaded knee extension training of 1h, five times per week, for 6 months In contrast, a study by Eser et al. [2003] on bone in recently injured patients shows no significant attenuation of bone loss from the tibial diaphysis before and after a program of FES cycling (2.3 sessions per week of 30min per session, for an average of 6 months). This result was also confirmed by the groups of Leeds et al. [1990] and Bloomfield et al. [1996], who found no bone adaptation after FES cycle training in persons with chronic SCI.

The discrepancies in the outcomes of these different studies on bone and SCI subjects can be attributed in part to the different places the bone measurements were taken. In those studies that measured bone density in the proximal femur and the femur shaft, there was no effect on bone density after FES [BeDell et al., 1996; Eser et al., 2003; Leeds et al., 1990]. In the studies that measured the density of the distal femur and proximal tibia after FES, however, increases in bone density were found [Belanger et al., 2000; Chen et al., 2005; Mohr et al., 1997b]. Duration and intensity of training have also differed greatly. Subjects that trained with more than 18W [Bloomfield et al., 1996], for 1h, five times per week [Belanger et al., 2000], or for a training period of 12 months [Mohr et al., 1997b] were able to show improvements in bone density.

Taking these results together, it seems that FES cycle training may be an ideal way to promote cardiopulmonary fitness and in addition might have the potential to increase BMD in persons with tetraplegia. However, the intensity, frequency, and duration have been very different in previous studies, varying from three times per week for 30min for 4 months, to five times per week for 1h for 6 months. For this reason, we set out to study the effect of high-volume FES cycle training (five times per week, 1h per session, maximal sustainable power output, 1 year duration) on maximal power output, cardiopulmonary fitness, and bone formation. Special attention is given to the training compliance of the subject.

**Methods**

**Participant**
A 31 year-old subject (81kg, 187cm) with a C6-level SCI (ASIA B), 3 years after injury, performed 1 year of FES cycle training at home. Exercise tests and bone measurements were performed at the Institute of the Swiss Paraplegic Research, Nottwil, Switzerland. The study was approved by the ethics committee of the Canton of Lucerne, and the subject gave his written informed consent.

**Study outline**
Before starting the one year exercise training program, the subject performed 3 months of muscle conditioning on an FES cycle ergometer (see description below) with varying
resistance three times per week for 20min. When the subject was not able to maintain the target cadence of 50 rpm, a physiotherapist supported the pedaling manually so that the cadence did not drop below 35 rpm, which happened only during the first two to three sessions. At the end of the muscle conditioning phase, the subject had to be able to cycle unloaded by means of his own leg muscle force generated by FES for 20min at 50 rpm. After these 3 months of muscle conditioning, the training of the subject was interrupted because of a lack of caregivers. This interruption lasted 3 months. Because the subject was still able to cycle for 20min at 50 rpm after this break, the first incremental exercise test (IETM0) was performed, and the 1 year training intervention with FES cycling started.

The muscle conditioning and the training both took place on the same FES cycle ergometer. During the muscle conditioning, however, the subject was not constrained by specific training targets; that is, there were no minimal limits set for the time or frequency that he spent on the ergometer. During the conditioning, he was focused on cycling for as long as possible. He cycled mostly without resistance. In total, he performed 17 training sessions of not always 1h. At the start of the training program (i.e., after the first incremental test), the subject had to cycle according to a formal training plan. For this purpose, he kept a weekly diary in which the frequency, duration, and mean power output of the cycle training were reported.

The ultimate target for the training program was to reach a training intensity of a 1h cycle training session five times per week with maximal possible power output. The time, resistance, and frequency of the interventions were increased gradually during the first 3 months up to this target. We considered a mean of 3.5 full hours per week (i.e., three to four sessions per week of up to 1h) for the first 3 months as 100% compliance (low-impact training). At the end of the first 3 months, the subject was given a training target of 1h, five times per week, for the remaining 9 months (high-impact training, Table 1).

Table 1: Overview of the training plan of the subject

<table>
<thead>
<tr>
<th>Training</th>
<th>Duration</th>
<th>100% Compliance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle conditioning</td>
<td>6 months</td>
<td>—</td>
</tr>
<tr>
<td>Low-impact training</td>
<td>3 months</td>
<td>In time: 210 minutes per week; In frequency: 3.5 times per week</td>
</tr>
<tr>
<td>High-impact training</td>
<td>9 months</td>
<td>In time: 300 minutes per week; In frequency: 5 times per week</td>
</tr>
</tbody>
</table>

Compliance
The subject filled in a diary every day after his cycle training, noting down the power output and the duration of cycling time. From this diary, the compliance in frequency (how many times the subject trained per week), time (i.e., the actual time the subject spent on the bike), and mean power output were calculated. We refer to Table 1 for the exact description of 100% compliance in frequency and time. The year of training was divided into four periods of 3 months. During the first 3 months, the subject was supposed to train 3.5 times per week.
(i.e. 100% compliance in frequency). As an example, a subject who trained 20 times in total during 12 weeks would have compliance in frequency of 20/42 (3.5 x 12 weeks), or 47.6%. Because he was supposed to train 3.5 x 60min (i.e. 210min/week during 12 weeks), that would make 2520min for this period. The minutes the subject was on the cycle were counted and divided by the minutes he was supposed to be cycling. For the remaining 9 months, the training compliance in frequency was 100% when the subject performed 60min of cycling, five times per week. The compliance was always calculated for the 3 months of training before an incremental exercise test (IET). The mean power output was likewise calculated. It was the average of the power outputs of every day’s training calculated for the 3 months of training before an IET.

Training equipment
Training was performed by means of a stationary FES cycle ergometer (StimMaster, ELA, Dayton, OH). This FES cycle consists of a lower-extremity ergometer with a flywheel, driven by a stimulus control unit that regulates the stimulation of the leg muscles. Quadriceps, gluteal, and hamstring muscles were stimulated bilaterally via pairs of adhesive surface electrodes. Stimulation was performed with a frequency of 50Hz. Pulse width was set at 300µs, pulse amplitude was increased automatically from threshold (just palpable contraction) to a maximum of 140mA. The controller regulated the intensity of the stimulation necessary to maintain 40–50 rpm. The maximum level of resistance was set before the start of the training; during the training session, the actual resistance was adjusted by the system according to muscle fatigue. When the pedaling frequency fell below 40 rpm, the resistance was reduced; when the subject was pedaling faster than 50 rpm, it was increased. The preset maximal level of resistance could be increased in 10 steps from 0 up to 11N, which resulted in 54W at 50 rpm.

Test equipment and procedure for the IET
Changes in training status were measured with a workrate- and cadence-controlled IET described in detail elsewhere [Hunt et al., 2004]. For this test, an adapted recumbent tricycle (Inspired Cycle Engineering Ltd, Cornwall, UK) equipped with an electric motor and a feedback system for controlling the workrate and cadence was used. IETs were performed every 3 months, starting with the first test (baseline) after the initial muscle conditioning phase.

For the IET, electrodes were placed on the quadriceps, gluteal and hamstring muscles and the subject was transferred on to the tricycle. The lower legs were fixed in knee-length orthoses to provide optimal support for the legs. After a 3min resting period, 4min of motor-assisted passive cycling was performed. Then, the stimulation started. The target workrate was linearly increased over time. Stimulation was performed using six channels by means of a Stanmore Stimulator [Phillips et al., 1993], starting with a pulse width of 0 µsecs, which was increased up to maximally 500µs by the feedback system, to maintain the target work rate. Stimulation was performed with a pulse frequency of 50Hz and a pulse amplitude between 100 and 140mA (depending on the muscle). The maximal power output was observed at 500µs. Heart rate (OxyTip, Datex-Ohmeda 3900, Louisville, KY), breath-by-breath gas exchange, and ventilator variables (Oxycon Alpha, Jaeger, Hoechberg, Germany) were measured continuously throughout the whole IET.
Capillary blood for enzymatic lactate analysis (Super GL Ambulance, Ruhrtal Labor Technik, Möhnesee, Germany) was sampled from an earlobe at rest, every 3 min during the IET, immediately after cessation of the exercise test, and every 2 min until the end of the recovery phase. Lactate sampling was done only at the first IET (after the muscle conditioning; i.e., IET-M0) and the IETs at 6 months (IET-M6) and 12 months (IET-M12).

Bone measurements
Bone measurements by means of peripheral Quantitative Computed Tomography (pQCT: XCT 3000, Stratec Medical Systems, Pforzheim, Germany) were performed before muscle conditioning, 9 months after the start of the muscle conditioning (i.e., 3 months into the cycle training program), and at cessation of FES cycle training. Trabecular and total BMD, bone mass in the distal femoral and tibial epiphyses, and bone mass and cortical crosssectional area (CSA) in the tibial and femoral diaphyses were measured bilaterally. Muscle and fat CSA of the thigh and lower leg were also determined during these measurements.

In summary, we refer to Figure 1 for an overview of the study design and tests that were carried out. The IETs (IET-M0 to IET-M12) were performed every 3 months after start of the cycle training, lactate measurements were made every half year during cycle training, and bone measurements were made every 9 months, with the first one at the start of the muscle conditioning.

![Figure 1: Overview of tests](image)

solid line = muscle conditioning; dotted line = FES cycle training; IET = incremental exercise test; LM = lactate measurement; BM = bone measurement

Statistics
We performed a Spearman rank correlation. A $P$ value of 0.05 or less was considered to be the level of significance.
Results

Muscle conditioning
Muscle conditioning was started successfully but was interrupted after 3 months because of a lack of caregivers. Then the subject did not perform muscle conditioning at all for the next 3 months. Nevertheless, he started formal cycle training after 6 months with the first IET as he could cycle for 20 min without resistance (prerequisite for entering the cycle training phase).

Compliance
The subject’s commitment during cycle training is expressed by the compliance in time and frequency. From 0 to 3 months, the compliance in frequency was 55.1%; the compliance in time was 22.9%. The subject not only trained less frequently than required, he also trained less than 1 h per session as he became tired before 1 h was completed (resulting in the low compliance in time of 22.9%). The subject trained more consistently in the period from 3 to 6 months. This resulted in a high compliance at 6 months. The compliance in frequency and time at 6 months were more than 80%. After 6 months, the compliance decreased steadily. For the actual frequency and time compliance, we refer to Figure 2.

Figure 2: Session and time compliance during the periods between the different incremental exercise tests (IETs) and maximal power output at these IETs. The horizontal dotted line indicates the training that the subject should have performed, the lines underneath indicate the actual training frequency and time. IET-M0 = IET at 0 months, IET-M3 = IET at 3 months, etc.
From the diary, the intensity of the training program was also deduced. We calculated a mean power output of 6.5, 20.4, 18.0, and 9.0W for the four 3 month periods of cycle training, respectively.

**Power output**
The measured peak power output from the IETs is displayed in Figure 2. Increases in peak power output were achieved rapidly. At the first IET, after the muscle conditioning phase, the subject reached 15W. The highest power output measured (increase of 113%) was at 6 months. Thereafter, the performance decreased steadily.

The peak power output on the IET at 6 months (IET-M6) was 32W. The mean power output of the training diary was also the highest in the period just before the IET-M6 (20.4W; i.e. 63.8% of the peak power output). The mean power outputs from the diary expressed in percentage of the peak power output at 6 months (32W) were 20.3, 63.8, 56.3, and 28.1%, respectively. The highest peak power was measured when the subject trained with the highest mean power output in the preceding training phase.

**Relationship between compliance and maximal power output**
The compliance and the mean power output during training varied considerably. The maximal power output during the IETs fluctuated accordingly. This was expressed by a high correlation between the compliance in frequency and peak power output (Spearman rank correlation, \( r = 0.8 \), \( P = 0.33 \)). The relation between the compliance in time and intensity of training and the peak power output turned out to be less strong (\( r = 0.4 \), \( P = 0.75 \) and \( r = 0.4 \), \( P = 0.75 \), respectively). The increase and decrease of compliance and peak power output can be observed to proceed in parallel (Fig. 2).

**Cardiopulmonary changes**
During the IET, peak heart rate (HRpeak), peak oxygen uptake (\( \text{VO}_2\text{peak} \)), and blood lactate concentrations (every 6 months) were registered. In Figure 3, the HRpeak and the \( \text{VO}_2\text{peak} \) that were achieved during each IET are displayed. The increase in HRpeak was 11.8%. \( \text{VO}_2\text{peak} \) increased by more than 100% at 6 months (to 1642ml·min\(^{-1}\)) in comparison with the baseline value (808ml·min\(^{-1}\)) at the beginning of the study (IET-M0). The changes in blood lactate concentrations during the different IETs are presented in Figure 4.
Figure 3: Course of maximum values during the different IETs. IET-M0 = IET at 0 months, etc.

Figure 4: Changes in lactate concentration during the different incremental exercise tests (IET-M0, IET-M6, IET-M12). IET-M0 = at the beginning of the cycle training; IET-M6 = after half a year cycle training; IET-M12 = at the end of the study.
Bone and muscle/fat changes
Mean changes of bone and soft tissue are presented in Table 2. The main increases of bone parameters were found to be in the distal femoral epiphyses, especially in the trabecular BMD. Furthermore, muscle CSA in the thigh reached its measured maximal peak within the first 3 months of cycle training. It is not clear whether this value increased even more after the second measurement, because the third measurement was 9 months later. In contrast, the bone and muscle tissues in the lower leg were found to have only minor changes during the 18 months of cycling; most of the bone and muscle tissues showed slight decreases, with the exception of the large decrease of fat CSA (-13.8%) at the end of the study (Table 2).

Table 2: Mean bone changes [%] of the femur and tibia during functional electrical stimulated cycle training.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Percent change BL to M3</th>
<th>Percent change BL to M12</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Femur</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epiphysis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trabecular BMD</td>
<td>-4.2</td>
<td>3.9</td>
</tr>
<tr>
<td>Total BMD</td>
<td>-2.2</td>
<td>1.5</td>
</tr>
<tr>
<td>Mass</td>
<td>-3.4</td>
<td>0.2</td>
</tr>
<tr>
<td>Diaphysis</td>
<td>-3.9</td>
<td>-5.5</td>
</tr>
<tr>
<td>Cortical CSA</td>
<td>-3.0</td>
<td>-4.8</td>
</tr>
<tr>
<td>Mass</td>
<td>26.8</td>
<td>25.3</td>
</tr>
<tr>
<td>Muscle CSA</td>
<td>-1.8</td>
<td>-2.8</td>
</tr>
<tr>
<td>Fat CSA</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Tibia</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epiphysis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trabecular BMD</td>
<td>1.1</td>
<td>0.9</td>
</tr>
<tr>
<td>Total BMD</td>
<td>-1.1</td>
<td>-0.4</td>
</tr>
<tr>
<td>Mass</td>
<td>-1.1</td>
<td>-1.4</td>
</tr>
<tr>
<td>Diaphysis</td>
<td>-0.5</td>
<td>-0.9</td>
</tr>
<tr>
<td>Cortical CSA</td>
<td>-2.8</td>
<td>-2.0</td>
</tr>
<tr>
<td>Muscle CSA</td>
<td>-1.1</td>
<td>-0.9</td>
</tr>
<tr>
<td>Fat CSA</td>
<td>-4.8</td>
<td>-13.8</td>
</tr>
</tbody>
</table>

Right and left values are averaged. BL: start of conditioning phase (-6 months); M3: after 3 months of cycle training; M12: after 12 months of cycle training; BMD: bone mineral density; CSA: cross-sectional area.
Discussion

In this study, we documented the influence of high-volume FES cycle training on power output, cardiopulmonary fitness, and BMD in a tetraplegic subject. Depending on the intensity of training, it was clearly possible to substantially increase maximal power output, \( \text{VO}_{\text{peak}} \), and \( \text{HR}_{\text{peak}} \). There was a close relationship between training compliance and the performance on the IET (Fig. 2). Also, for a given work rate, blood lactate concentration was much lower after half a year of FES cycle training. This concentration increased again when training became less intensive (Fig. 4).

As far as the correlations between the compliance in time, session, and intensity of the training to the peak power output were concerned, the relation between the compliance in session and peak power output was the strongest \((r = 0.8)\), although not significant. The other correlations were lower \((r = 0.4, \) both\). We presume that this low correlation was caused by the small sample size used for the rank correlation. However, we learned from it that the relation between the number of sessions the subject performed and his maximal power output on the IET was the strongest.

A high training compliance in tetraplegic patients is difficult to maintain because they usually need support for their FES cycle training (donning and doffing of electrodes, transfers, and presence of a caregiver during training). For the period from 3 to 6 months, the subject hired a caregiver to help him with his daily training. In this way, he achieved a high compliance during this period only, and good performance on the tests at 6 months. After 6 months of FES cycle training, the subject had difficulties finding assistance. In a study on the perceived barriers to exercise in people with SCI (tetra- and paraplegia), the persons with tetraplegia indicated significantly more difficulties with exercise in comparison with persons with paraplegia [Scelza et al., 2005].

Studies like this one are only possible with highly motivated subjects who have the regular assistance of a caregiver. More research should be performed in the field of increasing fitness in subjects with tetraplegia. Apparatus should be developed that can be used by the subject without assistance, because the level of required assistance may be a limiting factor.

Tetraplegic subjects are generally more susceptible to illness and disease, which may deprive them of regular sports activities [Hopman et al., 1996]. These illnesses make it difficult for this population to participate in regular training programs, which are a prerequisite for improvement of physical exercise performance on a long-term basis.

The importance of training for physical and psychological well-being in persons with SCI was demonstrated by different studies [Petrofsky and Stacy, 1992; Sipski et al., 1989; Tasiemski et al., 2005]. Of the 47 patients who participated in FES bicycle ergometry in a study by Sipski et al. [1989], more than 60% of patients reported improved endurance of the training program. Tasiemski et al. [2005] described a higher satisfaction with life in general in SCI subjects when they were involved in sports compared with those who were not participating in physical activities. It is clear that an activity program that is performed regularly is important for persons with tetraplegia.

As with most people with tetraplegia, \( \text{HR}_{\text{peak}} \) for the subject in this study was limited in its increase because of disruption of sympathetic innervations of the heart [Hooker et al., 1990].
For this reason the adaptation in the muscle must have given the higher contribution to the increase in cardiopulmonary fitness. FES cycle training results in a higher stimulus to adaptation in contracting muscle than in the heart muscle [Mohr et al., 1997a]. In this study we did not take muscle biopsies so we do not know whether the adaptation in the muscle was attributable to an increase in capillarization, mitochondrial content or muscle mass, all representing peripheral adaptations. From the pQCT data, we learned that during the course of the training, the muscle CSA increased by 26.8% (and may have been more at 6 months of FES cycle training when the subject had the highest compliance). Thus, the adaptation of the subject’s fitness could at least partly be attributed to the increase in muscle mass.

In able-bodied persons, there is a clear relationship between muscle mass and VO\textsubscript{2peak} [Tolfrey et al., 2006]. An improvement in VO\textsubscript{2peak} in persons with SCI can, therefore, be gained by restoration of muscle [Mohr et al., 1997a]. For persons with paraplegia, it is possible to perform arm cranking ergometry to improve cardiopulmonary fitness. However, it is also possible in this subgroup to increase VO\textsubscript{2peak} even further by performing FES cycling at the same time [Krauss et al., 1993]. For persons with tetraplegia, arm cranking exercise is more difficult, and, therefore, for this patient group, high-volume FES cycle training with the lower extremities may be a good alternative.

With reference to Figure 4, we see for the same amount of work much lower levels of blood lactate concentration for IET-M6 in comparison with the blood levels during IET-M0 and IET-M12. Whereas for the same amount of work, less lactate is produced, the muscle’s capacity for oxygen use and endurance must have been improved. The shift of the blood lactate concentration curve to the right after half a year of training and the shift back to the left when training became less frequent are in line with the changes in lactate concentration of able-bodied subjects before and after training periods [Donovan and Pagliassotti, 1990; Janssen et al., 1998].

Taken together, the effects of high-volume FES cycle training for the cardiopulmonary system suggest that with this high-volume FES cycle training we are able to offer one form of endurance training for the tetraplegic subject. The duration and frequency of the training (i.e. 1h, five times per week for 1 year) may have contributed to the positive changes in power output and VO\textsubscript{2peak} after the cycling intervention in comparison with other studies [Faghri et al., 1992; Figoni et al., 1990; Mohr et al., 1997a]. Also, for subjects with tetraplegia, the FES cycle training is considered more effective in comparison with voluntary arm cranking exercise, because it has a greater cardiac efficiency [Janssen et al., 1998].

For subjects with tetraplegia, the possibility to do exhaustive arm cranking exercise is very limited by the small amount of muscle mass available in the paralyzed upper extremities. To perform endurance training with the large muscle groups in the legs is, therefore, for this population, an ideal way to use metabolic energy and, at the same time, improve their cardiopulmonary fitness without stressing the arms.
With respect to bone adaptations to FES cycle training, we found a small increase of bone values in the distal femoral epiphyses (Table 2). In contrast, in the tibia there was no adaptation to FES cycle training (Table 2). This may be attributable to the fact that the muscles of the lower legs were not stimulated, and, thus, there was a lack of muscle-induced loading on the bones in this region. However, within the first 3 months of high-volume FES cycling, most of the femoral and tibial bone parameters were found to decrease, even though the muscle CSA increased. This trend of bone loss is typical in people with a lesion duration of less than 5 years, in which most of the bone substance is lost [Eser et al., 2004]. Because our subject had a lesion duration of 3 years, we assume that he was still in the phase of bone loss. We have no information about his rate of bone loss before he participated in the present study, so the question arises whether the bone loss in the lower leg might have been reduced by the FES cycle training. In the final 9 months of the training period, an increase of the femoral trabecular BMD of 3.9% took place in comparison with the baseline values. (Details of the measurements taken in the present study are described in a previously

Figure 5: Distal femoral trabecular bone mineral density (BMD) and thigh muscle cross-sectional area (CSA) before muscle conditioning (M-6) and after 3 months (M3) and 12 months (M12) of cycle training. Right and left leg values are averaged.
published study, with coefficients of variation for bone parameters at the femur and tibia of mostly better than 1% [Eser et al., 2004]). However, the thigh muscle CSA had already reached a higher value within the first 3 months in comparison with the final measurement (Table 2 and Fig. 5). This indicates a faster adaptation of muscle tissue to FES training compared with that of the bone tissue in the paralyzed legs. The bone parameters in the femoral epiphysis were found to adapt faster to muscle loading compared with the ones in the compact bone of the femoral diaphysis, which could be attributable to the greater bone surface available to be remodeled in the cancellous bone of the epiphysis. Hence, we assume that the observation time was too short to detect a bone gain in the femoral shaft. Our findings confirm the reports about a positive effect of FES cycle training on bones in the paralyzed legs [Belanger et al., 2000; Chen et al., 2005; Mohr et al., 1997b]. Thus, high-volume FES cycling may be a promising approach to strengthen bone, specifically in the paralyzed limbs of tetraplegic persons.

**Conclusions**

It is possible to increase maximal power output, cardiopulmonary fitness, and bone parameters of the paralyzed limbs in subjects with tetraplegia by high-volume FES cycle training. However, if training is not maintained, these improvements are lost. In subjects with tetraplegia, it may be difficult to maintain the high level of training required to achieve benefits. Because this was a case study, more tetraplegic subjects should be subjected to high-volume FES cycling, to prove the physical benefits of such training.
A spinal cord injury (SCI) is associated with a limited mobility, changes in metabolism and body composition [Chen et al., 2006]. One of the consequences is a reduced resting energy expenditure in these subjects [Groah et al., 2009; Monroe et al., 1998], which makes a lower daily energy intake sufficient to meet the caloric needs of this particular population. Thus, it seems not surprising that obesity is a well known problem in the SCI population [Lynch et al., 2002]. In fact, several studies showed a prevalence of obesity in subjects with SCI ranging from 40-66% [Anson and Shepherd, 1996; Chen et al., 2006; Liang et al., 2007] leading to severe medical problems such as pulmonary embolism [Green et al., 1994], metabolic syndrome [Maruyama et al., 2008] and cardiovascular diseases [Bauman and Spungen, 2008; Buchholz and Bugaresti, 2005]. However, such risk factors like cardiovascular diseases are the main reason for the reduced life expectancy in patients with SCI [Zlotolow et al., 1992]. In this context, a sufficient amount of physical activity and a balanced nutrition seem to play a key role for preventing from such complications. Further, the nutritional behaviour has also a major impact on secondary complications in people with SCI including pressure sores and prolonged wound healing [Aquilani et al., 2001], negative nitrogen balance [Rodriguez et al., 1997], digestion problems [Badiali et al., 1997; Cameron et al., 1996], reduced immunofunction [Cruse et al., 2000] or osteoporosis [Bauman and Spungen, 2000].

Despite the fact, that experts rate nutrition and the nutritional behaviour of persons with SCI as a very important issue, research concerning the diet of these subjects has received little attention in the past [Groah et al., 2008]. There exists a recently published study [Walters et al., 2009] revealing numerous nutrient inadequacies compared to the generally accepted recommendations in man and women with SCI. The first study of the following chapter [Perret and Stoffel-Kurt, 2011] aims to add some further knowledge in this field by comparing the nutritional intake of patients with acute and chronic SCI.

However, nutrition in conjunction with SCI not only gained some interest in a clinical environment but more and more in wheelchair sports as well. Thus, it seems not surprising that some athletes regularly ingest supplements with the intent to enhance competitive exercise performance or to accelerate the recovery process after exercise. As subjects with SCI reveal changes in metabolism and body composition [Chen et al., 2006] as well as a reduced resting energy expenditure [Groah et al., 2009; Monroe et al., 1998] it seems not advisable to transfer recommendations for e.g. ergogenic supplements from able-bodied persons directly into wheelchair sports. In fact, before using such substances a critical examination taking into account the special requirements of individuals with SCI becomes important and necessary. The second study of this chapter [Perret et al., 2006] offers an example how a nutritional supplement can be investigated under competitive conditions in elite wheelchair sports.
5.1 Comparison of nutritional intake between individuals with acute and chronic spinal cord injury

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Affiliations: ¹Institute of Sports Medicine, Swiss Paraplegic Centre, Nottwil, Switzerland ²Institute for Human Movement Sciences and Sports, ETH Zurich, Switzerland


Introduction

A spinal cord injury (SCI) causes a number of metabolic changes. The missing muscle innervation of paralysed limbs leads to muscle atrophy with a parallel increase in relative body fat mass. Such changes in body composition [Spungen et al., 2000] in combination with inactivity are responsible for disturbances in carbohydrate and fat metabolism of spinal cord injured patients. In these patients cardiovascular risk factors such as glucose intolerance, insulin resistance, obesity, hyperinsulinaemia, dyslipidaemia and hypertension are common [Bauman and Spungen, 2000]. This is of importance as cardiovascular diseases are the main reason for the reduced life expectancy in patients with SCI [Zlotolow et al., 1992]. From a preventive point of view a balanced nutrition seems to play a key role in this context. Moreover, nutrition of patients with SCI has a major impact on secondary complications like pressure sores and prolonged wound healing [Aquilani et al., 2001], negative nitrogen balance [Rodriguez et al., 1997], digestion problems [Badiali et al., 1997; Cameron et al., 1996], reduced immunofunction [Cruse et al., 2000] as well as osteoporosis [Bauman and Spungen, 2000].

Based on these findings, it is not surprising that nutritional information and support in this special population is a big challenge but also a matter of particular interest for nutritionists and health care professionals as malnutrition and obesity are well known problems [Lynch et al., 2002]. In fact, the nutritional behaviour of SCI patients has been investigated in several studies so far [Aquilani et al., 2001; Barboriak et al., 1983; Laven et al., 1989; Levine et al., 1992; Lynch et al., 2002; Tomey et al., 2005; Walters et al., 2009]. However, all these studies were focussed on particular patient groups with either acute or chronic SCI. To our knowledge there was no study so far, which investigated two comparable groups of acute and chronic spinal cord injured subjects at the same time. However, this seems to be helpful and necessary to warrant valid information.

The aim of the present study was to compare the nutritional intake of patients with acute (during first rehabilitation) and chronic (at least two years post injury) SCI. This comparison is of interest as leaving the hospital after first rehabilitation is often associated with a change of environmental and life style factors for patients. In many cases there is no predetermined daily routine and quality and quantity of nutritional intake can be determined by the patients themselves without any supervision, which might lead to a change of nutritional behaviour and point out the individual nutritional preferences. Therefore, we expected a higher energy intake and a less balanced nutrition of the group with chronic SCI. The findings of the present
study may provide helpful information for optimising future nutritional education during and after the rehabilitation process of patients with SCI.

Methods

Subjects
In total 24 motor complete (American Spinal Injury Association Impairment Scale (AIS) A or B), healthy spinal cord injured subjects participated in the study. A frequency matching creating two groups was applied, whereas the confounders sex (male or female) and lesion level (tetraplegia or paraplegia) were used as main matching criteria. Further, we were keen to align age, height, weight and body mass index (BMI) between subjects of the two groups as good as possible.

The first group consisted of hospitalized spinal cord injured patients with a lesion duration of less than 8 months who took part in a first rehabilitation program (acute group), whereas the second group were outpatients with a lesion duration of at least two years (chronic group). Detailed information about subjects’ characteristics can be found in Table 1.

The study was approved by the local ethics committee and subjects gave their written informed consent before the start of the study.

Nutritional protocol and analysis
Subjects were precisely instructed how to record their nutritional intake during seven consecutive days. For this purpose subjects were provided with a booklet containing seven previously prepared standard forms, detailed instructions as well as 26 photos showing different serving sizes of common foods. In order to make sure the correct use of the booklet, the content was discussed with each subject in detail and subjects were advised to complete the nutritional protocol with high diligence. The filled in protocol was analysed using the program EBIISpro (Universität Hohenheim, Stuttgart, Deutschland) which allowed evaluation of energy intake, macronutrients and micronutrients (including dietary supplements) as well as fluid and dietary fibre intake.

Body fat and resting energy expenditure
Body fat of the subjects was determined by means of the bioimpedance technique using the device Bodystat QuadScan 4000 (Bodystat Ltd., Douglas, Isle of Man, UK) and resting energy expenditure (REE) was measured via indirect calorimetry with the MedGem apparatus (Health Tech Inc., Golden, USA). All measurements were performed in the supine position after lying relaxed for 5-10min. In order to obtain reliable data, test preparations and measurement conditions were standardised. Thus, subjects were instructed to avoid food or fluid intake 4 to 5h before the measurement, as well as to abstain from caffeine or alcohol intake the last 24h. Further, physical exercise 12h before the measurements was prohibited. All subjects fulfilled this measurement conditions.
Table 1: Subjects' characteristics

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Sex</th>
<th>Lesion level</th>
<th>Time post injury [months]</th>
<th>Age [y]</th>
<th>Weight [kg]</th>
<th>Height [cm]</th>
<th>BMI [kg/m²]</th>
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<td>52</td>
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<td>6.5</td>
<td>14.5</td>
<td>6.1</td>
</tr>
</tbody>
</table>

BMI: body mass index; f: female; m: male; C: cervical lesion; Th: thoracic lesion; SD: standard deviation; *significant difference between groups (p<0.001)

Statistics:
Data are presented as means ± standard deviation (SD). For group comparisons an unpaired t-test was applied. All statistical analysis was performed using SYSTAT (Version 10, SPSS Inc., Richmond, California, USA). Statistical significance was set at p<0.05.
An undersupply of micronutrients was assumed if values were more than 20% lower compared to generally accepted guidelines [DACH, 2000].
Results

Subjects
Concerning lesion duration, there was a significant difference between the acute and chronic group (p<0.001). However, no significant differences were found for age (p=0.701), weight (p=0.544), height (p=0.236) or BMI (p=0.827) between groups (Table 1).

Nutritional analysis
No significant difference (p=0.467) was found for average daily energy intake between the acute (7.71±0.91MJ/d corresponding to 1842±217kcal) and the chronic (7.43±0.98MJ/d corresponding to 1775±234kcal) group. Whereas the acute group ingested 74.6±10.0g/d of protein, 67.8±15.3g/d of fat and 223.7±56.3g/d of carbohydrates corresponding average intakes in the chronic group were 71.4±7.9g/d, 71.5±9.8g/d and 193.8±55.9g/d, respectively and not significantly different (protein: p=0.399; fat: p=0.490; carbohydrates: p=0.205) between groups. Average percentage caloric daily intake of ingested macronutrients protein, fat and carbohydrate can be found in Figure 1 and was comparable and not significantly different (protein: p=0.776; fat: p=0.151; carbohydrates: p=0.180) between groups. Further it seems worth mentioning that subjects of the acute group consumed on average 15.6% of their daily carbohydrate intake as soft drinks, whereas it was only 7.4% in the chronic group.

Figure 1: Mean percentage caloric daily intake of ingested macronutrients in the acute and chronic group compared to generally accepted recommendations.
Mean daily micronutrients intake showed no significant differences between groups. Detailed information are presented in Table 2 and also shown in relation to the conventional recommendations for able-bodied subjects [DACH, 2000]. For the acute as well as for the chronic group intake of six vitamins (C, D, E, folic acid, pantothenic acid, biotin), potassium and iron was remarkable below the recommendations (more than 20%) for able-bodied subjects (Table 2).

Fluid intake was 2.6±0.8l per day in the acute and 3.1±1.2l per day in the chronic group but did not reach statistical significance (p=0.205) between groups. The acute group ingested an average amount of 14.4±4.9g and the chronic group of 15.6±2.4g of dietary fibres per day, which revealed no significant difference (p=0.455) between groups.

Body fat and REE
The acute group showed a significant (p=0.038) lower body fat content (15.7±4.3%) compared to the chronic group (19.4±3.8%). No significant difference (p=0.356) was found for REE between the acute (5.92±1.37MJ [1414±327kcal] per day) and the chronic group (5.46±0.97MJ [1304±232kcal] per day).

Discussion
Surprisingly, no significant differences between the nutritional behaviour of comparable groups of acute and chronic spinal cord injured subjects was found. The only difference between the two groups concerned the higher body fat content of the subjects with longer lesion duration. However, there are some interesting aspects, which might be helpful for future nutritional consulting during and after the rehabilitation process of patients with SCI. These issues will be discussed in detail below.

Energy intake
We found similar energy intakes of subjects with SCI as described earlier in the literature [Levine et al., 1992], which seem to be clearly below the values for able-bodied subjects [Buchholz et al., 2003]. In the study of Levine and co-workers [1992] the energy intake in subjects with SCI was 75% of the general recommendations for able-bodied persons. This is not surprising and reflects the reduced activity of subjects with SCI as well as the changed body composition [Monroe et al., 1998]. However, in this respect one has to keep in mind that daily activity plays a key role in energy consumption and therefore could highly influence dietary intake also in subjects with SCI. Thus, the recording of physical activity would possibly have been of interest for our study as well. Although such data are not available, we are confident that the study outcome was not significantly influenced by this limitation as we studied two comparable, frequency matched groups.
Table 2: Mean daily vitamin and mineral nutrient intake of acute (acute group; n=12) and chronic (chronic group; n=12) spinal cord injured subjects in comparison to widely accepted recommendations expressed in percent.

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Acute group (Mean ± SD)</th>
<th>Recommendation [%]</th>
<th>Chronic group (Mean ± SD)</th>
<th>Recommendation [%]</th>
<th>p-value</th>
</tr>
</thead>
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<tr>
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<td></td>
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</tr>
<tr>
<td>Vitamin A [µg/d]</td>
<td>1056.8 ± 249.8</td>
<td>132.1</td>
<td>1101.2 ± 338.0</td>
<td>137.7</td>
<td>0.718</td>
</tr>
<tr>
<td>Vitamin D [µg/d]</td>
<td>1.7 ± 1.6</td>
<td>34.3</td>
<td>1.9 ± 2.0</td>
<td>38.0</td>
<td>0.804</td>
</tr>
<tr>
<td>Vitamin E [mg/d]</td>
<td>7.6 ± 2.4</td>
<td>63.3</td>
<td>8.9 ± 2.4</td>
<td>74.2</td>
<td>0.167</td>
</tr>
<tr>
<td>Vitamin K [µg/d]</td>
<td>214.3 ± 82.0</td>
<td>357.2</td>
<td>195.2 ± 51.2</td>
<td>325.3</td>
<td>0.502</td>
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<tr>
<td>Vitamin B1 [mg/d]</td>
<td>1.0 ± 0.3</td>
<td>100.8</td>
<td>1.3 ± 0.8</td>
<td>133.3</td>
<td>0.234</td>
</tr>
<tr>
<td>Vitamin B2 [mg/d]</td>
<td>1.6 ± 0.4</td>
<td>129.9</td>
<td>1.8 ± 1.1</td>
<td>146.5</td>
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</tr>
<tr>
<td>Vitamin B3 [mg/d]</td>
<td>1.5 ± 0.4</td>
<td>123.6</td>
<td>2.1 ± 1.5</td>
<td>171.5</td>
<td>0.216</td>
</tr>
<tr>
<td>Vitamin B12 [µg/d]</td>
<td>2.8 ± 0.9</td>
<td>92.8</td>
<td>3.0 ± 1.4</td>
<td>100.0</td>
<td>0.656</td>
</tr>
<tr>
<td>Niacin [mg/d]</td>
<td>12.3 ± 3.5</td>
<td>94.6</td>
<td>15.2 ± 5.6</td>
<td>116.9</td>
<td>0.143</td>
</tr>
<tr>
<td>Folic acid [µg/d]</td>
<td>88.2 ± 25.0</td>
<td>22.1</td>
<td>96.1 ± 32.4</td>
<td>24.0</td>
<td>0.508</td>
</tr>
<tr>
<td>Pantothenic acid [mg/d]</td>
<td>4.3 ± 1.1</td>
<td>70.8</td>
<td>4.5 ± 1.2</td>
<td>75.1</td>
<td>0.582</td>
</tr>
<tr>
<td>Biotin [µg/d]</td>
<td>31.8 ± 8.5</td>
<td>70.6</td>
<td>36.2 ± 18.0</td>
<td>80.5</td>
<td>0.451</td>
</tr>
<tr>
<td>Vitamin C [mg/d]</td>
<td>79.1 ± 37.7</td>
<td>79.1</td>
<td>89.3 ± 61.7</td>
<td>89.3</td>
<td>0.631</td>
</tr>
</tbody>
</table>

| Mineral nutrient  |                         |                    |                           |                    |         |
| Sodium [mg/d]     | 2402.4 ± 533.5          | 120.1              | 2674.7 ± 616.3            | 132.4              | 0.246   |
| Potassium [mg/d]  | 2692.3 ± 925.3          | 76.9               | 2375.8 ± 570.9            | 67.9               | 0.315   |
| Calcium [mg/d]    | 1075.1 ± 255.7          | 107.5              | 1042.5 ± 311.7            | 104.3              | 0.927   |
| Magnesium [mg/d]  | 319.6 ± 78.2            | 106.5              | 329.6 ± 54.9              | 109.9              | 0.542   |
| Phosphorus [mg/d]| 1226.3 ± 246.5          | 175.2              | 1119.6 ± 181.1            | 159.8              | 0.402   |
| Iron [mg/c]       | 11.0 ± 2.9              | 73.1               | 12.7 ± 7.6                | 84.8               | 0.394   |
| Zinc [mg/d]       | 9.7 ± 1.4               | 139.1              | 9.6 ± 2.0                 | 137.7              | 0.781   |

Note that there were no significant differences between groups.
SD: standard deviation; p-values represent the comparison between the acute and the chronic group.
In general, one has to take also into account that compared to able-bodied persons - the REE of subjects with SCI is reduced [Groah et al., 2009; Monroe et al., 1998] and thus a lower daily energy intake is sufficient to meet the caloric needs of this particular population. Moreover, subjects with SCI often suffer from gastrointestinal problems, which compromise nutritional intake [Shizgal et al., 1986]. Further reasons for a reduced energy intake might also be a reduced appetite or an earlier “satiety feeling” in subjects with SCI [Laven et al., 1989].

Macronutrients
In contrast to our expectations mentioned above, no significant differences were found in percentage macronutrient intake between the acute and the chronic group (Figure 1). A possible explanation for this finding might be that long-term spinal cord injured subjects are aware of the consequences of possible changes in body composition, whereas patients with an acute SCI need to be sensitized first for their new situation. As a consequence, subjects with a chronic SCI possibly try to pay more attention to nutritional aspects, which seems to be supported by the fact, that - compared to the acute group - subjects of the chronic group consumed only half the amount of soft drinks. As a consequence, one could argue that nutritional consulting of subjects with acute SCI should be intensified and the food provided by the hospital kitchen might be reconsidered and adapted to even better meet the nutritional requirements of in-house patients.

However, both groups ingested a too high percentage of fat (acute group: 32%; chronic group: 36%) and an insufficient amount of carbohydrates (49% vs. 43%) compared to the general accepted recommendations (carbohydrates: 55-60%; fat 25-30%) for a balanced nutrition [DACH, 2000]. Our results are in line with data of a recently published study reporting macronutrient intake in 73 subjects with SCI at least one year post injury [Buchholz et al., 2003] and seem also not to differ in the nutritional behaviour of the general able-bodied population of our country [Eichholzer et al., 2010]. Taking into account that obesity [Buchholz et al., 2003] and cardiovascular diseases are common in patients with SCI [Zlotolow et al., 1992] a reduction of fat intake is strongly recommended in order to minimize cardiovascular risk factors reported by Baumann and Spungen [2000].

Protein supplementation was about 17% in both groups (Figure 1) corresponding to an average of 1.1g of proteins per kg body weight per day. In order to prevent from complications like pressure sores and tissue atrophy in persons with SCI a daily protein intake of 2g per kg body weight was recommended [Rodriguez et al., 1997]. Thus, protein intake of our subjects should be slightly increased to supply the higher protein needs due to SCI.

Micronutrients
As a result of the reduced energy intake reported in subjects with SCI [Groah et al., 2009] an adequate intake of micronutrients seems to be critical for this population. In fact, our study suggests the possibility of very low intakes of vitamin C, vitamin D, vitamin E, folic acid, pantothenic acid, biotin, potassium and iron for the acute as well as the chronic group, with no significant differences between groups (Table 2). However, although subjects were instructed in detail about completion of the nutritional protocol we can not entirely exclude some sources of error (e.g. underreporting or unsatisfactory estimation of serving size by the...
subjects; limited data set of foods or non-consideration of storage mode and detailed preparation of vegetables for data analysis), which might have lead to a moderate underestimation of reported values. Nevertheless, the present results provide some helpful information concerning micronutrient intake in people with SCI and are in line with results of recently published studies [Buchholz et al., 2003; Walters et al., 2009], which also reported numerous nutrient inadequacies in adults with chronic SCI. However, going into more detail, the amount of ingested micronutrients between different studies seems to vary widely. Whereas in the present study the amount of ingested folic acid reached on average only between 88 to 96µg per day (corresponding to 22 to 24% of daily recommendation) subjects consumed 75 to 79% of the daily recommended amount in the study of Walters et al. [2009] and 211 to 424µg per day (corresponding to 53 to 106%) in the study of Groah and coworkers [2009]. Similar observations can be found for example for vitamin D. For this vitamin the present study found ingested amounts of 34 to 38% of the daily recommendation, whereas Walters et al. [2009] reported 25 to 69% and Groah and colleagues [2009] 44 to 96%, respectively. As a consequence, an optimal consulting of patients with SCI concerning micronutrient intake should ideally base on a previous analysis of individual nutritional habits.

In general, an undersupply of micronutrients in combination with SCI has to be avoided as this may force or even cause complications already mentioned in the introduction. In this context, the question arises, if an additional, well directed micronutrient supplementation (e.g. by means of tablets) should be recommended for persons with SCI. However, the first goal should be to sensitise and educate subjects with SCI in order to change their individual dietary behaviour towards a balanced nutrition. Furthermore, interactions of medication and nutrition have to be specifically considered as most of the SCI subjects receive long-term medication (e.g. laxatives).

Fluid intake
Fluid intake in the acute as well as in the chronic group seemed to be adequate and met the general recommendations for able-bodied subjects [DACH, 2000]. An interesting finding was, that the acute group consumed a much higher (more than double) amount of soft drinks compared to the chronic group. In the acute group 15.6% of the total daily carbohydrate intake was covered by soft drink ingestion compared to 7.4% in the chronic group. During personal interviews most subjects indicated a conscious additional consumption of calories as main reason as not all subjects liked the food provided by the hospital and therefore tried to compensate the lacking calories by means of soft drinks. Based on the fact that excessive soft drink consumption has received considerable notoriety with regard to obesity, nutritionists should keep in mind our findings during counselling interviews with subjects with SCI.

Moreover, soft drinks were obviously the main reason why subjects reached a far too high phosphate intake (Table 2), which might negatively influence bone metabolism [Sax, 2001]. Indeed osteoporosis and the concomitant increase in fracture risk is known to be one of the main secondary complications in SCI [Eser et al., 2005]. It seems to be worthwhile and necessary to inform subjects about this fact and to provide some alternatives.
Dietary fibres intake
Many patients with SCI suffer from gastrointestinal complications and thus a sufficient supply with dietary fibres has to be carefully considered [De Looze et al., 1998; Han et al., 1998]. Whereas Badiali and colleagues [1997] reported positive effects of a daily dietary fibres intake of 18g in subjects with SCI, a daily intake of more than 31g per day is not recommended in this population [Cameron et al., 1996]. It seems to be difficult to provide generally accepted recommendations [Dapoigny et al., 2003] and further studies are needed to clarify this issue. In the present study mean daily dietary fibre intake of the acute group was 14.4g and 15.6g in the chronic group. Possibly our subject groups might benefit from a slightly increased intake of dietary fibres with respect to cholesterol metabolism [Kirby et al., 1981].

Body fat content
Although no differences were found in body weight, BMI and nutritional behaviour between groups, the chronic group showed a higher body fat content compared to the acute group. Possibly, the main reason for this finding is the significant difference in lesion duration between groups (Table 1). This assumption is supported by the study of Spungen et al. [2003], where a direct relationship between time post injury and total body fat content was demonstrated. The question arises if the continuously increasing body fat content of subjects with a long-term SCI can be avoided or at least retarded by means of dietary interventions, which might be helpful in reducing the risk for cardiovascular diseases in this population. In this respect, an additional well directed physical activity program might also show beneficial effects. However, to prove our speculations further research is needed.

It also seems worthwhile to mention that although no difference in BMI between groups was found, body fat content was significantly higher in the chronic group. This finding argues against the use of BMI as an estimation of fat mass in the SCI population and is in line with results of former publications [Buchholz and Bugaresti, 2005]. Therefore, in order to monitor changes in body composition in persons with SCI, e.g. during medical checkups, the application of bioelectrical impedance analysis was recommended [Desport et al., 2000].

Limitations of the study
Based on the limited number of subjects the possibility of underpowerment exists. Further, study results have to be interpreted with caution as a selective sample (only motor complete subjects; AIS A or B) was studied. Before a generalization of the present findings for the broader population with SCI can be made, a larger number of subjects including further patient groups with SCI (e.g. subjects with incomplete SCI) is necessary. However, the present study might serve as pilot work for future projects, as to our knowledge, it was the first study so far directly comparing two similar groups of acute and chronic spinal cord injured subjects at the same time. In fact, this approach seems to be helpful and necessary to warrant valid information.
Conclusions

Our study results reveal that there seem to be no differences in the nutritional behaviour of acute and chronic spinal cord injured subjects. Independent of lesion duration, subjects with SCI showed considerable deviations from the general accepted nutritional recommendations concerning macro- and micronutrients intake. As a consequence, professional nutritional education for persons with SCI should start as soon as possible after injury to prevent from nutritional related secondary complications like cardiovascular diseases. In addition, determination of body fat content and REE on regular time intervals combined with a physical activity program might be helpful as well.
5.2 Influence of creatine supplementation on 800m wheelchair performance: a pilot study

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Published in: Spinal Cord 2006; 44: 275-279.

Introduction

The positive ergogenic effect on exercise performance after creatine supplementation in healthy, able-bodied subjects was shown by several studies in the past few years [Balsom et al., 1995; Birch et al., 1994; Bosco et al., 1997; Casey et al., 1996; Greenhaff et al., 1993; Jacobs et al., 1997; McNaughton et al., 1998; Prevost et al., 1997; Vandenberghe et al., 1997]. In general, enhanced performance was found in repetitive, high-intensity, short-term exercise tasks. Furthermore, creatine was successfully used also in patients with chronic heart failure, mitochondrial cytopathies and neuromuscular disease [Gordon et al., 1995; Tarnopolsky et al., 1997; Tarnopolsky and Martin, 1999]. In spinal cord injured (SCI) persons, there exists only one study [Jacobs et al., 2002] that shows a beneficial effect of creatine supplementation on exercise performance in a group of 16 untrained tetraplegic subjects. In this study, subjects significantly increased peak power output in an incremental peak arm ergometry test by 6.7% and maximal oxygen uptake by 17.4% [Jacobs et al., 2002]. So far, to our knowledge, no study investigated the influence of oral creatine supplementation on exercise performance in competitive wheelchair athletes. Moreover, scientific data on single bout exercise performance under sport specific competition-like conditions are limited in highly trained athletes [Mujika and Padilla, 1997].

Independent of this fact, some wheelchair athletes regularly ingest creatine expecting an increased exercise performance during competitions. Since arm muscles contain more type II fibres than leg muscles and since type II fibres have initially a higher phosphocreatine content than type I fibres [Edstrom et al., 1982], it could be speculated that creatine supplementation would be less efficient for arm exercise. Thus, the aim of the present study was to investigate the influence of a short-term oral creatine supplementation on 800m wheelchair performance in competitive SCI athletes.

Methods

Subjects

In total, six healthy, non-smoking, trained wheelchair racers (four male, two female subjects) participated in the study. Their anthropometric data as well as impairment and training information are shown in Table 1.
All subjects were familiar with exercise testing procedures and the equipment used. Five out of six athletes reported that they have never supplemented any creatine so far. The sixth subject confirmed not to have ingested creatine for at least 6 months preceding the study. The study was approved by the local ethical committee. Written informed consent of the subjects was obtained prior to the start of the study.

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age</th>
<th>Height</th>
<th>Weight</th>
<th>Lesion</th>
<th>ASIA / impairment</th>
<th>Impairment since [years]</th>
<th>Training volume [h/week]</th>
<th>Training since [years]</th>
</tr>
</thead>
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<td>m</td>
<td>23</td>
<td>170</td>
<td>68.9</td>
<td>L 1</td>
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<td>23</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>m</td>
<td>39</td>
<td>173</td>
<td>61.1</td>
<td>Th 4</td>
<td>A</td>
<td>17</td>
<td>10</td>
<td>15</td>
</tr>
<tr>
<td>m</td>
<td>45</td>
<td>180</td>
<td>61.2</td>
<td>Th 5</td>
<td>A</td>
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</tr>
<tr>
<td>m</td>
<td>36</td>
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<td>spina bifida</td>
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<td>9</td>
<td>5</td>
</tr>
<tr>
<td>f</td>
<td>33</td>
<td>162</td>
<td>54.9</td>
<td>Th12</td>
<td>A</td>
<td>28</td>
<td>10</td>
<td>11</td>
</tr>
<tr>
<td>f</td>
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<td>-</td>
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<td>6</td>
<td>2</td>
</tr>
<tr>
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<td>171.5</td>
<td>63.1</td>
<td></td>
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<td>22.0</td>
<td>10.0</td>
<td>10.5</td>
</tr>
<tr>
<td>SD</td>
<td>9.1</td>
<td>7.7</td>
<td>6.2</td>
<td></td>
<td></td>
<td>11.2</td>
<td>3.7</td>
<td>7.2</td>
</tr>
</tbody>
</table>

Study design
A double-blind, placebo-controlled, crossover study was performed. Therefore, subjects were randomly assigned into two groups A and B. The study protocol consisted of two treatment phases lasting for 6 days, separated by a washout period of at least 28 days, which was found to be adequate to allow serum creatine levels to return to baseline [Febbraio et al., 1995; Hultman et al., 1996; McKenna et al., 1999]. Before and after each treatment phase, subjects had to perform an exercise test as described in detail below.

Group A received creatine monohydrate (4x5g per day) during the first and placebo (maltodextrin; 4x5g per day) during the second treatment phase. Subjects in group B were supplemented conversely with placebo during the first and creatine during the second treatment period. Both supplements were similar in colour and texture, so that subjects were not able to identify which supplement they ingested.

Subjects were asked to perform no strenuous exercise the day before a test and to abstain from caffeine intake on the day of the test as well as during the 2 weeks of supplementation during the study. Additionally, subjects were instructed to follow their habitual dietary regimen during the study. Training over the period of the study as well as nutrition of the day before and on the test days was held constant and recorded.
Equipment
All tests were performed on a free wheeling trainer (Spinner, New Halls Wheels, Cambridge, USA). Distance covered as well as top and average speed was measured by a speedometer (CicloMaster, CM 209, KW Hochschorner GmbH, Krailling, Deutschland), which was calibrated and mounted on the training roller. Heart rate was recorded by a heart rate monitor (Polar Vantage NV, Polar Electro, Kempele, Finland) and rate of perceived exertion (RPE) was determined by a Borg scale ranging from 6 to 20 [Borg, 1982]. Blood lactate concentration was analysed enzymatically (Super GL Ambulance, Ruhrtal Labor Technik, Möhnesee, Germany).

Experimental procedure
Before each test session, body weight of the subjects was determined. The test session started with a warm-up period of 10min at a predetermined velocity corresponding to 65% of the velocity of the personal best time over the distance of 800m. After 4 and 6min of the warm-up session, subjects had to start sprinting and to hold top speed for 10s and subsequently continued at the predetermined velocity. This regimen was chosen to simulate the warm-up before a competition.

The warm-up period was followed by a 10min rest. Thereafter, subjects had to complete 800m as fast as possible. Verbal encouragement was given and subjects were informed about the distance completed every 100m. Completion of the 800m distance was followed by a 6min resting period.

Heart rate was measured from the beginning of the warm-up period to the end of the test. Lactate was sampled before and after warm-up, before and after completion of the 800m distance, as well as 2, 4 and 6min post exercise. RPE was asked before and after the warm-up and before and at the end of the 800m distance.

Statistics
Results are given as means±SD. A two-way ANOVA (analysis of variance) for repeated measures was used to assess differences in measured parameters. Values were considered to be significantly different if P<0.05.

Results
Creatine supplementation showed no influence on 800m all-out wheelchair performance compared to placebo (Figure 1).

Further, no differences were found between creatine and placebo intake during 6 days for body weight, RPE, peak heart rate, mean heart rate, maximal velocity and lactate concentrations (Table 2) during the 800m exercise test.

During the warm-up periods preceding the 800m exercise tests, no significant differences were found for all measured data.
Figure 1: Time to complete an all-out 800m wheelchair exercise test before and after a short-term creatine and placebo supplementation. Note that there were no significant differences between tests.

Table 2: Parameters measured before and after creatine as well as placebo supplementation of an all-out 800m wheelchair racing exercise test

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Creatine supplementation</th>
<th>Placebo supplementation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>pre</td>
<td>post</td>
</tr>
<tr>
<td>Body weight [kg]</td>
<td>63.1 ± 6.2</td>
<td>63.1 ± 6.2</td>
</tr>
<tr>
<td>RPE</td>
<td>18.5 ± 1.9</td>
<td>18.2 ± 1.6</td>
</tr>
<tr>
<td>Lactate [mmol·l⁻¹]</td>
<td>7.07 ± 1.82</td>
<td>7.21 ± 2.42</td>
</tr>
<tr>
<td>Peak HR [bpm]</td>
<td>181.3 ± 5.6</td>
<td>180.5 ± 6.2</td>
</tr>
<tr>
<td>Max. velocity [km·h⁻¹]</td>
<td>31.8 ± 4.4</td>
<td>32.0 ± 3.2</td>
</tr>
</tbody>
</table>

RPE: rate of perceived exertion; HR: heart rate
Discussion

The main finding of the study was that a short-term creatine supplementation did not enhance 800m wheelchair racing performance compared to placebo (Figure 1). Furthermore, all other parameters measured, for example, body weight, lactate concentrations, heart rate and RPE were not different between the creatine and placebo treatment (Table 2).

Although the creatine kinase system is not the principal energy supplier during an 800m wheelchair race, an increased level of phosphocreatine may possibly diminish lactic acid formation [Williams and Branch, 1998] and therefore enhance performance. This provides some evidence for the use of creatine supplementation for exercise bouts lasting longer than 30s. In fact, a significant increase of 8.5% of time to exhaustion from 130 to 141s at a workload corresponding to 125% of maximal oxygen uptake during cycle ergometry was demonstrated [Jacobs et al., 1997], and Prevost et al. [1997] found a 23.5% increase of total work time during cycling at 150% peak oxygen uptake. Further, creatine supplementation enhanced exercise performance in elite kayak paddlers during time trials between 90 and 300s duration [McNaughton, 1998]. As during the propulsion of a wheelchair, less but similar muscle groups are involved as during kayaking, a positive ergogenic effect of a creatine supplementation was also expected during an all-out 800m wheelchair test, a hypothesis that was not confirmed by our study. Possibly, the differences in total muscles mass involved during kayaking compared to wheelchair racing seemed to be of higher impact than expected and might be responsible for the reported discrepancies concerning an ergogenic effect. Moreover, lactate concentrations were not influenced by creatine supplementation in the present study, which supports the hypothesis that the glycolytic pathways were unaltered by creatine supplementation as proposed by Birch et al. [1994].

Many studies in the past reported positive effects on performance of different types and durations after a short-term creatine supplementation [Balsom et al., 1995; Birch et al., 1994; Bosco et al., 1997; Casey et al., 1996; Greenhaff et al., 1993; Jacobs et al., 1997; McNaughton et al., 1998; Prevost et al., 1997; Volek et al., 1997], whereas others found no effect [Febbraio et al., 1995; Burke et al., 1996; Mujika et al., 1996; Odland et al., 1997; Snow et al., 1998; Terrilllon et al., 1997]. Interestingly, in most of the studies showing no ergogenic effect on exercise performance after creatine supplementation, a time or distance trial was performed [Burke et al., 1996; Mujika et al., 1996; Odland et al., 1997; Snow et al., 1998; Terrilllon et al., 1997], whereas in studies with a positive effect the time to exhaustion at a given workload was determined [Bosco et al., 1997; Prevost et al., 1997; Vandenberghhe et al., 1997; Volek et al., 1997]. This type of exercise test depends mainly on metabolism and remaining energy stores, whereas during time trials higher movement frequencies are required to improve test results. This is also a neuromuscular problem that not only depends on energy sources. Hence, also during our 800m wheelchair test, neuromuscular coordination possibly was one of the limiting factors of exercise performance and might explain that in the present study performance did not improve due to creatine supplementation. Nevertheless, one could speculate that athletes indirectly benefit from creatine supplementation during intense interval training sessions using repeated bouts of shorter distances with limited recovery durations, which may lead to an enhanced performance during competitions. Further investigations are needed to prove this hypothesis.

The lack of beneficial effects of creatine ingestion on exercise performance in the present study may also have further reasons. Most studies investigated the influence of creatine
supplementation on leg muscle performance [Balsom et al., 1995; Birch et al., 1994; Bosco et al., 1997; Casey et al., 1996; Febbraio et al., 1995; Greenhaff et al., 1993; Jacobs et al., 1997; Odland et al., 1997; Prevost et al., 1997; Snow et al., 1998; Terrillion et al., 1997; Vandenberghe et al., 1997]. Since arm muscles contain more type II fibres than leg muscles and since type II fibres have initially a higher phosphocreatine content than type I fibres [Edstrom et al., 1982], it could be hypothesised that creatine supplementation would be less efficient for arm exercise, an assumption that is supported by our findings. However, this hypothesis has to be investigated in further studies, as one cannot conclude definitely if an initially higher phosphocreatine content of arm muscles limits the effectiveness of a creatine supplementation in this muscle group.

Further, a methodological bias in the oral supplementation design is possible. There is some evidence that this assumption can be discarded, as the present type of supplementation programme has previously been successful in other studies [Casey et al., 1996; Harris et al., 1992; Jacobs et al., 2002]. In the study of Jacobs et al. [2002], an increased peak power and maximal oxygen uptake in an incremental peak arm ergometry test after short-term creatine supplementation was even found in SCI patients. As subjects (tetraplegic patients versus top class wheelchair athletes) and exercise testing (incremental test on an arm ergometer versus 800m all-out test in a racing wheelchair) completely differ from the present study, results are difficult to compare. It has also to be taken into account that SCI subjects often suffer from malnutrition [Lee et al., 1985] and therefore possibly benefit from creatine supplementation. In contrast, athletes usually pay attention to their diet, being aware of the positive effects of nutrition on exercise performance. This might explain the different results between the two studies investigating SCI subjects.

Test preparation and warm-up procedure may influence results of a subsequent exercise test. Hence, warm-up in the present study was standardised and no significant differences were found between different test days concerning all measured parameters. It was also demonstrated by Vandenberghe et al. [1996] that caffeine intake counteracts the ergogenic effect of muscle creatine loading. In order to avoid this negative effect in the present study, subjects had to abstain from caffeine intake in any form on test days as well as during the 2 weeks of supplementation. We are therefore confident that caffeine intake is not responsible for the lack of a beneficial effect on 800m wheelchair performance.

Body mass increases up to 2kg were reported after short-term creatine supplementation [Balsom et al., 1995; McNaughton et al., 1998; Muijika et al., 1996; Kraemer and Volek, 1999]. Interestingly, one study with women only found a small, non-significant increase in body mass compared to placebo [Vandenberghe et al., 1997]. This could possibly be due to their smaller muscle mass. This assumption is supported by other investigations in older men, where only a small or no increase in body mass was reported after creatine supplementation [Rawson and Clarkson, 2000; Rawson et al., 1999].

A SCI leads to a muscle atrophy particularly in the paralysed limbs but also in the lower trunk, depending on lesion level. Thus, the decreased total muscle mass of SCI compared to able-bodied athletes possibly impedes weight gain after creatine supplementation due to lower net creatine retention in total.

Finally, concerning exercise performance, the placebo effect may be an important phenomenon [Clark et al., 2000]. In our study, the mean improvement in 800m exercise
performance was 2.3s after creatine supplementation and 2.1s after placebo treatment, which suggests that also in the present study a placebo effect may not be excluded.

**Conclusions**

The present study suggests that a short-term creatine supplementation seems not to enhance 800m wheelchair performance in trained SCI athletes.
A spinal cord injury (SCI) results in an impairment or loss of motor, sensory and vegetative functions leading to unique changes in metabolic, cardiorespiratory, neuromuscular and thermoregulatory systems [Bhambhani, 2002]. As a consequence, overall physical capacity in individuals with SCI is reduced compared to able-bodied persons and highly depends on completeness and level of the lesion. Although the possibilities to exercise for persons with SCI are restricted due to several complications (for more details please refer to Chapter 1) physical activity on a regular base seems to play a key role for this population and reveals a lot of benefits [Mohr et al., 1997a]. These advantages include the prevention from risk factors such as cardiovascular diseases [Franklin et al., 2003], less depressions [Dunn et al., 2005], pain and stress reduction [Ditor et al., 2003] as well as an increased quality of life [Ditor et al., 2003; Stevens et al., 2008]. However, the specific restrictions, limitations and complications related to a SCI need special attention when exercising. The present work focused on the topic "exercise in persons with SCI". The investigations performed aimed to show new possibilities of exercise testing, to implement new training methods (e.g. respiratory muscle training and functional electrical stimulated cycling) into daily clinical practice as well as to optimize exercise performance of patients and athletes with SCI. The following paragraphs will briefly summarize the findings of this thesis and present some future research directions.

Exercise testing in persons with SCI
In order to monitor and guide the training process of patients and athletes with SCI sport specific and feasible exercise training methods are needed taking into account the above mentioned unique physiological requirements of this special population. At the same time valid and reliable measurement equipment is a prerequisite to obtain accurate data. The importance of this statement was exemplarily shown in the present thesis for a new portable ergospirometric device [Perret and Mueller, 2006] and revealed that new equipment should be critically evaluated before it can be used in daily clinical or scientific practice. Moreover, we were able to develop and implement two new exercise testing methods – namely the 5x1500m-test [Mueller et al., 2004] and the heart-rate based lactate minimum test [Perret et al., 2012; Strupler et al., 2009] - which turned out to be feasible and highly reproducible testing methods for the application in wheelchair endurance sports. However, future studies should further develop and optimize exercise testing protocols and tailor them to the special needs of the population with SCI. Finally valid, reliable and sport specific laboratory-based and field testing methods should be developed not only for endurance sports but also for team sports such as wheelchair basketball, wheelchair rugby or wheelchair tennis in the future.

Respiratory muscle training in persons with SCI
Respiratory complications in individuals with SCI are common and still the leading cause of death in this population [Garshick et al., 2005; van den Berg et al., 2010]. Therefore, it seems obvious that one goal of the rehabilitation process in patients with SCI should be to preserve
an optimal functioning of the respiratory pump. A promising approach to reach this aim might be a well-directed respiratory muscle training program. Beside this patient’s perspective also wheelchair athletes might benefit from such a respiratory muscle training regime to enhance exercise performance or to accelerate the recovery process after intense upper body exercise. Keeping this two aspects in mind several investigations were performed on this topic. Mueller and colleagues [2006] were able to determine the optimal training intensities for respiratory endurance training by means of isocapnic hyperpnoea in patients with para- and tetraplegia. Based on this knowledge, further studies should investigate the impact of an isocapnic hyperpnoea training in patients with SCI on respiratory complications such as respiratory tract infections or sleep-disordered breathing, quality of life and physical fitness. Moreover, the detailed mechanisms and effects of isocapnic hyperpnoea training on e.g. cardiac output, lactate metabolism or respiratory mechanics in patients with SCI seem to be still unclear and need further clarification. Additionally, it might be of interest to compare different respiratory muscle training methods to each other (e.g. isocapnic hyperpnoea vs. inspiratory and expiratory resistive loaded breathing) in patients with SCI.

Concerning the application of respiratory muscle training in athletes with SCI a significant increase in 10km time trial performance in wheelchair racing athletes after a six week isocapnic hyperpnoea training period was found [Mueller et al., 2008], whereas blood lactate elimination after exhaustive upper body exercise was not accelerated by this method compared to passive or conventional active recovery strategies [Perret and Mueller, 2007]. The use of another respiratory muscle training method (inspiratory resistive loaded breathing) in wheelchair basketball players revealed no improvements in repetitive sprint performance but showed the potential to enhance lung function and to contribute to a better quality of life even in trained wheelchair users [Goosey-Tolfrey et al., 2010]. This finding might have some interesting implications for the use of inspiratory resistive loaded breathing also in patients with SCI.

In spite of these promising results the ideal timing, duration and frequency as well as the best scheduling of respiratory muscle training interventions into the normal daily physical training activity remains still unclear. Future studies should also address the question how long such advantageous effects of a respiratory muscle training program will last and if there are other potential application fields (e.g. respiratory warm-up before wheelchair competitions) of respiratory muscle training methods.

Functional electrical stimulated cycling in persons with SCI

Regular physical activity plays a key role in reducing health-related risk factors and complications in individuals with SCI [Mohr et al., 1997a]. However, the possibilities for physical activities in these individuals are obviously restricted due to a loss of motor function in the paralysed limbs. Additionally, overuse symptoms of the upper extremities, in particular of the shoulder, are very common [Sie et al., 1992; Silfverskiold and Waters, 1991]. Keeping these facts in mind, it seems to be a good option for persons with SCI to involve the lower extremities in a regular physical activity program. For this purpose, the use of functional electrical stimulated (FES) cycling might offer a promising approach. However, to accurately characterize the effects of a FES cycling program a sensitive test bed is necessary and was developed by Perret et al. [2009] and successfully applied in a 12-month high-volume home-based FES cycling study [Berry et al., 2008].
Summarizing the results of the present thesis, FES cycling seems to be a feasible and promising alternative to upper body exercise for persons with SCI. The benefits of this training modality include high metabolic costs [Hunt et al., 2007; Perret et al., 2010], increased cardiovascular fitness [Berry et al., 2008; Kakebeeke et al., 2008] as well as positive site-specific effects on bone and muscle tissue [Frotzler et al., 2008b; Frotzler et al., 2009]. Whereas the metabolic and cardiovascular adaptations contribute to reduce cardiovascular risk factors, the positive musculoskeletal adaptations have important clinical relevance to decrease the fracture risk in the paralysed limbs of persons suffering from SCI.

Taking the above mentioned promising results into account, future studies should focus on the optimization of muscle stimulation parameters of the paralysed limbs to further increase power output and FES cycling efficiency. Such technical improvements and adaptations seem to play a key role to establish mobile FES cycling systems for recreational outdoor activities in the future and to find ways to make FES cycling accessible to a broader population with SCI. Additionally, the possible implementation and application of any kind of FES interventions into daily life of individuals with SCI should be considered as this might be a good option to increase energy expenditure of this population. This seems to be of importance keeping in mind that the prevalence of obesity in persons with SCI ranges from 40-66% [Anson and Shepherd, 1996; Chen et al., 2006; Liang et al., 2007]. Although physical activity such as FES cycling plays a key role in weight management programs in subjects with SCI nutritional aspects are of utmost importance as well and will be elucidated in the following paragraph.

Nutrition in persons with SCI
Despite the fact, that experts rate nutrition and the nutritional behavior of persons with SCI as a very important issue, research concerning the diet of these subjects has received little attention in the past [Groah et al., 2008]. In fact, the study presented in this thesis by Perret and Stoffel-Kurt [2011] was the first one comparing the nutritional intake between two similar groups with motor complete acute and chronic SCI and revealed several nutritional inadequacies in both groups. These findings underline the need of professional nutritional education for patients with SCI as soon as possible after injury to prevent from nutrition-related secondary complications like cardiovascular diseases. Moreover, several research questions should be addressed in this field in the future. So far, the detailed time course of changes in resting energy expenditure and body composition during the acute phase after traumatic SCI is still unknown and needs clarification. Further, the ideal amount of dietary fibre intake to optimize bowel management and to minimize gastrointestinal complications in persons with SCI is still an important issue worth to investigate in more detail. Finally, the effects of well-directed, controlled nutritional interventions in patients with SCI should be studied in order to reduce nutrition-related complications and risk factors.

Whereas nutrition-related topics in patients with SCI are mainly focussed on health-related factors, wheelchair athletes are increasingly interested in the performance enhancing aspects of dietary interventions such as the ingestion of supplements. However, before using such substances a critical evaluation taking into account the special circumstances of individuals with SCI (e.g. lower resting energy expenditure, increased gastrointestinal transit time and digestion problems) compared to able-bodied persons becomes important and necessary. In the present thesis, the study of Perret et al. [2006] supports this statement, as no performance enhancing effect after a short term creatine supplementation could be found.
in wheelchair athletes, although this substance was proven to be ergogenic in able-bodied subjects in several investigations [e.g. Balsom et al., 1995; Birch et al., 1994; Bosco et al., 1997; Casey et al., 1996; Greenhaff et al., 1993; Jacobs et al., 1997; McNaughton et al., 1998; Prevost et al., 1997; Vandenberghe et al., 1997]. As a consequence, several further investigations dealing with potentially performance enhancing supplements (e.g. caffeine, sodium bicarbonate) are warranted, as data from supplementation studies in able-bodied athletes can’t be directly transferred into wheelchair sports.

Conclusion
Although several limitations and restrictions exist, regular physical activity and a balanced nutrition play key roles in reducing health-related risk factors and complications in persons suffering from SCI. In general, also subjects with SCI are trainable and exercise performance can be determined based on specifically tailored exercise tests presented in the course of this thesis. Additionally, special training methods such as respiratory muscle training or functional electrical stimulated cycling as well as nutritional interventions were applied and adapted to the special needs of individuals with SCI and revealed some promising results and future research directions. In fact, several findings of the present work were already implemented into our daily clinical and sport practice and seem to have the potential to contribute towards a better quality of life in patients with SCI and additionally offer some options to athletes with SCI to compete at their highest performance level possible.


Chen YM, Ho SC, Lam SS, Chan SS. Validity of body mass index and waist circumference in the classification of obesity as compared to percent body fat in Chinese middle-aged women. Int J Obes (Lond) 2006; 30: 918-925.


8 Curriculum vitae

Personal data

Name          Claudio Perret
Date of birth February 2nd, 1969
Place of birth Chur, Switzerland
Native place  Mels, Switzerland
Nationality   Swiss
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Education

1995 - 2000  PhD student in the group of Exercise Physiology, ETH and UZH Zurich, Switzerland
1989 - 1994  Master in Pharmaceutical Sciences, ETH Zurich, Switzerland
1984 - 1988  Gymnasium, Sargans, Switzerland
1982 - 1984  Secondary school, Sargans, Switzerland
1976 - 1982  Primary school, Wangs, Switzerland

Professional Experience

2012 to date  Member of the scientific European Advisory Board of the Coca-Cola founding for “Physical Activity and Disability”, Loughbourough University, UK
2010 to date  Member of the expert group “Sports Nutrition” of the Swiss Olympic Association
2009 to date  Lecturer at the ETH Zurich, Switzerland in exercise physiology (main topic: “Spinal cord injury and exercise”)
2007 to date  Deputy leader of the Institute of Sports Medicine / Swiss Olympic Medical Center Nottwil, Switzerland

2002 to date  Advisor and teacher of the courses for Swiss Wheelchair Coaches

2002 to date  Member and scientific consultant of the Medical Committee of the Swiss Paralympic Committee

2002 to date  Member of the expert group “Endurance” of the Swiss Olympic Association

2007  Member of the organising committee of the Annual Congress of the Swiss Sports Medical Society, 25th-26th October 2007 in Nottwil

2006  Exercise testing of the Brasilian elite soccer team (Seleção) as part of the preparation for the FIFA-World Cup 2006 in Germany

2002 to 2006  Research group leader for respiratory and exercise physiology at the Swiss Paraplegic Research, Nottwil, Switzerland

2001 - 2004  Chief Medical Team and Antidoping-Responsible of the Swiss Canoe Federation

2000 - 2002  Pharmacist at the “Landi-Apotheke” Chur, Switzerland

1997-2000  Member of the Ethical Committee of the Institute of Physiology and Pharmacology, University of Zurich, Switzerland

1996-2000  Advisor for courses in exercise physiology of the Swiss Association of Physiotherapy

1995  Manager of a Pharmacy (“Schwanen-Apotheke”), Schübelbach, Switzerland

Awards


Forschungspreis 2012 der Deutschsprachigen Medizinischen Gesellschaft für Paraplegie (DMGP) für die Arbeit: „Frauen sind anders... Männer auch! – Erste Resultate zu Osteoporose bei chronischer Querschnittlähmung“
9 List of publications

9.1 Peer reviewed original articles


9.2 Book chapters and further publications


