



ACTA KINESIOLOGIAE UNIVERSITATIS TARTUENSIS

**VOLUME
5**

ACTA KINESIOLOGIAE
UNIVERSITATIS TARTUENSIS

TARTU
2008

Editor

Toivo Jürimäe
University of Tartu
18 Ülikooli street
50090 Tartu
Estonia

Editorial board

Steven Blair, USA	Dragan Milanovic, Croatia
Carmelo Bosco, Italy	Ants Nurmekivi, Estonia
David Brodie, UK	Pekka Oja, Finland
Albrecht Claessens, Belgium	Jana Parizkova, Czech Rep.
Otto Eiben, Hungary	Johannes Piiper, Germany
Gigliola Gori, Italy	Teet Seene, Estonia
Li Hongzi, China	Wlodzimierz Starosta, Poland
Jaak Jürimäe, Estonia	Bohumil Svoboda, Czech Rep.
Vassilis Klissouras, Greece	Risto Telama, Finland
Jaak Maaros, Estonia	Atko Viru, Estonia

This journal is indexed in: Sport Discus Database

© University of Tartu, 2000

Printed by Tartu University Press
Tiigi 78, Tartu 50410, Estonia
Order No. 733

CONTENTS

Tiidus P. M. Effectiveness of massage and ultrasound therapy in promoting post-exercise muscle repair and recovery	7
Hofmann P., Pokan R., von Duvillard S. P. Heart rate performance curve and heart rate turn point	23
Welde B., Vikander N. O. Effect of training on dry land and on snow: training intensity and aerobic power of male junior cross country skiers	44
Hein V., Kivimets M. The effects of two styles of teaching and teachers qualification on motor skill performance of the cartwheel	67
Medbø J. I., Jebens E., Gramvik P. Rate of lactate production during 10 and 30 s bicycle sprints versus phosphofruktokinase activity	79
Pääsuke M., Ereline J., Gapeyeva H., Sander P. Age-related differences in vertical jumping performance in women	93
Raudsepp L., Päll P. Effects of a health-related physical education classes on physical activity and fitness in elementary schoolchildren	105
Poderys J. Dependence of muscle working capacity on arterial blood flow intensity at onset of calf exercise	119
Mäestu J., Jürimäe J. Anthropometrical and physiological factors of rowing performance: a review	130
Jürimäe J., Jürimäe T., Purge P. Responses of blood hormones to two hours of endurance rowing on single skulls in male competitive rowers	151

Skurvydas A., Mamkus G. Muscle fatigue and post-tetanic potentiation in boys and adult men when performing jumping exercises of maximal intensity 162

Sudi K., Gallistl S., Möller R., Tafelt E., Borkenstein M. H. Gender dependent association between different estimates of body fat distribution and blood pressure in obese children and adolescents 174

Instruction to authors 189

EFFECTIVENESS OF MASSAGE AND ULTRASOUND THERAPY IN PROMOTING POST-EXERCISE MUSCLE REPAIR AND RECOVERY

P. M. Tiidus

Department of Kinesiology and Physical Education
Wilfrid Laurier University, Waterloo, ON Canada

ABSTRACT

Massage and ultrasound are common interventions employed in athletic settings to treat exercise induced muscle damage. Despite their widespread use, there is relatively little empirical data available to support or refute their efficacy in enhancing post-exercise muscle recovery and repair related physiological processes. The evidence currently available is often contradictory and usually not strongly supportive of a major therapeutic effect of either massage or ultrasound treatment on physiological or functional indices of short or longer term recovery from exercise induced muscle damage. Nevertheless, enough positive evidence and potential exist to warrant further investigation into their effects on recovery from exercise induced muscle damage. The potential psychological or relaxation inducing effects particularly of massage also cannot be discounted in the overall post-exercise recovery of athletes. There appears to be relatively stronger evidence for positive physiological effects of massage and ultrasound therapy in healing of tendon injuries than in influencing post-exercise muscle recovery.

Key words: muscle damage, massage, ultrasound, muscle repair, tendon repair, exercise, muscle soreness

INTRODUCTION

The use of manual massage as a therapeutic intervention in muscle recovery following training has long been a popular practice among athletes and those who treat them [3]. Among its reputed sports related therapeutic benefits are, quicker recovery following intense exercise, enhanced healing of skeletal muscles and connective tissues following exercise induced muscle damage, reduction of muscle soreness and swelling, attenuation of post-exercise muscle inflammatory response and promotion of muscle relaxation [4, 23, 43]. The practice of using ultrasound therapy as a means of enhancing muscle healing also has a long history, primarily in clinical settings [33, 35]. While less commonly used to enhance muscle recovery following exercise, ultrasound has routinely been used to treat exercise induced soft tissue injuries including muscle and tendon strains and muscle soreness [34]. This review will examine the surprisingly limited empirical data on the potential of these common treatment modalities in enhancing recovery from exercise induced muscle damage and their potential physiological mechanisms of action.

MODES OF MANUAL MASSAGE AND ULTRASOUND APPLICATION

The three most common forms of manual massage therapy include effleurage, petrissage and tapotement. Effleurage involves muscle stroking along the long axis of the muscle with varying degrees of pressure while petrissage involves rolling or kneading muscle manipulations [22, 28]. Both effleurage and petrissage are alleged to be effective in muscle "restoration" [22, 28]. Tapotement involves a sustained series of light percussive blows to the muscle with relaxed hands and is generally used as a muscle "stimulant" prior to exercise [22].

Therapeutic ultrasound can be applied in either continuous or pulsed modes, usually at frequencies between 1–3 MHz. Muscle

penetration is greater (up to 5 cm) with lower frequencies and more superficial (1–2 cm) with higher frequencies. Ultrasound in the continuous mode is often used for muscle heating effects, based on the dissipation of ultrasound energy as heat as the ultrasound wave travels through tissue [33]. For example, increases in muscle temperature of over 5 C at a depth of 5.0 cm has been reported with 10 minutes of 1.0 MHz of continuous ultrasound treatment at an intensity of 1.5 watts/cm² [9]. Pulsed ultrasound involves periodical interruption of the ultrasound beam (i.e. 1 msec on, 4 msec off = 20% duty cycle) such that non-thermal or mechanical effects of the ultrasound beam on tissue are emphasized and tissue heating minimized. The non-thermal effects of ultrasound generally involve an increase in cellular fluid movement or “streaming”. By continuously moving the ultrasound transducer head the potential muscle damaging effects by ultrasound such as overheating and unstable cavitation can be minimized [33]. Length of treatment time varies among practitioners but usually ranges around ten minutes per application.

MUSCLE DAMAGE AND REPAIR PROCESSES

As suggested by practitioners, manual massage and ultrasound treatment for exercise damaged muscle should accelerate the healing process. This review will focus particularly on their potential to influence recovery from acute novel physical activity or from chronic overtraining induced muscle damage as opposed to clinical muscle tears and strains. This type of damage is often induced in animal and human models by eccentric exercise [31]. If manual massage and ultrasound therapies are to influence muscle recovery following exercise induced damage, their application should affect one or more of the physiological factors associated with exercise induced muscle damage and repair. These include (but are not limited to) muscle swelling, muscle sarcolemma disruption and enzyme leakage, leukocyte infiltration, heat shock protein synthesis, sarcomere disruption and muscle ultra-structural damage, lysoso-

mal enzyme activation, as well as alterations in muscle protein metabolism and calcium homeostasis [5, 15, 31]. In addition, the activation of muscle satellite cells and their influence in new myotube and myoblast formation is critical to ultimate muscle recovery [17]. Some of the more overt physical symptoms of exercise induced muscle damage include delayed onset muscle soreness (DOMS), loss of muscle force generating capacity, and muscle swelling and stiffness [1, 5, 45]. The effects of massage or ultrasound treatment on most of these processes and symptoms of muscle damage and repair have not been extensively researched [43, 45]. In fact there is currently almost no data available on the ability of either massage or ultrasound to directly influence any of these parameters. The following is a summary of what empirical evidence is currently available regarding the influence of massage or ultrasound on post-exercise muscle repair, functional recovery and related parameters.

POTENTIAL EFFECTS OF MASSAGE ON INDICES OF MUSCLE DAMAGE AND RECOVERY

While numerous potential mechanisms of therapeutic action for massage have been proposed, the most likely effects of massage which may influence post-exercise muscle repair include its reputed potential to influence muscle blood flow, reduce edema formation, reduce pain sensation and induce muscle "relaxation" [3, 4].

It has been suggested, but never actually empirically demonstrated, that increased blood flow to muscle following exercise induced injury may have therapeutic effects [3, 33]. Although some early studies suggested that manual massage might be able to influence blood flow to the treated muscles [3] most, but not all, studies have not shown a strong influence of massage on measures of muscle blood flow [43]. Two recent studies employed Doppler ultrasound to determine limb blood flow [37, 47]. These well controlled studies presented strong evidence for a lack of effect of any

of effleurage, petrissage or tapotement massage techniques on arterial or venous blood flow into or out of either large (quadriceps) or small (forearm) muscle groups during or after application of massage in normal humans [37, 47]. Based on these studies and the weight of evidence from most other well controlled previous studies, it seems that any type of manual massage is unlikely to have a significant influence on the degree of blood flow directed into or out of the treated muscle(s) either during or following massage treatment. In fact as Tiidus *et al.* [47] and Shoemaker and Tiidus [37] demonstrated, even very light muscular contractions will have significantly greater effects on arterial and venous muscle blood flows than massage could ever hope to achieve. Hence, if an increase in muscle blood flow following muscle damage is indeed therapeutic (which is by no means certain), then very light muscular contractions rather than massage should be prescribed [37].

Post-exercise muscle inflammatory response and neutrophil/macrophage invasion of muscle is essential to subsequent muscle healing [38, 42, 44]. Inhibition of this process leads to lack of muscle recovery, while an overstimulation potentially leads to inflammation related muscle damage [42, 44]. Very few studies have attempted to examine the potential for massage to affect post-exercise muscle inflammatory responses [39]. Those that have been conducted, have used indirect measurement methods and have found little evidence that massage has any substantial effect on post-exercise muscle inflammatory processes [39, 45].

A common inflammatory related effect of exercise induced muscle damage is post-exercise muscle swelling and edema formation which may peak at 2–3 days post-damage and last for upwards of one week [15]. If massage is able to diminish edema formation, it is conceivable that this might benefit post-damage recovery in some, as yet undefined, way. Because of the difficulties in its measurement, little direct evidence exists to support or refute the claims that massage may be able to influence muscle edema formation or level by improving muscle lymph flow or drainage in humans. There is some indirect evidence that massage may be able to reduce local subcutaneous edema in some clinical conditions [18, 48]. However in humans, this potential effect has never been

measured in relation to muscle [3, 45, 48]. The use of a few animal models have demonstrated some potential for massage to influence muscle lymph flow [48]. However, these results are not conclusive, may be non-physiological and have shown only small benefits to lymph flow, which may be no greater than that achievable by light muscular contractions [4, 12, 48]. Hence, the potential for massage to influence muscle lymph flow following damage in humans and the benefits, if any, of such an influence are currently unknown. Therefore, any claims for beneficial effects of massage on post-damage muscle recovery, based on improved edema clearance, are rooted more in wistful thinking than any empirical evidence gathered to date.

A commonly heard, related myth regarding the benefits of massage therapy are centered on its potential to remove "toxins" or lactic acid from muscle after exercise or muscle damage [26, 43]. Proponents of this myth unwittingly suggest that lactic acid or other unnamed "metabolic toxins" cause muscle damage and muscle soreness following exercise and have to be removed by massage in order to restore or heal the muscle [43, 45]. It has been well established that muscle lactic acid accumulation does not cause post-exercise muscle soreness (DOMS) or result in muscle damage [1, 5, 15, 31, 38]. In addition, several human studies have demonstrated that massage has no influence on post-exercise blood lactate clearance [24, 26] and that light exercise is much more effective in this regard [24]. Since, as previously mentioned, massage has no influence on muscle blood flow, this result is not surprising. Therefore these sorts of suggestions can be totally discounted. Several recent studies have also found little consistent effect of massage on post-exercise muscle soreness sensation (DOMS) [3, 39, 43, 47]. Therefore the potential analgesic effects of massage on DOMS appear to be minimal. However a recent review did suggest that further well controlled research was needed to fully ascertain the potential for massage to influence post-exercise soreness sensation [14].

A ubiquitous characteristic of exercise damaged muscle is a prolonged (4–10 days) depression of ability to generate force [5, 15]. This is likely due to exercise induced structural disruption of

sarcomeres, which recover their integrity during the repair process [15, 31]. Warren *et al.* [49] have suggested that rate of recovery of muscle force in an eccentrically damaged muscle model is one of the most sensitive measures of recovery from muscle damage. Few studies have directly examined the potential for massage to influence post-exercise muscle force recovery in humans. One study [47] which did report on the potential for daily massage therapy to influence post-eccentric exercise return of muscle force over 4 days in human subjects was unable to demonstrate any positive effects of massage on this parameter in either isometric or dynamic isokinetic movements. Rodenburg *et al.* [36] did report some minor effects of the combination of massage in combination with warm up and stretching on post-exercise strength recovery. However, they attributed these benefits to warm up and stretching and discounted massage as a contributing factor [36]. Several studies have also looked at the potential for massage to influence exercise performance and speed recovery from exercise induced muscle fatigue. Although there is some conflicting evidence, the majority of well controlled studies have found little evidence for any beneficial effect of massage treatment either before exercise; in enhancing subsequent performance, or immediately post-exercise in aiding recovery from fatigue [3, 4, 43]. Studies using elite cyclists [10] and boxers [26] have also not substantiated post-exercise massage as an aid to rapid recovery of muscle function from exercise induced fatigue in sports settings.

While not directly related to exercise induced muscle damage, it has often been suggested that massage can eliminate muscle stiffness and "knots" and induce muscle relaxation [3, 48]. Although there is significant anecdotal evidence for such potential benefits, again empirical evidence for this is relatively scarce and difficult to measure. Some evidence for reduced sympathetic nervous activity post-massage is available [4] and two studies have suggested a reduced H-reflex response (a measure of muscle tone) in calf muscle during, but not immediately following massage [30, 41]. Another study [29] found that massage temporarily reduced electromyographic (EMG) activity in back muscles of healthy volunteers. In contrast, six weeks of massage and exercise therapy had only

minor effects on myofascial trigger points (muscle knots) and neck and shoulder pain in subjects with chronic muscle pain [20].

However, several studies have found that massage can induce psychological relaxation, reduce anxiety and have a positive effect on mood states in both athletes and normal individuals [26, 29]. Hence it is possible that while massage may have little effect on the physiological course of post-exercise muscle damage and recovery, it may to some degree influence the psychological response to muscle damage and aid individuals in coping with the discomfort and in perceived recovery. How these types of benefits may aid in overall recovery are not yet fully documented.

POTENTIAL EFFECTS OF ULTRASOUND ON INDICES OF MUSCLE DAMAGE AND RECOVERY

Ultrasound has also been proposed to have physiological effects similar to massage, which could potentially enhance muscle recovery from exercise induced damage including; increased muscle blood flow, reduced muscle edema or swelling, as well as enhanced inflammation leading to enhanced repair and reduced pain sensation [25, 33]. Heating via ultrasound is thought to promote tissue repair, possibly by increasing muscle blood flow, or by promoting inflammation related repair processes [33]. Non-thermal effects of ultrasound have been suggested by some practitioners to be of greater importance than thermal effects on enhancing general tissue repair, possibly by invoking fibroblast proliferation as well as other responses similar to those invoked by heating within tissues [33].

Despite its wide use in treating a variety of soft tissue injuries and damage, experimental data to explain its mechanisms of therapeutic action or physiological effects on tissues is limited and can be contradictory [34]. Studies on a variety of soft tissue injuries on animal models have reported both enhanced healing rates as well as no effect of ultrasound treatments [34].

Little direct evidence is available to assess the ability of ultrasound treatment to influence post-exercise muscle damage and repair. One recent study employed a contusion injury model in rat muscle to assess the ability of ultrasound treatment in influencing post-injury muscle regeneration [35]. This study concluded that while ultrasound treatment may to some extent promote the satellite cell proliferation stage of muscle regeneration it did not have significant effects on the overall manifestations of muscle regeneration [35].

The influence of ultrasound on other mechanisms which can potentially influence post-exercise muscle repair processes are also limited. As with massage, experimental evidence for the effect on ultrasound on muscle blood flow is mixed, with some studies finding no effect while others report small increases with treatment [16, 45]. Theoretically ultrasound may be able to influence tissue blood flow by changes in vascular tone brought on by induction of ion streaming or possibly by increasing local generation of histamine (a known vasodilator) caused by its degranulation of mast cells [16]. Tissue heating by ultrasound may also be capable of increasing tissue blood flow [45]. It is noteworthy that one study which found a small increase in arterial blood flow to the triceps surae muscle applied at 1.0 MHz at 1.0–1.5 W/cm² also found that placebo application of ultrasound treatment with the ultrasound device turned off, could induce a similar small increase in muscle blood flow in most subjects [16]. It appears that if ultrasound can influence muscle blood flow, this effect is relatively small, inconsistent and may in part be due to a placebo effect.

As previously noted, ultrasound treatment of at least 8–10 minutes, particularly in the continuous mode, can significantly elevate muscle temperature in muscle up to depths of 5.0 cm [8, 9]. While increased muscle temperature can theoretically at least enhance healing, little direct evidence is currently available as to its exact mechanisms. In addition, little empirical evidence exists which demonstrates enhanced healing in exercise damaged muscle following heating by whatever means [46]. Thus the potential to enhance post-exercise muscle repair by tissue heating induced via

ultrasound requires further research to define its actual effectiveness.

As also noted earlier, the inflammatory response that follows exercise induced muscle damage is critical for the ultimate healing of the muscle. Yet an over reaction by inflammatory agents (i.e. leukocyte invasion) may also result in further tissue damage [44]. How ultrasound may influence this critical balance is not yet certain. Based primarily on evidence from superficial wound healing, it has been suggested that the application of ultrasound shortly after injury may augment the initial inflammatory response and perhaps thereby speed up healing [11]. Alternatively, Hasson *et al.* [25] have suggested that ultrasound may have the potential to diminish post-muscle damage inflammatory response and hence minimize any inflammation induced tissue damage which may occur during the recovery process. Few studies have directly evaluated the potential for ultrasound to influence soft tissue inflammation. There are no such studies which directly examine the potential for ultrasound treatment to influence post-exercise muscle inflammatory response. However a related study [40], reported that ultrasound treatment did not affect skin temperature (used as a crude marker of inflammation) in ultraviolet radiation damaged skin in normal humans when applied immediately post-injury. The applicability of this finding to exercise induced muscle damage is unknown. Hence the potential for ultrasound treatment to influence post-exercise induced muscle damage related inflammatory response and the effects of this influence on ultimate muscle injury and repair remains untested.

Three studies have examined the potential for ultrasound to influence post-exercise muscle strength recovery and/or soreness sensation. Hasson *et al.* [25] reported that a single pulsed ultrasound treatment immediately following eccentric exercise induced damage to the quadriceps muscles was able to significantly reduce soreness sensation and enhance strength recovery at 24 hours following damaging exercise. In contrast Plaskett *et al.* [32] reported no effect of daily pulsed ultrasound treatments on the time course or degree of quadriceps muscle strength loss or recovery, up to 96 hours after eccentric exercise. The time course and magnitude of

muscle soreness sensation was similarly unaffected by daily ultrasound treatments over this time period [32]. Similarly a second recent study also reported no significant influence of high or low intensity pulsed ultrasound treatment on post-eccentric exercise induced muscle soreness sensation up to 48 hours following the exercise protocol [6]. In contrast to these findings a recent preliminary study found a small but significant acceleration of strength return in post-eccentric exercise damaged muscles in rats that had been treated daily with ultrasound for one week [2]. Thus the few studies concluded to date present conflicting evidence as to the efficacy of ultrasound treatment in influencing overt soreness symptoms and muscle strength indices following exercise induced muscle damage. A recent study on patients with chronic musculo-skeletal related neck pain also found no significant effects of ultrasound treatment on reducing pain over a 6 week period [20]. More work, particularly examining the range of ultrasound treatment modalities, is required before any conclusions can be drawn regarding the ability of ultrasound to influence these overt aspects of muscle damage and repair.

MASSAGE AND ULTRASOUND AFFECT CONNECTIVE TISSUE AND TENDON REPAIR

As well as inducing damage to the muscle itself, intense exercise may also cause damage to inter-muscular connective tissue and to tendons. In contrast to the limited data which show a relative ineffectiveness of ultrasound and massage treatments on post-exercise muscle damage and repair, there is relatively more positive data on the effectiveness on these therapeutic modalities on connective tissue related repair processes [45]. Ultrasound has been documented to effectively treat tendonitis in humans [27] and tendon damage in animals [19]. Improved repair and post-damage strength have been reported in surgically damaged Achilles tendons in animals treated with ultrasound during recovery compared to untreated controls [13]. Ultrasound application has also increased fibroblast prolif-

eration and collagen synthesis in cultured cells derived from rat Achilles tendons [34].

Massage like manipulation known as Augmented Soft Tissue Mobilization (ASTM) has also been reported to enhance healing of tendonitis in a rat model, possibly by increasing fibroblast proliferation [7]. Gehlsen *et al.* [21] found further support for this by demonstrating that ASTM will increase healing rate of artificially damaged rat tendons by increasing fibroblast numbers and mobilization in the damaged tissues. Hence at this time there appears to be more preliminary evidence for positive effects of massage and ultrasound treatment on tendon and connective tissue injuries than on exercise induced muscle damage.

CONCLUSION

Massage and ultrasound are common interventions employed in athletic settings to treat exercise induced muscle damage. Despite their widespread use, there is relatively little empirical data available to support or refute their efficacy in enhancing post-exercise muscle recovery and repair related physiological processes. The evidence currently available is often contradictory and usually not strongly supportive of a major therapeutic effect of either massage or ultrasound treatment on physiological or functional indices of short or longer term recovery from exercise induced muscle damage. Nevertheless, enough positive evidence and potential exist to warrant further investigation into their effects on recovery from exercise induced muscle damage. The potential psychological or relaxation inducing effects particularly of massage also cannot be discounted in the overall post-exercise recovery of athletes. There appears to be relatively stronger evidence for positive physiological effects of massage and ultrasound therapy in healing of tendon injuries than in influencing post-exercise muscle recovery.

REFERENCES

1. **Armstrong R. B.** (1984) Mechanisms of exercise-induced muscle soreness: A brief review. *Med. Sci. Sports Exerc.* 16: 529–538.
2. **Burton H., Karnes J.** (2000) Ultrasound treatment of contraction-induced muscle injury. *Med. Sci. Sports Exerc.* 32: S321 (Abs.).
3. **Cafarelli E., Flint F.** (1992) The role of massage in preparation for and recovery from exercise. *Sports Med.* 14: 1–9.
4. **Callaghan M. J.** (1993) The role of massage in the management of the athlete: A review. *Br. J. Sports Med.* 27: 474–478.
5. **Clarkson P. M., Sayers S.** (1999) Etiology of exercise induced muscle damage. *Can. J. Appl. Physiol.* 24: 234–248.
6. **Craig J., Bradley J., Walsh D., Baxter D., Allen J.** (1999) Delayed onset muscle soreness: Lack of effect of therapeutic ultrasound in humans. *Arch. Phys. Med. Rehabil.* 80: 318–323.
7. **Davidson C., Ganion L., Gehlsen G., Verhoestra B., Roepke J., Sevier T.** (1997) Rat tendon morphologic and functional changes resulting from soft tissue mobilization. *Med. Sci. Sports Exerc.* 29: 313–319.
8. **Draper D., Schulthies S., Sorvisto P., Hautala A. M.** (1995) Temperature changes in deep muscles of humans during ice and ultrasound therapies: An in vivo study. *J. Orthop. Sports Phys. Ther.* 21: 153–157.
9. **Draper D., Sunderland S., Kirkendall D., Ricard M.** (1993) A comparison of temperature rise in human calf muscles following application of underwater and topical gel ultrasound. *J. Orthop. Sports Phys. Ther.* 17: 247–251.
10. **Drews T., Kreider R. B., Drinkard B.** (1991) Effects of post-event massage therapy on repeated ultra-endurance cycling. *Int. J. Sports Med.* 23: 91 (Abs.).
11. **Dyson M.** (1987) Mechanisms involved in therapeutic ultrasound. *Physiotherapy* 73: 116–120.
12. **Elkins E., Herrick J., Grindlay J., Mann, F, DeForest R.** (1963) Effect of various procedures on the flow of lymph. *Arch. Phys. Med. Rehab.* 34: 31–39.
13. **Enwemeka C. S., Rodriquez O., Mendosa S.** (1990) The bio-mechanical effects of low-intensity ultrasound on healing tendons. *Ultrasound Med. Biol.* 16: 801–807.

14. **Ernst E.** (1998) Does post-exercise massage treatment reduce delayed onset muscle soreness? A systematic review. *Br. J. Sports Med.* 32: 212–214.
15. **Evans W., Cannon J.** (1991) Metabolic effects of exercise-induced muscle damage. *Exerc. Sports Sci. Rev.* 19: 99–125.
16. **Fabrizio P., Schmidt J., Clemente F. R., Lankiewicz L., Levine Z.** (1996) Acute effects of therapeutic ultrasound delivered at varying parameters on the blood flow velocity in a muscular distribution artery. *J. Orthop. Sport Phys. Ther.* 24: 294–302.
17. **Faulkner J., Brooks S., Opitck J.** (1993) Injury to skeletal muscle fibers during contractions: Conditions of occurrence and prevention. *Phys. Ther.* 73: 911–921.
18. **Flowers K.** (1988) String wrapping versus massage for reducing digital volume. *Phys. Ther.* 68: 57–59.
19. **Freider S., Weisberg J., Fleming B., Stanek A.** (1988) The therapeutic effect of ultrasound following partial rupture of Achilles tendon in male rats. *J. Orthop. Sport Phys. Ther.* 10: 39–46.
20. **Gam A., Warming S., Larsen L., Jensen B., Hoydalsmo O., Al-lon I., Andersen B., Gotzche N., Petersen M., Mathiesen B.** (1998) Treatment of myofascial trigger-points with ultrasound combined with massage and exercise- a randomized control study. *Pain* 77: 73–79.
21. **Gehlsen G., Ganion L., Helfst R.** (1999) Fibroblast responses to variation in soft tissue mobilization pressure. *Med. Sci. Sports Exerc.* 31: 531–535.
22. **Goats G. C.** (1994) Massage — The scientific basis of an ancient art: Part 1. The techniques. *Br. J. Sports Med.* 28: 149–152.
23. **Goats G. C.** (1994) Massage — The scientific basis of an ancient art: Part 2. Physiological and therapeutic effects. *Br. J. Sports Med.* 28: 153–156.
24. **Gupta S., Goswami A., Sadhukhan A.** (1996) Comparative study of lactate removal in short term massage of extremities, active recovery and passive recovery period after supramaximal exercise sessions. *Int. J. Sports Med.* 17: 106–110.
25. **Hasson S., Mundorf R., Barnes W., Williams J., Fuji M.** (1990) Effect of pulsed ultrasound versus placebo on muscle soreness perception and muscular performance. *Scand. J. Rehab. Med.* 22: 199–205.
26. **Hemmings B., Smith M., Graydon J., Dyson R.** (2000) Effects of massage on physiological restoration, perceived recovery, and repeated sports performance. *Br. J. Sports Med.* 34: 109–115.

27. **Klaiman M., Shrader J., Danoff J., Hicks J., Pesce W., Ferland J.** (1998) Phonophoresis versus ultrasound in the treatment of common musculoskeletal conditions. *Med. Sci. Sports Exerc.* 30: 1349–1355.
28. **Lehn C., Prentice W. E.** (1994) Massage. In: W.E. Prentice (ed). *Therapeutic modalities in sports medicine* (3rd ed.) St. Louis: Mosby-Yearbook. 335–363.
29. **Longworth J. C.** (1982) Psychophysiological effects of slow stroke back massage on normotensive females. *Adv. Nurs. Sci.* 4: 44–61.
30. **Morelli M., Seabourne D., Sullivan J.** (1990) Changes in H-reflex amplitude during massage of triceps surae in healthy subjects. *J. Orthop. Sports Phys. Ther.* 12: 55–59.
31. **Morgan D. L., Allen D. G.** (1999) Early events in stretch-induced muscle damage. *J. Appl. Physiol.* 87: 2007–2015.
32. **Plaskett C., Tiidus P. M., Livingston L.** (1999) Ultrasound treatment does not affect post-exercise muscle strength recovery or soreness. *J. Sports Rehab.* 8: 1–9.
33. **Prentice W. E.** (1994) Therapeutic ultrasound. In: W.E. Prentice (ed). *Therapeutic modalities in sports medicine* (3rd ed.) St. Louis: Mosby-Yearbook. 255–287.
34. **Ramirez A., Schwane J. A., McFarland C., Starcher B.** (1997) The effect of ultrasound on collagen synthesis and fibroblast proliferation in vitro. *Med. Sci. Sports Exerc.* 29: 326–332.
35. **Rantanen J., Thorsson O., Wollmer P., Hurme T., Kalimo H.** (1999) Effects of therapeutic ultrasound on the regeneration of skeletal myofibers after experimental muscle injury. *Am. J. Sports Med.* 27: 54–59.
36. **Rodenburg J. B., Steenbeck D., Schiereck P., Bär P.** (1994) Warm-up stretching and massage diminish harmful effects of eccentric exercise. *Int. J. Sports Med.* 15: 414–419.
37. **Shoemaker J. K., Tiidus P. M., Mader R.** (1997) Failure of manual massage to alter limb blood flow: Measures by Doppler ultrasound. *Med. Sci. Sports Exerc.* 29: 610–614.
38. **Smith L. L.** (1991) Acute inflammation: The underlying mechanism in delayed onset muscle soreness. *Med. Sci. Sports Exerc.* 23: 542–551.
39. **Smith L. L., Keating M. N., Holbert D., Spratt D., McCammon M., Smith S., Israel R.** (1994) The effects of athletic massage on delayed onset muscle soreness, creatine kinase and neutrophil count: a preliminary report. *J. Orthop Sports Phys. Ther.* 19: 93–99.

40. **Snow C. J., Johnson K. A.** (1988) Effect of therapeutic ultrasound on acute inflammation. *Physiother. Can.* 40: 162–167.
41. **Sullivan J., Williams L., Seaborne D.** (1991) of massage on alpha-motor neuron excitability. *Phys. Ther.* 71: 555–560.
42. **Tidball J. G.** (1995) Inflammatory cell response to acute muscle injury. *Med. Sci. Sports Exerc.* 27: 1022–1032.
43. **Tiidus P. M.** (1997) Manual massage and recovery of muscle function following exercise: A literature review. *J. Orthop. Sports Phys. Ther.* 25: 107–112.
44. **Tiidus P. M.** (1998) Radical species in inflammation and overtraining. *Can. J. Physiol. Pharmacol.* 76: 533–538.
45. **Tiidus P. M.** (1999) Massage and ultrasound as therapeutic modalities in exercise-induced muscle damage. *Can. J. Appl. Physiol.* 24: 267–278.
46. **Tiidus P. M.** (1999) Letter to the Editor. *Can. J. Appl. Physiol.* 24: vi–viii.
47. **Tiidus P. M., Shoemaker J. K.** (1995) Effleurage massage, muscle blood flow and long term post-exercise strength recovery. *Int. J. Sports Med.* 16: 478–483.
48. **Vickers A.** (1996) *Massage and aromatherapy; a guide for health professionals.* London, Chapman-Hall. 74–110.
49. **Warren G., Lowe D., Armstrong R. B.** (1999) Measurement tools used in the study of eccentric contraction-induced injury. *Sports Med.* 27: 43–59.

Correspondence to:

Peter M. Tiidus,
Department of Kinesiology & Physical Education
Wilfrid Laurier University,
Waterloo ON, N2L 3C5, Canada

HEART RATE PERFORMANCE CURVE AND HEART RATE TURN POINT

P. Hofmann^{*}, R. Pokan^{}, S. P. von Duvillard^{***}**

^{*}Institute of Sports Sciences, Karl-Franzens-University Graz

^{**}Institute of Sport Science, University Vienna, Austria

^{***}Department of PEXS, University of North Dakota, Grand Forks, USA

ABSTRACT

In 1982 a simple method for the non invasive determination of the anaerobic threshold by means of heart rate curve analysis has been presented. The deflection of the heart rate performance curve (HRPC) near maximal performance was suggested to give a valid measure of the lactate threshold. However, several investigators failed to repeat these results and raised critical questions about the validity, objectivity and reliability of the concept termed the "Conconi-test". Although a great number of investigations was presented in the last years almost none of these authors investigated the origin of the heart rate deflection phenomenon itself.

Aim of our work group was to objectively describe the time course of the HRPC during incremental exercise, to prove the validity, objectivity and reliability of the threshold concept and moreover to investigate possible physiological causes of the heart rate deflection phenomenon. Several papers will be presented in an overview of the last 15 years.

Key words: Conconi-test, heart rate deflection, lactate turn point, maximal lactate steady state

INTRODUCTION

In 1982 Conconi *et al.* [16] presented for the first time a noninvasive method to determine the “anaerobic threshold” by means of heart rate performance curve (HRPC) analysis only. These authors used the known effect that during incremental exercise the heart rate (HR) usually shows an s-shape [10] and they defined the running speed at the deflection of the HR near maximal load as the “velocity deflection” (vd). Conconi *et al.* [16] presented a significant relationship between vd and the lactate anaerobic threshold. Figure 1 shows the principle of the determination of the HR deflection.

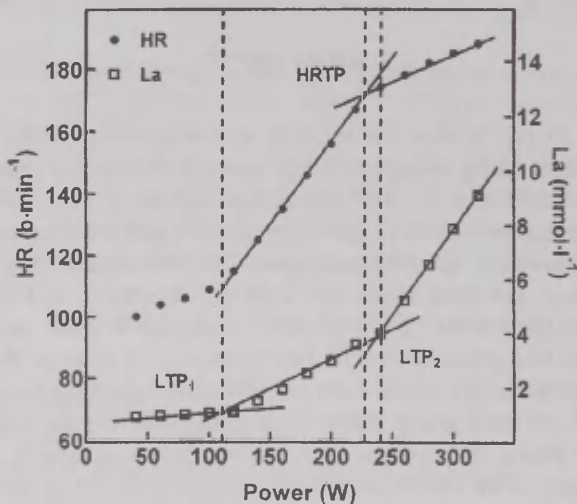


Figure 1. Principle of the determination of the heart rate turn point (HRTP) and the first (LTP₁) and the second (LTP₂) lactate turn point by means of linear regression break point analysis.

This method has been modified by the working group of Conconi for various groups of subjects and sports activities such as for children [1], sprinters and endurance trained runners [7, 16], swimmers [13], rowers [22] and other sports activities such as canoeing, cross country skiing, cycling, roller and iceskating and walking [17, 18,

23]. Because this test is very simple to perform it is very popular in training practice [9, 14, 31, 34, 35, 58, 61, 62, 64, 82, 83] and it has been modified for several applications like for children and untrained subjects [2, 28, 77], patients [37, 73, 77] and older subjects [75, 77], white water kayakers [34, 35], marathon runners [31, 58], race walking [64], and laboratory conditions such as cycle ergometer [30, 32, 36, 39, 41], treadmill [59, 60], rowing ergometer [9, 22] and crank ergometer [54].

The method was supported by many authors [4, 6, 11, 12, 14, 30, 32, 34, 38, 39, 59, 60, 61, 62, 67, 76, 77, 82] but was also criticized and rejected by a great number of investigators [9, 15, 27, 29, 50, 51, 54, 55, 63, 79, 80, 81]. The main point of criticism was that a deflection of the HRPC necessary to detect a "threshold" could not be found in a certain number of subjects. Heck *et al.* [29] presented a review of the literature showing 7.1 to 100% of HRPC's with no detectable heart rate deflection point. Methodological differences for the determination of the deflection point may explain this widespread of outcomes. In part there was a very low correlation found between the heart rate deflection point later called heart rate turn point (HRTP) or heart rate threshold (HRT) and other methods to determine the anaerobic threshold [15, 81]. In a review of about 400 papers dealing with the Conconi method we could not find any paper investigating the phenomenon of heart rate deflection itself although most critical papers discuss the lack of a physiological basis for the heart rate deflection. Only one group presented an attempt using a model simulation to describe physiological causes for the phenomenon but did not support their assumptions by measured data [65].

Most questions regarding the method in the literature were about physiological explanations of the phenomenon. Additionally, low objectivity and reproducibility of the method were mentioned as reasons to reject the method [15, 27, 29, 50, 51, 55, 76, 81] which was in contrast to the high reproducibility presented by the working group of Conconi *et al.* [3, 16, 18]. The methodology itself has been discussed critically [2, 18, 19, 20, 40, 49, 74].

Aim of our work group was to objectively describe the time course of the HRPC during incremental exercise, to investigate

possible physiological causes of the heart rate deflection and to prove the practical application in exercise training.

METHODS

To objectively describe the HRPC we developed in a first step a computer program (PA7000) for HR data analysis [33, 39, 56, 57]. The direction and the degree of the HRPC which is the basis for the determination of a heart rate threshold according to Conconi *et al.* [16] was calculated by means of software PA7000 [57]. Due to the s-shaped response of the HRPC it was necessary to divide the HRPC into three phases to give a valid and objective quantification of the deflection of the HRPC. Three phases of energy supply were determined by means of the lactate performance curve analysis [78] using two lactate turn points [21]. The first lactate turn point (LTP_1) was defined as the first sustained increase of blood lactate concentration (La) above base level values; the second lactate turn point (LTP_2) was defined as the second abrupt increase of La between LTP_1 and maximal performance (P_{max}). Both LTP_1 and LTP_2 were calculated by means of linear regression break point analysis [30, 39]. Figure 1 shows the principle of the determination of LTP_1 and LTP_2 .

The degree and the direction of the HRPC was calculated exclusively between LTP_1 and P_{max} in all of our investigations. As a measure of the direction of the HRPC a second degree polynomial was fitted into the HR curve and classified as regular (deflection of the HRPC according to Conconi *et al.* [16]), linear (without any deflection) or inverted (up sloping HRPC above LTP_2). To measure the degree and the direction of the deflection two tangents were calculated at LTP_1 and P_{max} , respectively and the difference of angles of both tangents gave a measure of the degree and the direction of the deflection between LTP_1 and P_{max} defined as factor k_{HR} . The principle of the determination is presented in Figure 2 [see also 70].

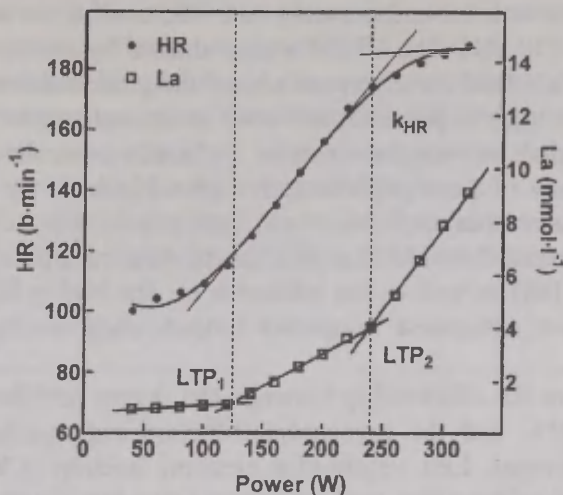


Figure 2. Principle of the determination of the degree and the direction of the deflection of the heart rate performance curve (k_{HR}). Second degree polynomial fitting was applied to the curve between LTP_1 and P_{max} and the differences of angles of the tangents at LTP_1 and P_{max} were calculated and defined as k_{HR} .

Three different HR response groups could be identified [39]:

1. regular HR response as described by Conconi *et al.* [16]: $k_{HR} > 0.1$
2. linear HR response: $0.1 > k_{HR} > -0.1$
3. inverted HR response: $k_{HR} < -0.1$

Figure 3 shows three examples of this different HR response pattern in young healthy male subjects of similar age and performance.

The determination of a heart rate turn point (HRTP) was only performed in subjects with a clear deflection of the HRPC with k_{HR} minimally set at ± 0.2 . The calculation of the deflection point was performed by means of linear regression break point analysis [30, 56, 57]. The principle is presented in Figure 1. This objective description of the HRPC was the basis for several following studies.

The relationship between the HRTP and the maximal lactate steady state (MLSS) important for exercise training regulation was

investigated both under laboratory and field conditions and in various sports [30, 34]. The HRTP was evaluated by means of one or more constant load exercise tests around the predetermined HRTP.

To investigate a possible influence of the autonomous nervous system during incremental exercise a placebo controlled study of the influence of parasympathetic receptor blockade by means of intravenous atropin application was performed [69]. Additionally, the relationship between k_{HR} and the plasma catecholamine concentration [68] as well as the influence of the highly β_1 -selective adrenoceptor antagonist bisoprolol (unpublished results) was investigated.

To prove the relationship between the degree and the direction of the HRPC and the myocardial function radionuclide studies were performed. Left ventricular ejection fraction (LVEF) as a measure of contractility was measured in young healthy subjects [38, 70] in older healthy subjects [37] and in patients after myocardial infarction [37, 73].

As an additional hypothesis the influence of potassium on the HRPC was investigated [42, 45]. Blood pH was suggested to be a possible cause for the different response pattern found [45].

RESULTS AND DISCUSSION

Contrary to the results by Conconi and co-workers [13, 16, 17, 23] we found the HRPC not to present a uniform HR response pattern like described by these authors (Figure 3). Both using a modified protocol on the cycle ergometer ($N=227$) or the originally described field test for runners [16] ($N=293$) we found approximately 16% (cycle ergometer) and 13% (field test) of HRPC's with a different HR response pattern [39]. Independent of the method of testing we found an inverted HRPC in 6–8% in young healthy male [39] subjects (Figure 3). Similar results were found in female subjects ($N=231$) with approximately 15% HRPC's with no regular HR response pattern (unpublished results). This is in contrast to the results presented by Heck *et al.* [29] who reported a wide range

from 7.1 to 100% of HRPC without a detectable heart rate deflection. The reason for this widespread of data may be seen in a different number of subjects of the reviewed investigations and in methodological problems to identify the HRTP [39, 40, 46]. Most of the investigators rely on visual inspection of the HRPC and a subjective determination of the HRTP. None of the investigators tried to validate their visual inspection criteria by means of objective data such as lactate threshold or MLSS measures [see also 30, 40, 41].

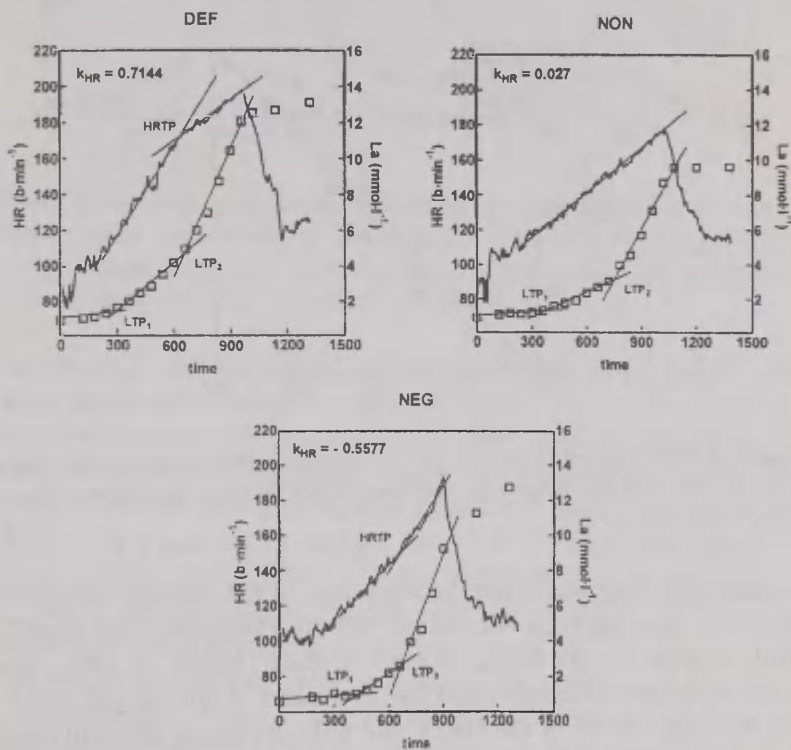


Figure 3. Examples of regular (DEF, $k_{\text{HR}} +$), linear (NON, $k_{\text{HR}} \pm 0$) and inverted (NEG, $k_{\text{HR}} -$) HR response during incremental cycle ergometer exercise in young healthy subjects.

A heart rate deflection point could be detected independent of the direction of the HRPC and HR and power output at the heart rate

turn point were not significantly different from the second lactate turn point and the second ventilatory turn point (VTP₂) defined as the second abrupt increase of pulmonary ventilation between LTP₁ and P_{max} (Figure 4) and they were significantly related [39]. These results supported our earlier findings [12, 38] and that from other work groups [76]. Kara *et al.* [53] supported our findings using a different method of HRTP detection. In older subjects [37, 75] and patients after myocardial infarction [37, 73] a higher percentage of HRPC being linear or inverted was found.

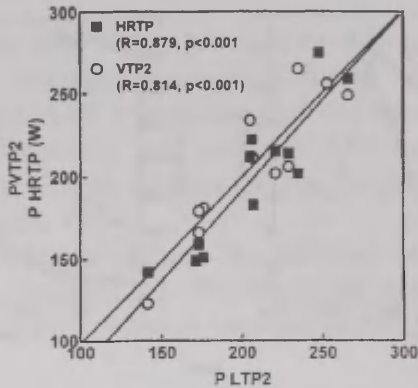


Figure 4. Relationship between power output at the second lactate turn point (LTP₂) and the heart rate turn point (HRTP) and the second ventilatory turn point (VT₂).

Additionally, k_{HR} was found independent of the plasma catecholamine response but both adrenaline and noradrenaline were significantly related to the blood lactate increase (Figure 5) [68]. The parasympathetic receptor blockade by means of atropin just modified the time course of the HRPC but did not change the individual characteristics of the curve [69]. As shown earlier, the parasympathetic receptor blockade increased the resting heart rate [24] but maximal heart rate did not change significantly. The increase of HR at submaximal load steps decreased k_{HR} in subjects with a regular HR deflection and increased k_{HR} in subjects with an inverted HRPC. The typical individual pattern was not changed. The

different HRPC patterns could not be explained by an influence of the autonomous nervous system.

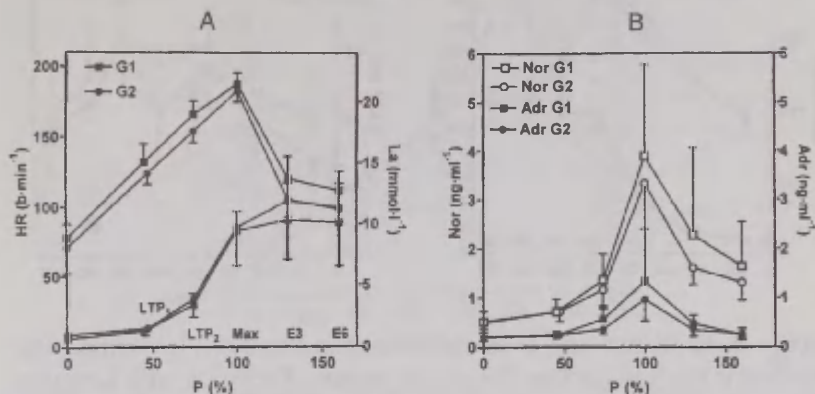


Figure 5. Heart rate and lactate performance curve (A) as well as plasma catecholamine response (B) in two groups of subjects with a regular (G1) or non regular (G2) heart rate response pattern during an incremental cycle ergometer exercise in young healthy male subjects.

The direction and the degree of the deflection of the HRPC was found significantly related to the myocardial function expressed as the left ventricular ejection fraction (LVEF) [38, 70]. The more the HRPC deflection was inverted ($k_{HR} < -0.1$) the more was the decrease of LVEF above the LTP₂ (Figure 6). The decrease of the LVEF was significantly related to the LTP₂ and was found in young healthy subjects [38, 72], in older healthy subjects [38, 70, 72] and in patients after myocardial infarction [37, 72, 73]. Similar results have been presented by Boucher *et al.* [8] and Foster *et al.* [25] for the relationship to the ventilatory threshold.

Additionally, Pokan *et al.* [71] found a significant influence of posture during the test (supine versus upright) on k_{HR} which was discussed also in relationship to the influence of posture on exercise hemodynamics. A change of the duration of steps from one to three minutes had no significant influence on k_{HR} [43]. So from this data it was obvious that myocardial function was related to the HR response pattern but could not explain any cause and effect relationship.

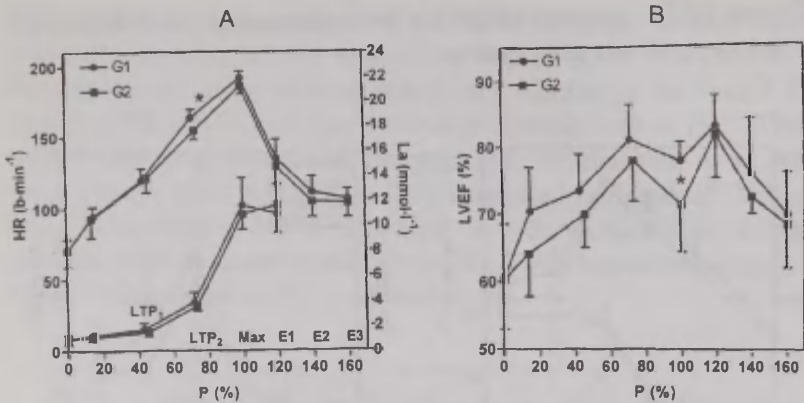


Figure 6. Heart rate and lactate performance curve (A) as well as left ventricular ejection fraction (B) in two groups of subjects with a regular (G1) or non regular (G2) heart rate response pattern during an incremental cycle ergometer exercise in young healthy male subjects.

We suggested a compensatory HR increase above LTP₂ due to a diminished myocardial function. This is an attractive view but fails to explain the lower HR response between LTP₁ and LTP₂ in subjects without a regular HR response pattern especially in light of a similar catecholamine response [68].

As an hypothesis we followed the idea that increased release of potassium (K^+) during heavy muscular work above LTP₂ (Figure 7) could influence indirectly myocardial function. We found a weak but significant relationship between maximal blood potassium concentration and k_{HR} which supported our hypothesis [42, 45] but still failed to explain the lower HR response in subjects without a regular HRPC.

A second hypothesis was a mechanical limitation of myocardial function related to the breathing pattern. A low relationship could be found between k_{HR} and tidal volume (VT) where the higher exercise related VT the lower k_{HR} was found [5]. This relationship supports the idea of a mechanical limitation [47, 48] but fails to explain the phenomenon.

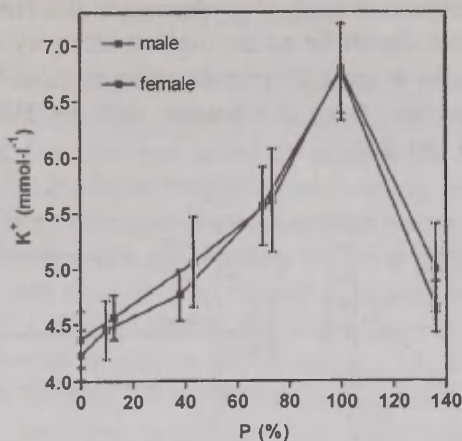


Figure 7. Blood potassium concentration during incremental cycle ergometer exercise in young healthy male and female subjects.

Constant load cycle ergometer exercise just below the heart rate threshold was shown to give a steady state in blood lactate concentration (LaSS) in female [30] and male (unpublished results) subjects. Figure 8 shows both the incremental (A) and the steady state (B) tests at the predetermined H RTP in a well trained white water kayaker on an isokinetic kayak ergometer. It has been described that constant load cycle ergometer exercise at the H RTP gave in some cases an indifferent response with both lactate steady state conditions and increasing La with early termination of the test. This may be explained by methodological errors in the detection of the heart rate deflection point as discussed before as well as by individual day to day changes of performance.

In kayakers [34], badminton players [82], sports students with and without highly β 1-selective adrenoceptor antagonist bisoprolol [unpublished results] and runners (unpublished results) lactate steady state was found in all cases if the subjects were able to keep the given pace and similar performance compared to the incremental test was given. Figure 9 shows results from steady state exercise tests on a cycle ergometer in young healthy male subjects. Our results are in contrast to findings by Heck *et al.* [29], and Krüger *et al.* [54] but we have to question their results as these authors

did not use an objective method to determine the HRTP. If we define the anaerobic threshold as the highest intensity of load a subject can sustain for at least 20 min duration without an increase of La we may conclude from our studies that the HRTP gives the maximal MLSS [30, 82].

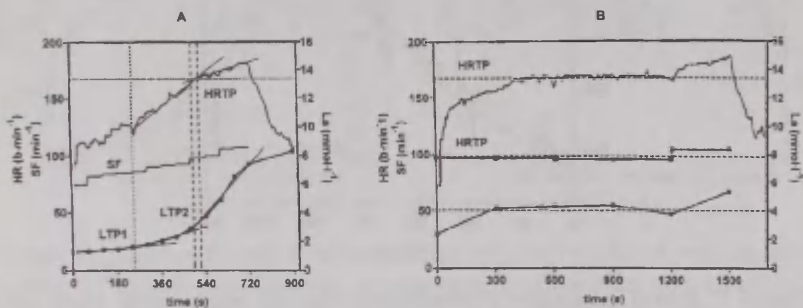


Figure 8. Determination of the heart rate turn point (HRTP) during an incremental exercise test on an isokinetic kayak ergometer in a well trained white water kayaker (A) as well as 20 min of steady state exercise at the predetermined HRTP and 5 min of maximal exercise (B).

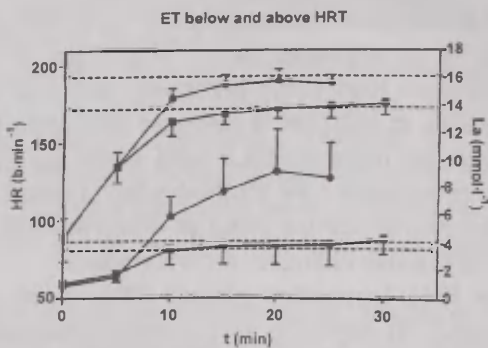


Figure 9. Heart rate and blood lactate concentration during 25 min of steady state cycle ergometer exercise below and above the predetermined heart rate turn point (indicated by dashed lines) in young healthy male subjects.

CONCLUSIONS

The different HR response patterns found during incremental exercise may be explained by a variable load dependent adaptation of the myocardial function but failed to explain the causes for this different interplay between frequency and volume regulation of the healthy heart. The autonomic nervous system seems to play not the major role. Although the relationship between HRTP and muscle metabolism is not clear at the moment a significant relationship between HRTP, LTP_2 and the MLSS can be described. Although in a certain number of subjects a HRTP can not be detected we may recommend the method for the practical application under laboratory and field conditions in combination with lactate steady state tests. Additionally, the analysis of the whole HRPC may offer some additional easy to get information about the myocardial function in heart disease patients [26]. As an alternative hypothesis cardioactive substances related to muscular work such as adenosine [66] or phospholamban [52] as well as a limited mechanical heart-lung interaction [47, 48] may be discussed.

REFERENCES

1. **Ballarin E., Borsetto C., Cellini M., Patracchini M., Vitiello P., Ziglio P. G., Conconi F.** (1989) Adaptation of the "Conconi test" to children and adolescents. *Int. J. Sports Med.* 10: 334-338.
2. **Ballarin E., Borsetto C., Campi S., Casoni I., Grazi G., Guglielmini C., Manfredini F., Mazzoni G., Patracchini M., Conconi F.** (1992) The methodology of the "CONCONI TEST": An update after 10 years. In: *Pediatric Work Physiology*, Coudert J., van Praagh E. (eds.): Masson, Paris-Milan 31-33.
3. **Ballarin E., Sudhues U., Borsetto C., Casoni I., Grazi G., Guglielmini C., Manfredini F., Mazzoni G., Conconi F.** (1996) Reproducibility of the Conconi test: Test repeatability and observer variations. *Int. J. Sports Med.* 17: 520-524.

4. **Baraldi E., Zanconato S., Santuz P. A., Zacchello F.** (1989) A comparison of two noninvasive methods in the determination of the anaerobic threshold in children. *Int. J. Sports Med.* 10: 132–134.
5. **Bisail H., Seibert F.-J., Hofmann P.** (1999) Relationship between heart rate performance curve and breathing pattern. *Österr. J. Sport-med.* 29: 42.
6. **Bodner M. E., Rhodes E. C., Coutts K. D.** (1998) Reliability of a mathematical model to reproduce heart rate threshold and the relationship to ventilatory threshold. *Med. Sci. Sports Exerc.* 30: S310.
7. **Borsetto C., Ballarin E., Casoni I., Cellini M., Vitiello P., Conconi F.** (1989) A field test for determining the speed obtained through anaerobic glycolysis in runners. *Int. J. Sports Med.* 10: 339–345.
8. **Boucher C.A., Kanarek D. J., Okada R. D., Hutter A. M., Strauss H. W., Pohost G. M.** (1983) Exercise testing in aortic regurgitation: comparison of radionuclide left ventricular ejection fraction with exercise performance at the anaerobic threshold and peak exercise. *Am. J. Cardiol.* 52: 801–808.
9. **Bourgeois J., Vrijens J.** (1998) The Conconi test: A controversial concept for the determination of the anaerobic threshold in young rowers. *Int. J. Sports Med.* 19: 553–559.
10. **Brooke J. D., Hamley E. J.** (1972) The heart-rate — physical work curve analysis for the prediction of exhausting work ability. *Med. Sci. Sports Exerc.* 4: 23–26.
11. **Bunc V., Heller J.** (1992) Comparison of two methods of noninvasive anaerobic threshold determination in middle-aged men. *Sports Med. Training & Rehab.* 3: 87–94.
12. **Bunc V., Hofmann P., Leitner H., Gaisl G.** (1995) Verification of the heart rate threshold. *Eur. J. Appl. Physiol.* 70: 263–269.
13. **Cellini M., Vitiello, P., Nagliati A., Ziglio P. G., Martinelli S., Ballarin E., Conconi F.** (1986) Noninvasive determination of the anaerobic threshold in swimming. *Int. J. Sports Med.* 7: 347–351.
14. **Chwilkowski N., Völker K., Hollmann W.** (1988) The validity of the Conconi-test in the determination of the anaerobic threshold in swimming. *Int. J. Sports Med.* 9: 372.
15. **Coen B., Urhausen A., Kindermann W.** (1988) Value of the Conconi test for determination of the anaerobic threshold. *Int. J. Sports Med.* 9: 6.
16. **Conconi F., Ferrari M., Ziglio P. G., Droghetti P., Codeca L.** (1982) Determination of the anaerobic threshold by a noninvasive field test in runners. *J. Appl. Physiol.* 52: 869–873.

17. **Conconi F., Ferrari M., Ziglio P. G., Droghetti P., Borsetto C., Casoni I., Cellini M., Paolini A. R.** (1984) Determination of the anaerobic threshold by a noninvasive field test in running and other sport activities. In: *Current Topics in Sports Medicine*, Bachl N. *et al.* (Eds.): Proc. World Congress, Vienna 1982, Urban & Schwarzenberg, Wien 271–281.
18. **Conconi F., Grazzi G., Casoni I., Guglielmini C., Borsetto C., Ballarin E., Mazzoni G., Patracchini M., Manfredini F.** (1996) The Conconi test: Methodology after 12 years of application. *Int. J. Sports Med.* 17: 509–519.
19. **Conconi F., Grazzi G., Casoni I., Borsetto C., Mazzoni G., Alfieri, N., Manfredini F.** (1997) Reply to: Hofmann P., Pokan R., von Duvillard S. P., Schmid P. Letter to the editor re: Conconi *et al.*: The Conconi Test: Methodology after 12 years of application. *Int. J. Sports Med.* 18: 398–399.
20. **Conconi F., Grazzi G., Casoni I., Borsetto C., Mazzoni G., Alfieri, N., Manfredini F.** (1997) Reply to: Jeukendrup A. E., Hesselink M. K. C., Kuipers H., Keizer H. A.: Letter to the editor re: Conconi *et al.*: The Conconi Test: Methodology after 12 years of application. *Int. J. Sports Med.* 18: 394–396.
21. **Davis H. A., Bassett J., Hughes P., Gass G. C.** (1993) Anaerobic threshold and lactate turn point. *Eur. J. Appl. Physiol.* 50: 383–392.
22. **Droghetti P.** (1986) Determination of the anaerobic threshold on a rowing ergometer by the relationship between work output and heart rate. *Scand. J. Sports Sci.* 8: 59–62.
23. **Droghetti P., Borsetto C., Casoni I., Cellini M., Ferrari M., Paolini A. R., Ziglio P. G., Conconi F.** (1985) Noninvasive determination of the anaerobic threshold in canoeing, cross-country skiing, cycling, roller, and iceskating, rowing, and walking. *Eur. J. Appl. Physiol.* 53: 299–303.
24. **Eklblom B., Kilbom A., Soltysiak J.** (1973) Physical training, bradycardia, and autonomic nervous system. *Scand. J. Clin. Lab. Invest.* 32: 251–256.
25. **Foster C., Gal R. A., Port S. C., Schmidt D. H.** (1995) Left ventricular ejection fraction during incremental and steady state exercise. *Med. Sci. Sports Exerc.* 27: 1602–1606.
26. **Foster C., Spatz P., Georgakopoulos N.** (1999) Left ventricular function in relation to the heart rate performance curve. *Clin. Exerc. Physiol.* 1: 29–32.

27. **Francis K. T., McClatchey P. R., Sumsion J. R., Hansen D. E.** (1989) The relationship between anaerobic threshold and heart rate linearity during cycle ergometry. *Eur. J. Appl. Physiol.* 59: 273–277.
28. **Gaisl G., Hofmann P., Bunc V.** (1991) Standardization of a noninvasive method of determining the anaerobic threshold in children. In: *Children and Exercise. Pediatric Work Physiology XV.* Frenkl R., Szmodis I. (eds.): National Institute for Health Promotion (NEVI), Budapest (Hungary), 234–241.
29. **Heck H., Tiberi M., Beckers K., Lammerschmidt W., Pruin E., Hollmann W.** (1988) Lactic acid concentration during bicycle-ergometer exercise with preselected percentages of the Conconi-threshold. *Int. J. Sports Med.* 9: 367.
30. **Hofmann P., Bunc V., Leitner H., Pokan R., Gaisl G.** (1994) Heart rate threshold related to lactate turn point and steady-state exercise on a cycle ergometer. *Eur. J. Appl. Physiol.* 69: 132–139.
31. **Hofmann P., Gaisl G., Leitner H.** (1994) Comparison of noninvasively determined anaerobic threshold with running results in the marathon race in recreational runners. In: *HPER — Moving Toward the 21st Century.* Duffy P., Dugdale L. (eds.): Human Kinetics, Champaign. IL, 217–225.
32. **Hofmann P., Leitner H., Gaisl G.** (1992) Heart rate threshold, lactate turn point and anaerobic threshold determination by electromyography. *Hung. Rev. Sports Med.* 33: 13–20.
33. **Hofmann P., Leitner H., Gaisl G., Neuhold Ch.** (1990) A computer supported evaluation of the modified Conconi test on bicycle ergometer. In: *A Collection of European Sports Sciences Translations.* Jarver J. (ed.): Kidman Park, S.A., South Australian Sports Institute, Australia, 51–52.
34. **Hofmann P., Peinhaupt G., Leitner H., Pokan R.** (1994) Evaluation of heart rate threshold by means of lactate steady state and endurance tests in white water kayakers. In: *The Way to Win.* Viitasalo J. T., Kujala U. (eds.): Proc. of the Int. Congress on Applied Research in Sports. Helsinki (Finland) 217–220.
35. **Hofmann P., Peinhaupt G., Pokan R., Zweiker R.** (1996) Relationship between treadmill performance and sport specific aerobic performance in white water kayakers. In: *First Annual Congress Frontiers in Sports Science.* Marconnet P., Gaulard J., Margaritis I., Tessier F. (eds.): Nice, France, May 28–31, 664–665.

36. Hofmann P., Niederkofler W., Pokan R., Bunc V. (1996) Comparison between heart rate threshold and individual physical working capacity. *Kinanthropologia* 32: 47–50.
37. Hofmann P., Pokan R., Beaufort F., Schumacher M., Fruhwald F. M., Zweiker R., Eber B., Gasser R., Schmid P., Brandt D., Klein W. (1996) Left ventricular function during incremental cycle ergometer exercise related to aerobic and anaerobic threshold in patients after myocardial infarction, healthy older subjects and young sports students. In: *Sport Kinetics* 95, Chytráková J., Kohoutek M. (eds): Prague, 192–198.
38. Hofmann P., Pokan R., Preidler W., Leitner H., Szolar D., Eber B., Schwabberger G. (1994) Relationship between heart rate threshold, lactate turn point and myocardial function. *Int. J. Sports Med.* 15: 232–237.
39. Hofmann P., Pokan R., von Duvillard S. P., Seibert F.-J., Zweiker R., Schmid P. (1997) The heart rate performance curve during incremental cycle ergometer exercise in healthy young male subjects. *Med. Sci. Sports Exerc.* 29: 762–768.
40. Hofmann P., Pokan R., von Duvillard S. P., Schmid P. (1997) Letter to the editor re: Conconi *et al.*: The Conconi Test: Methodology After 12 Years of Application. *Int. J. Sports Med.* 18: 397–398.
41. Hofmann P., Pokan R., Bachl N., Schmid P. (1997) Determination of the heart rate deflection point. *J. Sports Med. Phys. Fit.* 37: 151–154.
42. Hofmann P., Seibert F. J., Öhlknecht A., Sudi K. M., Pokan R., Schmid P. (1998) Relationship between blood potassium level and the deflection of the heart rate performance curve. *Int. J. Sports Med.* 19: 25.
43. Hofmann P., Pokan R., von Duvillard S. P. (1998) Influence of step length during incremental exercise on the heart rate performance curve. *Med. Sci. Sports Exerc.* 30: S242.
44. Hofmann P., Pokan R., Öhlknecht A., Sudi K. M., Schwabberger G. (1998) Invasive and non-invasive determination of the „Anaerobic threshold”. *J. Sports Med. Phys. Fit.* 38: 88.
45. Hofmann P., Seibert F. J., Pokan R., Golda M., Wallner D., von Duvillard S. P. (1999) Relationship between blood pH, potassium and the heart rate performance curve. *Med. Sci. Sports Exerc.* 31: 150.
46. Hofmann P., Pokan R., von Duvillard S. P. (1999) The Conconi-method: Current state of affairs. In: *Sport Science '99 in Europe*. Pro-

- ceedings of the 4th Annual Congress of the European College of Sport Science, Rome 14–17 July, 125.
47. **Hofmann P., Golda M., Pokan R., Schwabberger G., von Duvillard S. P.** (1999) Influence of cycling cadence and breathing pattern on the heart rate performance curve. In: Sport Science'99 in Europe, Parisi P., Pigozzi F. (eds.): Proceedings of the 4th Annual Congress of the European College of Sport Science, Rome 14–17 July, 816.
 48. **Hsia C. C. W.** (1999) Cardiopulmonary limitations to exercise in restrictive lung disease. *Med. Sci. Sports Exerc.* 31: S28–S32.
 49. **Jeukendrup A. E., Hesselink M. K. C., Kuipers H., Keizer H. A.** (1997) Letter to the editor re: Conconi *et al.*: The Conconi Test: Methodology After 12 Years of Application. *Int. J. Sports Med.* 18: 393–394.
 50. **Jones A. M., Doust J. H.** (1995) Lack of reliability in Conconi's heart rate deflection point. *Int. J. Sports Med.* 16: 541–544.
 51. **Jones A. M., Doust J. H.** (1997) The Conconi test is not valid for the estimation of the lactate turn point in runners. *J. Sports Sci.* 15: 385–394.
 52. **Kalinski M. I., Antipenko A. Y., Dunbar C. C., Michielli D. W.** (1995) Exercise and Intracellular Regulation of Cardiac and Skeletal Muscle. HK Sport Science Monograph Series Vol. 7, Human Kinetics, Champaign, IL.
 53. **Kara M., Gökbel H., Bediz C., Ergene N., Ücok K., Uysal H.** (1996) Determination of the heart rate deflection point by the Dmax method. *J. Sports Med. Phys. Fit.* 36: 31–34.
 54. **Krüger J., Mortier R., Heck H., Hollmann W.** (1988) Relationship between the Conconi-threshold and lactic-acid at endurance workload on the during crank ergometer. *Int. J. Sports Med.* 9: 367.
 55. **Leger L., Tokmakidis S. P.** (1988) Use of the heart rate deflection point to assess the anaerobic threshold. *J. Appl. Physiol.* 64: 1758–1759.
 56. **Leitner H., Hofmann P., Pokan R.** (1994) The application of Fuzzy Logic in the determination of the anaerobic threshold performance diagnostics. *Int. J. Sports Med.* 15: 351.
 57. **Leitner H., Hofmann P., Gaisl G.** (1988) A method for the micro-computer — aided determination of the anaerobic threshold by means of heart rate curve analysis. Conference Proc. 15 years Biomedical Engineering in Austria 88' Graz, June 9–11: 136–141.
 58. **Lenzi G.** (1983) The women's marathon: Preparing for an important event in the season. In: Running the IAAF. Symp. on Middle and

- Long Distance Events. Alford J. W. (ed.): IAAF, London, 1983: 59–66.
59. **Maffulli N., Sjödin B., Ekblom B.** (1987) A laboratory method for non invasive anaerobic threshold determination. *J. Sports Med.* 27: 419–423.
60. **Maffulli N., Testa V., Capasso G.** (1994) Anaerobic threshold determination in master endurance runners. *J. Sports Med. Phys. Fit.* 34: 242–249.
61. **Maffulli N., Capasso G., Lancia A.** (1991) Anaerobic threshold and performance in middle and long distance running. *J. Sports Med. Phys. Fit.* 31: 332–338.
62. **Maffulli N., Testa V., Lancia A., Capasso G., Lombardi S.** (1991) Indices of sustained aerobic power in young middle distance runners. *Med. Sci. Sports Exerc.* 23: 1090–1096.
63. **Mahon A. D., Vaccaro P.** (1991) Can the point of deflection from linearity of heart rate determine ventilatory threshold in children? *Ped. Exerc. Sci.* 3: 256–262.
64. **Marin J.** (1990) Controlling the development of training in race walkers. *New Studies in Athletics* 5: 49–53.
65. **Pessenhofer H., Meier A., Schwaberg G., Sauseng N.** (1991) Verification of hypotheses about the physiological basis of the Conconi test by model simulation. *Int. J. Sports Med.* 12: 119.
66. **Pelleg A., Belardinelli L.** (1999) Effects of Extracellular Adenosine and ATP on Cardiomyocytes. *Molecular Biology Intelligence Unit 6*, R.G. LANDES Company, Austin, Texas.
67. **Petit M. A., Nelson C. A., Rhodes E. C.** (1997) Comparison of a mathematical model to predict 10-km performance from the Conconi test and ventilatory threshold measurements. *Can. J. Appl. Physiol.* 22: 562–572.
68. **Pokan R., Hofmann P., Lehmann M., Leitner H., Eber B., Gasser R., Schwaberg G., Schmid P., Keul J., Klein W.** (1995) Heart rate deflection related to lactate performance curve and plasma catecholamine response during incremental cycle ergometer exercise. *Eur. J. Appl. Physiol.* 70: 175–179.
69. **Pokan R., Hofmann P., von Duvillard S. P., Schumacher M., Gasser R., Zweiker R., Fruhwald W., Eber B., Smekal G., Bachl N., Schmid P.** (1998) Parasympathetic receptor blockade and the heart rate performance curve. *Med. Sci. Sports Exerc.* 30: 229–233.
70. **Pokan R., Hofmann P., Preidler K., Leitner H., Dusleag J., Eber B., Schwaberg G., Fügler G. F., Klein W.** (1993) Correlation be-

tween inflection of heart rate/work performance curve and myocardial function in exhausting cycle ergometer exercise. *Eur. J. Appl. Physiol.* 67: 385–388.

71. Pokan R., Katary U., Hofmann P., Smekal G., Tschan H., Baron R., Bachl N., Schmid P. (1996) Heart rate deflection and heart rate deflection point during upright and supine incremental cycle ergometer exercise. In: First Annual Congress Frontiers in Sports Science. Marconnet P., Gaulard J., Margaritis I., Tessier F. (eds.): Nice, France, May 28–31, 690–691.
72. Pokan R., Hofmann P., von Duvillard S. P., Beaufort F., Schumacher M., Fruhwald F. M., Zweiker R., Eber B., Gasser R., Brandt D., Smekal G., Klein W., Schmid P. (1997) Left ventricular function in response to the transition from aerobic to anaerobic metabolism. *Med. Sci. Sports Exerc.* 29: 1040–1047.
73. Pokan R., Hofmann P., von Duvillard S. P., Beaufort F., Smekal G., Gasser R., Klein W., Eber B., Bachl N., Schmid P. (1998) The heart rate performance curve and left ventricular function during exercise in patients after myocardial infarction. *Med. Sci. Sports Exerc.* 30: 1475–1480.
74. Pokan R., Hofmann P., von Duvillard S. P., Smekal G., Högler R., Tschan H., Baron R., Schmid P., Bachl N. (1999) The heart rate turn point reliability and methodological aspects. *Med. Sci. Sports Exerc.* 31: 903–907.
75. Pokan R., Smekal G., Hofmann P., von Duvillard S. P., Tschan H., Baron R., Schmid P., Bachl N. (1999) Performance diagnostics in aging population. In: Sport Science '99 in Europe. Parisi P., Pigozzi F. (eds.): Proceedings of the 4th Annual Congress of the European College of Sport Science, Rome 14–17 July, 483.
76. Ribeiro J. P., Fielding R. A., Hughes V., Black A., Bochesse M. A., Knuttgen H. G. (1985) Heart rate break point may coincide with the anaerobic and not the aerobic threshold. *Int. J. Sports Med.* 6: 220–224.
77. Rogers R. K. L., Reybrouck T., Weymans M., Dumoulin M., Geuillig M., Vaccaro P. (1995) The relationship between heart rate deflection and ventilatory threshold in children following heart surgery. *Ped. Exerc. Sci.* 7: 263–269.
78. Skinner J. S., McLellan T. (1980) The transition from aerobic to anaerobic metabolism. *Res. Q. Exerc. Sport* 51: 234–248.

79. **Thorland W., Podolin D. A., Mazzeo R. S.** (1994) Coincidence of lactate threshold and HR-power output threshold under varied nutritional states. *Int. J. Sports Med.* 15: 301–304.
80. **Tiberi M., Böhle E., Zimmermann E., Heck H., Hollmann W.** (1988) Comparative examination between Conconi- and lactate threshold on the treadmill by middle distance runners. *Int. J. Sports Med.* 9: 372.
81. **Tokmakidis S. P., Leger L. A.** (1992) Comparison of mathematically determined blood lactate and heart rate “threshold” points and relationship with performance. *Eur. J. Appl. Physiol.* 64: 309–317.
82. **Wonisch M., Hofmann P., Schwabinger G., Pokan R., von Duvillard S. P., Klein W.** (2000) Evaluation of a field test for the noninvasive determination of badminton specific endurance. *Med. Sci. Sports Exerc.* 32: S309
83. **Zacharogiannis E., Farrally M.** (1993) Ventilatory threshold, heart rate deflection point and middle distance running performance. *J. Sports Med. Phys. Fit.* 33: 337–347.

Correspondence to:

Peter Hofmann
Institute of Sports Sciences
KF-University Graz
Mozartgasse 14/1
A-8010 Graz
Austria

EFFECT OF TRAINING ON DRY LAND AND ON SNOW: TRAINING INTENSITY AND AEROBIC POWER OF MALE JUNIOR CROSS COUNTRY SKIERS

B. Welde, N. O. Vikander

North-Troendelag University College, Department of Physical Education,
Roestad, Levanger, Norway

ABSTRACT

The purpose of this study was to examine how intense training may affect the maximal O_2 uptake and lactate threshold of 11 well-trained male junior cross country skiers (18.7 ± 0.2 yr) during a 12 wk period which included training on dry land and on snow. The subjects, who had a maximal O_2 uptake of 71 ± 2 ml \times kg⁻¹ \times min⁻¹ (mean \pm SEM) and a lactate threshold at $75 \pm 1\%$ of the maximal O_2 uptake, were assigned to either a high training-intensity group ($n=5$) or a moderate training-intensity group ($n=6$). Low intensity training was carried out at 40–60% of the maximal O_2 uptake (that is below the subjects' lactate threshold), whereas high intensity training was executed at 70–90% of the maximal O_2 uptake (at or above their lactate threshold). All such training was carried out as running, rollerskiing, or cross country skiing. The subjects' lactate threshold and maximal O_2 uptake during uphill treadmill running were determined before, during, and after the training period. The high training-intensity group trained nearly twice as much at high intensity as the moderate training-intensity group. The maximal O_2 uptake fell for the moderate training-intensity group, whereas it peaked at midpoint test and then returned to the pretest level for the participants in the high training-intensity group. The lactate threshold did not change for either group. Thus, intensive training at or above the lactate threshold may be more effective in

maintaining the maximal O_2 uptake of well-trained skiers with no negative effect on the lactate threshold.

Key words: cross country skiing, intensive training, maximal O_2 uptake, lactate threshold.

INTRODUCTION

A cross country ski-race lasts from many minutes to several hours, placing dominant requirements upon aerobic energy release. Consequently, the ability to take up O_2 at a high rate and for an extended time is perhaps the most central physiologic parameter for cross country skiers. In accordance with this, a number of studies have shown that elite skiers are among the athletes showing the highest values of maximal O_2 uptake [5, 27, 29; for a review, see also 22]. In addition to the maximal O_2 uptake, the "lactate threshold" is considered another highly important physiologic variable for cross country skiers.

Traditionally, cross country skiers have performed mainly long-distance training lasting one hour or more at a moderate intensity, well below the level that corresponds to the lactate threshold. Although the training intensity may be the key factor in raising the maximal O_2 uptake [18, 23, 26, 34], training at higher intensities and, therefore, of shorter duration has been given little attention [3, 12].

Many studies have shown that the blood lactate response to exercise is better correlated with endurance performance than is the maximal O_2 uptake [1, 2, 6, 11, 14, 19, 32]. Moreover, some studies suggest that endurance-training at or above the lactate threshold may be advantageous in raising the exercise intensity level that corresponds to the lactate threshold [1, 17, 19, 30, 33, 35]. Other studies report similar improvements for training below versus above the lactate threshold [4, 24]. Recently Evertsen *et al.*, [9, 10] found that for elite cross country skiers, training at intensities slightly below the lactate threshold was more favorable than training at lower intensities in terms of increasing the running speed at the

lactate threshold, as well as for several other performance parameters. Gaskill *et al.* [13], who manipulated and quantified the training load over an entire competitive season, showed that increased volume of high-intensity training is favorable for skiers with respect to O_2 uptake at lactate threshold, maximal O_2 uptake, and competitive results. However, their training regime was based on intensive training at or above the lactate threshold. These somewhat contradictory findings may be due to lactate threshold being possibly more sensitive to specific training protocols than is the maximal O_2 uptake [24].

Most of the studies of cross country skiers have been carried out during the dry-land training period. Consequently, there is little information on how skiers respond to the transition from dry land to snow, although this is a commonly voiced problem among practitioners. Some data indicate that maximal O_2 uptake plateaus in the transition period [13]. Since it is suggested that the maximal O_2 uptake sets the upper limit for the lactate threshold [6], this stagnation may not be favorable. Although the Gaskill *et al.* study [13] of long-term responses to training involved testing that corresponded with the major changes in training periods, it did not specifically investigate the dynamics of the transition from dry land to snow conditions. Their three-pronged test strategy of mid-summer, mid-December, and late February provided no such information.

There is a dearth of studies examining how more intense training affects maximal O_2 uptake and lactate threshold for well trained skiers in the transition from dry land to snow. The present investigation addressed this deficiency by monitoring two groups of well-prepared junior cross country skiers who trained systematically for 3 mo, including the dry land and on snow periods, using either a moderate or a combined moderate and high training-intensity protocol. It was hypothesized that an increased volume of intensive training in the region of the lactate threshold would be favorable for enhancing maximal O_2 uptake and the blood lactate response to exercise when training on dry land and on snow.

MATERIALS AND METHODS

Subjects

Eleven male juniors, 18.7 ± 0.2 year old (mean \pm SEM), who were students at two Norwegian high-schools with specialized cross country ski-programs, served as subjects. The subjects had been training and competing regularly for at least 3 yr before commencement of the study. The skiers at one school ($n=5$) were assigned to the "high training-intensity group", whereas the skiers at the other school ($n=6$) were designated the "moderate training-intensity group". Although it is desirable from a theoretical point of view to randomize the group assignments without regard to school, it was deemed not practical from field experience. Tight social relations within the teams would have rendered significant training variations problematic. The subjects were 177 ± 2 (high training-intensity group) and 178 ± 2 cm (moderate training-intensity group) tall, weighed 71 ± 3 and 69 ± 2 kg, had a maximal O_2 uptake of 71 ± 2 and 71 ± 3 ml \times kg⁻¹ \times min⁻¹, and a lactate threshold of 74 ± 1 and $76 \pm 1\%$ of their maximal O_2 uptake, respectively. There were no statistically significant differences in any of these parameters between the two groups at recruitment. The subjects were given detailed information both orally and in written form about the purposes, risks, and possible discomforts of the test procedures before they gave their written consent. Subjects were also told that they served as volunteers and thus had the right to withdraw from the study at any stage without giving any reason for doing so.

Tests and measurements

The investigation took place during a 12 wk period from the middle of October to early January. Thus the study comprised a 6 wk dry land training period followed by a 6 wk (mainly) on snow training period. The maximal O_2 uptake and the lactate threshold were determined before, at the transition midpoint, and immediately after the 12 wk training period. All treadmill tests were car-

ried out on a motorized treadmill (Jaeger LE 5000, Eric Jaeger GmbH *et Co.*, Würzburg, Germany) at 6° (10.5%) inclination. The skiers wore a wireless heart rate monitor with digital display (Polar Sports Tester 4000, Polar Electro OY, Kempele, Finland) during all treadmill tests. The subjects were instructed not to exercise in the morning before testing. The day prior to tests they were permitted some slow distance training only. It was stated explicitly to the subjects that they should do the same preparations before every test.

To establish the lactate threshold, a ramp test originally designed for marathon runners [15] was modified by increasing the treadmill inclination from 1° (1.75%) to 6° (10.5%). After a 10 min warm-up at an exercise intensity corresponding to 50% of the maximal O₂ uptake, the subjects ran for 5 min in each of four consecutive tests in the range of 60–90% of their maximal O₂ uptake. The runs were separated by 30 s breaks so that a capillary blood sample could be taken from a finger. The heart rate and the volume of expired air were recorded over the last 2 min of each run. The lactate threshold was taken as the O₂ uptake associated with a blood lactate concentration 1.5 mmol × l⁻¹ above the baseline value [15], and the exact point was determined by linear interpolation between the measured values.

After a 2 h recovery, a second test was carried out to establish the maximal O₂ uptake. During this second test the treadmill speed was gradually increased in steps of 0.15–0.3 m × s⁻¹, to a level that, under the test leader's supervision and in accordance with previous test results, brought the subjects close to exhaustion in about 3 min. During these 3 min the subject wore a nose-clip and mouthpiece connected to the O₂ analyzer (Jaeger EOS-sprint system, Eric Jaeger GmbH *et Co.*, Würzburg, Germany). The analyzer recorded the O₂ consumption and respiratory exchange ratio (R-value) at 30 s intervals. If the O₂ consumption showed no further increase (or increased only slightly) with a further increase in the treadmill speed, and in addition the respiratory exchange ratio was > 1.10 and the heart rate ≥97% of the maximal heart rate, the test was considered successful. Otherwise the run was continued until a level-

ling-off was seen, or to exhaustion. In a previous test, the subjects' maximal heart rate were established by adding 5 beats \times min⁻¹ to the highest stable heart rate during the last min of a maximal O₂ uptake test [20].

Training during the study

Two main training intensities were used during the investigation period. The low intensity training was mainly carried out at 60–75% of the maximal heart rate (corresponding to 40–60% of the maximal O₂ uptake; that is well below the subjects' lactate threshold). The high intensity training was carried out at 85–95% of the maximal heart rate, corresponding to 70–90% of the maximal O₂ uptake. (The upper part of this intensity zone is well above the subjects' lactate threshold). The subjects' heart rate during each training session was recorded, and all the endurance training was classified according to a five-level intensity scale (Table 1). All such training was carried out as running, rollerskiing, or cross country skiing.

Table 1. Training intensity zones used in the study of two groups of Norwegian male junior cross country skiers over a 12 wk period.

Intensity zone	HR as % of HR _{max}	HR ¹	Examples of training methods
1	60–75%	120–150	Active rest and restitution work. Easy distance training.
2	75–85%	150–170	Moderate distance training. Continous work.
3	85–90%	170–180	Fartlek training. Natural/ systematic interval training. Lactate threshold work.
4	90–95%	180–190	Systematic hard interval training. Longer race pace-training.
5	95–100%	190–200	Tempo/speed/explosive training. Maximal short-duration efforts. Shorter race pace-training.

¹ Based on HR_{max} of 200.

For the high training-intensity group 70% of all training was carried out at low intensities, whereas 20% was carried out at high intensities consisting of continuous work (15 min to 40 min) and interval training (systematic intervals of 30 s to 3 min duration). These subjects carried out high-intensity training three times per week. Strength training, speed-training, and explosive training made up 10% of the total training time (Table 2).

Table 2. Amount of training in two intensity groups of Norwegian male junior cross country skiers over a 12 wk period. First 6 weeks = dry land period; second 6 weeks = mixed conditions and on snow.

	Mainly dry-land (6 wk)		Mainly on-snow (6 wk)		Means (12 wk)	
	HTI	MTI	HTI	MTI	HTI	MTI
Low intensity ^a (h wk ⁻¹)	7.50	8.74	5.16	6.49	6.34	7.60
(% of total)	71	81	70	84	71	82
High intensity ^b (h wk ⁻¹)	1.89 ^{\$\$\$}	1.14	1.63 ^{\$}	0.84	1.76 ^{\$\$}	0.98
(% of total)	18	11	22	11	20	11
Strength/speed/ explosive ^c (h wk ⁻¹)	1.13	0.96	0.59	0.42	0.84	0.68
(% of total)	11	9	8	5	10	7
Totals	10.52	10.83	7.38	7.75	8.94	9.28

HTI, High training-intensity group; MTI, Moderate training-intensity group.

^a Low intensity includes intensity zones 1–2.

^b High intensity includes intensity zones 3–4.

^c Strength, speed and explosive training: Speed and explosive training generally do not generate HR_{max}. This form of training appears to have more in common with strength training and, thus, is placed in this combined category.

^{\$} Significant differences between groups, ^{\$}p<0.05, ^{\$\$}p<0.01, ^{\$\$\$}p<0.001.

For the moderate training-intensity group the low intensity training constituted 82% of the total training time, each session lasting between 45 min and 2–3 h. Scarcely 10% of this low intensity train-

ing was carried out at 75–85% of the maximal heart rate, i.e. 60–70% of the maximal O₂ uptake. Training at high intensities comprised 11%, whereas strength training, speed training, and explosive training combined constituted 7% of the total training time. This intensity distribution reflected their normal non-intervention training pattern.

The strength training for both groups consisted of two training programs. The first program was made up of general exercises with no use of weights, and was organized as circle training. The work periods were 1 min and the rest periods were 30 s. Total duration of the program was 45 min. The other strength training program consisted of exercises designed to specifically train arm and leg muscles that are viewed to be of particular importance in cross country skiing. This program had work periods of 30 s and rest periods of 15 s. Note that in Table 2 strength training is combined with speed and explosive training to define a third category of training. The reason is that speed training and explosive training generally do not generate maximal heart rate although the exertion is maximal and are, therefore, viewed to have more in common with strength training. Thus, the intensity scale seen in Table 1 was not used for any of these three types of training.

When training on dry land, the explosive training was carried out with movements imitating freestyle and the classical techniques, and were frequently performed with ski poles. Speed training was of shorter duration where the subject ran or roller-skied. It was made clear to the subject that speed as well as explosive training should be done with maximal effort: A work period was 10–15 s, and rest periods between repetitions lasted 2 min. When training on snow, the speed training and explosive training consisted of shorter intervals (10–15 s) with double-poling, diagonal striding or another ski technique performed in easy terrain.

Intensity control and training reports

For each subject an intensity scale was determined based on data from the first test and the subject's maximal heart rate. During each

training session the skier wore a wireless heart rate monitor (Polar 4000) to index training intensity. The results from the heart rate registrations were transferred to a computer by an interface and a specialized computer program (Polar Software). A daily diary describing training activities was submitted.

Blood lactate analysis

Unhemolyzed blood lactate concentration was measured using a YSI Model 1500 Sport Lactate Analyzer (Yellow Springs Instruments; Yellow Springs, Ohio, USA).

Statistics

The data are given as means \pm standard error of mean (SEM) or individual values unless otherwise stated. Two-factor analysis of variance (ANOVA: Groups by repeated measures) tested overall group differences, time differences and the interaction of the group and time factors. Follow-up tests involved t-tests to precisely define the source of a significant effect in the ANOVAs. One-sample t-test was used when the same group of subjects was compared at different times, and between subjects t-test was applied in pairwise between-group comparisons. An alpha level of .05 was used as significance criterion in all statistical tests. The analyses were carried out by the SPSS Windows version 7.5.1.

RESULTS

Weekly running sessions

The amount of low intensity exercise (< 85% of the maximal heart rate) did not differ between the two groups ($F_{1,9}=1.29$; Table 2). The high training-intensity group trained nearly twice as much as the moderate training-intensity group at high training-intensity (>85% of the maximal heart rate [$F_{1,9}=16.58$, $p=0.003$]). This overall group difference was mainly reflected in the intensity zone near the lactate threshold ($F_{1,9}=12.39$, $p=0.007$; Figure 1). With respect to (a) total volume of training and (b) strength, speed and explosive training combined, no substantial effect of the group factor could be revealed ($F_{1,9}=0.07$ and $F_{1,9}=0.93$, respectively).

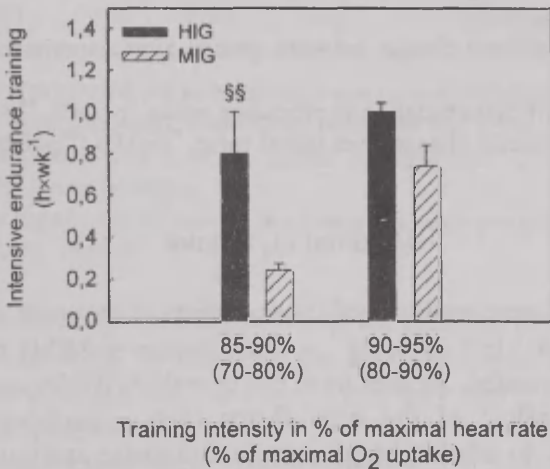


Figure 1. Distribution of intensive endurance training in two groups of Norwegian male junior cross-country skiers during a 12 wk training period (mean \pm SEM).

HIG: High training-intensity group ($n=5$), MIG: Moderate training-intensity group ($n=6$).

§ denotes significant difference between groups, §§ $p < 0.01$.

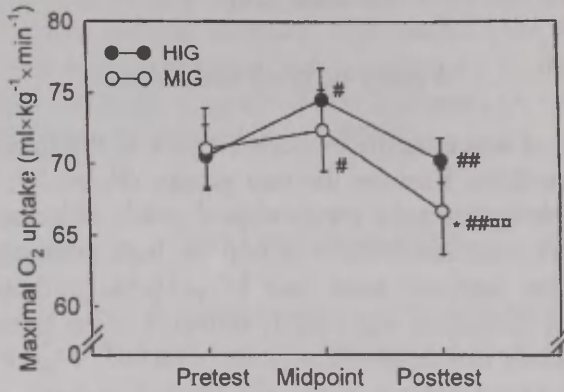


Figure 2. The maximal O_2 uptake for two groups of Norwegian male junior cross country skiers during a 12 wk training period (mean \pm SEM). HIG: High training-intensity group ($n=5$), MIG: Moderate training-intensity group ($n=6$).

* denotes significant change between groups after experimental period, $p < 0.05$.

[#] denotes significant change from preceding value, $^{\#} p < 0.05$, $^{##} p < 0.01$.

$^{\square}$ denotes significant change from initial value, $^{\square} p < 0.05$, $^{m\square} p < 0.01$.

Maximal O_2 uptake

All subjects were well-trained cross country skiers with a maximal O_2 uptake of $71 \pm 2 \text{ ml} \times \text{kg}^{-1} \times \text{min}^{-1}$ (mean \pm SEM) before the study was initiated. Results from the overall ANOVA stated that a substantial effect of the time factor (pre – midpoint – post) emerged due to relatively high scores at midpoint and low scores at posttest ($F_{2,18}=32.22$, $p < 0.0004$) (see Figure 2). The two groups revealed no overall difference in maximal O_2 uptake ($F_{1,9}=0.24$). However, the time and group factors yielded a significant interaction due to a relative drop in maximal O_2 uptake from pretest over the two following tests in the moderate training-intensity group, when compared with the high training-intensity group ($F_{2,18}=5.59$, $p=0.013$). More detailed results from t-tests stated that during the first 6 wk of the training (dry land period) the values increased by

4.0 ± 1.2 (+5.6%, $t_4=3.23$, $p=0.03$) and 1.3 ± 0.5 $\text{ml} \times \text{kg}^{-1} \times \text{min}^{-1}$ (+1.8%, $t_5=2.57$, $p=0.05$) for the high training-intensity group and the moderate training-intensity group, respectively (Figure 2). Moreover, there was a tendency towards a group difference in training effect from pretest to midpoint ($t_9=2.17$, $p=0.058$) when calculated as difference scores.

Table 3. Mean scores and SEM for lactate threshold variables in two groups of Norwegian male junior cross country skiers during a 12 wk training period (pretest, after 6 wk = midpoint, after 12 wk = posttest).

Parameter	High training-intensity group (n=5)			Moderate training-intensity group (n=6)		
	Pretest	Midpoint	Posttest	Pretest	Midpoint	Posttest
Running velocity at LT ¹ ($\text{m} \times \text{s}^{-1}$)	2.75±0.08	2.72±0.06	2.75±0.06	2.72±0.08	2.84±0.11 [§]	2.75±0.08
O ₂ uptake at LT ($\text{l} \times \text{min}^{-1}$)	3.68±0.13	3.78±0.13	3.70±0.09	3.74±0.18	3.94±0.19	3.73±0.13
Heart rate at LT ($\text{beats} \times \text{min}^{-1}$)	175±8	176±8	179±8	183±2	183±2	182±3

The testing was carried out as treadmill running at 6° (10.5%) inclination.

¹ LT = Lactate threshold. The lactate threshold was taken as the O₂ uptake associated with a blood lactate concentration 1.5 $\text{mmol} \times \text{l}^{-1}$ above the warm-up value.

[§] Denotes significant difference in change between groups from pretest to midpoint, [§] $P < 0.05$.

During the next 6 wk (mainly on snow) the maximal O₂ uptakes fell by 4.3 ± 0.7 $\text{ml} \times \text{kg}^{-1} \times \text{min}^{-1}$ for the high training-intensity group (-5.8%, $t_4=-6.51$, $p=0.003$) and by 5.7 ± 0.8 $\text{ml} \times \text{kg}^{-1} \times \text{min}^{-1}$ for the moderate training-intensity group (-7.9%, $t_5=-7.0$, $p=0.001$). Pre- to post-difference scores stated that the maximal O₂ value was almost unchanged (decreased by 0.3 ± 1.2 $\text{ml} \times \text{kg}^{-1} \times \text{min}^{-1}$; -0.4%, $t_4=-0.27$) for the high training-intensity group, whereas this value for the moderate training-intensity group fell significantly by 4.4 ± 0.8 $\text{ml} \times \text{kg}^{-1} \times \text{min}^{-1}$ (-6.6%, $t_5=-5.67$, $p=0.002$). In this way, the effect of training differed between the two groups when examined over the whole 12 wk training period ($t_9=2.94$, $p=0.016$) and, thus,

explained the significant interaction of the group and time factors in the ANOVA. There were no systematic changes in body weight during the twelve week training period, meaning that the changes in the maximal O_2 uptake, relative to the body mass, reflected changes in the maximal O_2 uptake in absolute terms as well.

Lactate threshold variables

Before the training period the subjects reached their lactate threshold at an O_2 uptake of 51.9 ± 1.7 and 53.6 ± 1.9 $\text{ml} \times \text{kg}^{-1} \times \text{min}^{-1}$, corresponding to a running velocity of 2.75 ± 0.08 and 2.72 ± 0.08 $\text{m} \times \text{s}^{-1}$ for the high training-intensity and moderate training-intensity groups, respectively (Table 3). The ANOVA for running velocity at lactate threshold showed no overall significant effects of the time factor ($F_{2,18}=0.65$) and the group factor ($F_{1,9}=0.04$) (see Table 3). However, there was a tendency of an overall interaction of the time and group factors because running velocity peaked at midpoint for the moderate training-intensity group and was reduced at midpoint in the high training-intensity group ($F_{2,18}=3.09$, $p=0.07$). More detailed results from the t-tests stated that the high training-intensity group did not substantially change their running speed at lactate threshold during the first 6 wk of training (-0.03 ± 0.06 $\text{m} \times \text{s}^{-1}$, -1.0%), whereas the moderate training-intensity group increased their running speed by 0.12 ± 0.03 $\text{m} \times \text{s}^{-1}$ ($+4.1\%$, $t_5=3.46$, $p=0.02$). Between the midpoint test and the posttest, and between pre- and posttest, there were no significant changes in the treadmill speed at the lactate threshold for either of the groups.

The overall ANOVA for the absolute lactate threshold ($\text{ml } O_2 \times \text{kg}^{-1} \times \text{min}^{-1}$) (Figure 3a) stated that a significant time effect occurred ($F_{2,18}=4.73$, $p=0.022$). This was due to a trend for the threshold to peak at midpoint for both groups. Moreover, there was no overall group difference in lactate threshold ($F_{1,9}=0.79$). Also, the interaction between the time and group factors yielded no significant effect ($F_{2,18}=1.47$) despite a trend for scores to increase more at midpoint among the moderate intensity skiers than among

the high intensity group. Moreover, at the end of the first 6 wk of training the O_2 uptake at the lactate threshold did not change in the high training-intensity group ($1.1 \pm 1.3 \text{ ml} \times \text{kg}^{-1} \times \text{min}^{-1}$, +2.1%). In contrast, the moderate training-intensity group increased their O_2 uptake at the lactate threshold by $2.7 \pm 0.6 \text{ ml } O_2 \times \text{kg}^{-1} \times \text{min}^{-1}$ (+5.0%, $t_5=4.08$, $p=0.01$). (Table 3, Figure 3a). From the midpoint test to the posttest the O_2 uptake at the lactate threshold was left nearly unchanged in the high training-intensity group (-1.5%), whereas for the moderate training-intensity group the O_2 uptake at the lactate threshold fell significantly by $3.4 \pm 1.2 \text{ ml} \times \text{kg}^{-1} \times \text{min}^{-1}$ (-6.0%, $t_5=-2.84$, $p=0.04$). The differences between the pre- and posttest revealed no significant changes within the two groups concerning the O_2 uptake at the lactate threshold (Figure 3a).

The overall ANOVA for the relative O_2 uptake at lactate threshold (% of maximal O_2 uptake, see Figure 3b) stated that a significant time effect occurred ($F_{2,18}=4.06$, $p=0.035$). Also, there was an overall group difference in the relative O_2 uptake at lactate threshold ($F_{1,9}=10.76$, $p=0.010$), and there was a weak tendency of an overall interaction of the time and group factors ($F_{2,18}=2.72$, $p=0.093$). More detailed t-tests showed that these findings were partly due to a continuous increase from pretest over the two following tests in the moderate training-intensity group, ending 5.1% above the pretest value ($t_5=2.63$, $p=0.046$). In contrast, the relative O_2 uptake at the lactate threshold for the high training-intensity group first decreased at midpoint, and then increased at posttest ($t_4=3.83$, $p=0.019$). The effect was also reflected in difference scores from pretest to midpoint ($t_9=-2.44$, $p=0.037$). However, in spite of these different patterns, there was no significant group difference in change between pretest and posttest ($t_9=-1.76$, $p=0.112$).

For the heart rate at lactate threshold, results from the overall ANOVA indicated no significant effect of the time factor ($F_{2,18}=0.50$). Moreover, neither the group factor ($F_{1,9}=0.81$) nor the interaction of the time and group factors ($F_{2,18}=1.34$) yielded significant group differences in heart rate at lactate threshold.

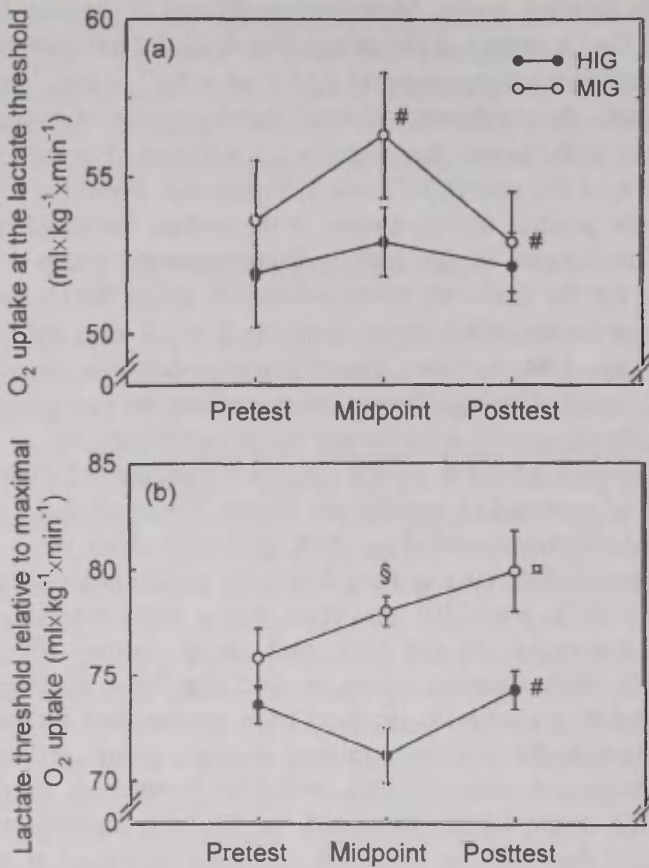


Figure 3. Lactate threshold scores at pretest, after 6 wk (midpoint) and after 12 wk for two training groups of Norwegian male junior cross country skiers (mean \pm SEM).

(a) Scores on O₂ uptake at the lactate threshold and (b) lactate threshold relative to the maximal O₂ uptake in two groups of skiers over the course of a 12 wk training period.

HIG: High training-intensity group (n=5), MIG: Moderate training-intensity group (n=6).

§ denotes significant change between groups, §p<0.05.

denotes significant change from preceding value, #p<0.05.

□ denotes significant change from initial value, □p<0.05.

DISCUSSION

A main finding of this study is that the high training-intensity group improved and maintained their maximal O_2 uptake when compared with a relative drop of maximal O_2 uptake in the moderate training-intensity group over a 12 wk training period. The maximal O_2 uptake peaked after 6 wk and returned to pretest level for the high training-intensity group, whereas a drop in maximal O_2 uptake was observed in the moderate training-intensity group. Thus, the hypothesis of a significant improvement of maximal O_2 uptake for the high training-intensity group was most strongly supported by training on dry land. A second main finding was that during the whole training period the O_2 uptake at the lactate threshold did not significantly change for either group. This finding was not according to prediction.

Training during the study

The training period was carried out from October to January, thus comprising both dry land training and on snow training. Both groups followed standardized training programs with minimal differences between the groups with respect to movement patterns and training methods. The subjects were good cross country skiers, although not among the top ten in their age group in Norway. Evertsen *et al.* [9, 10] investigated elite skiers with a lactate threshold close to 90% of their maximal O_2 uptake, who consequently carried out more than 80% of their training at intensities close to their lactate threshold. The lactate threshold of the subjects in the present study was a great deal lower, approximately 75% of the maximal O_2 uptake. Exercise at or slightly above the threshold was therefore indicated for these subjects, intensities that lead to exhaustion within 10–30 min if no pause is permitted. Intensive training was accordingly restricted to three sessions per week, even for the high training-intensity group.

Maximal O₂ uptake

The maximal O₂ uptake was measured during uphill treadmill running regardless of whether the subjects trained by running, roller-skiing, or by skiing. Cross country skiers may show higher values of the maximal O₂ uptake when tested on uphill skiing than on uphill treadmill running, at least in the on snow period [31]. Thus, the drop in the maximal O₂ uptake observed during the on snow training period could be a consequence of the method of exercise used for the testing. However, the maximal O₂ uptake rose more and dropped less for the high training-intensity group than for the moderate training-intensity group in the two phases of this study. This finding corroborates earlier studies showing that intensity appears to be the key factor in improving the maximal O₂ uptake [18, 23, 26, 28, 35]. Training at high intensity may increase the aerobic potential of fast-twitch fibres and enhance cardiac adaptation to a greater extent than training at lower intensity [7, 8, 16, 21]. The present investigation extends the findings of earlier studies [18, 23, 26, 34] by showing that while the maximal O₂ uptake dropped below the pretest value for the subjects training at moderate intensity when they started their on snow training, those training at high intensity maintained their maximal O₂ uptake, as tested on treadmill running. This suggests that an increased training intensity in the on snow training may be of importance as it appears to reduce the negative effects of slow distance training on the maximal O₂ uptake.

The changes observed in the maximal O₂ uptake during the study may also reflect that the subjects reduced their total training volume as they moved into the on snow period compared with the dry land period. This reduction was partly due to the difficult weather conditions common in the early phase of training on snow, but it also may reflect a tradition of lowering total training volume in the last part of the pre-competition period. However, this reduction in total training volume was not compensated for by a relative shift toward high-intensive training and, consequently, the overall training stimulus was reduced. Although cross country skiers must take into account peak performance strategies and seasonal varia-

tions, there should be no need to reduce the maximal O_2 uptake in the important transition period, especially since it has been suggested that the maximal O_2 uptake sets the upper limit for lactate threshold [6]. This further points out the importance of paying attention to high-intensive training in the transition period.

Lactate threshold variables

The O_2 uptake at the lactate threshold did not change for either group during the study. The finding of an unchanged O_2 uptake at the lactate threshold for the high training-intensity group over the two phases of the study, in conjunction with the changes in maximal O_2 uptake, explains why their lactate threshold relative to the maximal O_2 uptake showed first an initial decrease with a subsequent increase to the pretest level (cf. Figure 3b). The lactate threshold relative to the maximal O_2 uptake rose for the moderate training-intensity group, but this effect appears at least partly to have been a consequence of a reduced maximal O_2 uptake. This is consonant with the findings of Rusko [26] who suggested that adaptations at the sub-maximal level, as indicated by the lactate threshold, seem to be independent of training intensity. Rusko [26] further showed that only distance-trained skiers were able to increase their O_2 uptake at the lactate threshold relative to the maximal O_2 uptake, concluding that distance training at relatively low intensity (i.e. below the lactate threshold) is the most effective way to improve sub-maximal work capacity. Conversely, Evertsen *et al.* [9, 10] in the elite skier study referred to above, found that training at intensities close to the lactate threshold was advantageous in improving the performance on a 20 min running test and in elevating the lactate threshold, as well as in terms of the enhancement of biochemical factors in the muscles. The well-trained subjects in that investigation carried out most of their training at intensities close to the lactate threshold. Their findings could mean that the subjects in the present inquiry might have benefited from some reduction of the high-intensity training. It could also be that large doses of training at high intensity require a high initial lactate

threshold. This issue is clearly not settled because several studies conclude that endurance training at or above the lactate threshold is pre-eminent with respect to raising the lactate threshold [17, 19, 30, 33, 35]. Others report similar improvements for training below versus above the lactate threshold [4, 25].

Training specificity

While the subjects during the first half of the study carried out mainly dry land training, they did largely on snow ski training during the latter half. This may explain the changes observed between the pretest and the midpoint test, and from the midpoint test to the posttest. However, little information is available in the literature on the effect of changes in training methods since most studies on the physiology of cross country skiing have been carried out during the dry land period, when skiers are more available. Training method specificity may be important for the maximal O_2 uptake, and it could be that the reduced maximal O_2 uptake observed for the moderate training-intensity group is a consequence of the disparity between training by skiing and testing by running. Pierce *et al.*, [24] examined changes in the lactate threshold and the maximal O_2 uptake of men training either on the cycle ergometer or in running. Their results suggest that the O_2 uptake at lactate threshold is more sensitive to the specificity of training than is the maximal O_2 uptake.

In conclusion, intensive training at or above the lactate threshold appeared to be more effective in improving or maintaining the maximal O_2 uptake of cross country skiers than training at moderate intensity, and there may be no negative effect on the lactate threshold. The maximal O_2 uptake may drop more during moderately intensive training on snow when compared with intensive training. Hence, well-trained cross country skiers may be well-advised to include more training at intensities close to the lactate threshold as a general prescription, and in particular during training on snow. Further investigations are under way to examine the effects of strength training upon the performance in cross country skiing.

ACKNOWLEDGMENTS

We are grateful to Jon Ingulf Medbø and Sven Svebak for statistical advice and for critical reviews of the manuscript, and to Oddbjørn Floan for technical assistance and excellent laboratory work. We also acknowledge Peder Einar Slind and Magne Sjøli for their collaboration at the two high-schools involved in this study. An excerpt of the study was presented orally at the Norwegian Society for Sport Research Annual Conference (Boe, Telemark, November 11–12, 1999). This study was partly supported by the Norwegian Olympic Committee, the Norwegian Association of Cross Country Ski Coaches, and by Polar Electro Inc., Finland.

REFERENCES

1. **Acevedo E. O., Goldfarb A. H.** (1989) Increased training intensity effects on plasma lactate, ventilatory threshold, and endurance. *Med. Sci. Sports Exerc.* 21: 563–568.
2. **Allen W. K., Seals D. R., Hurley B. F., Ehsani A. A., Hagberg J. M.** (1985) Lactate threshold and distance-running performance in young and older endurance athletes. *J. Appl. Physiol.* 58: 1281–1284.
3. **Andersen I., Nymoen P.** (1991) Langrenn. Universitetsforlaget, Oslo.
4. **Belman M. J., Gaesser G. A.** (1991) Exercise training below and above the lactate threshold in the elderly. *Med. Sci. Sports Exerc.* 23: 562–568.
5. **Bergh U.** (1982) *Physiology of Cross-Country Ski Racing.* Human Kinetics, Champaign IL.
6. **Coyle E. F., Martin W. H., Ehsani A. A., Hagberg J. M., Bloomfield S. A., Sinacore D. R., Holloszy J. O.** (1983) Blood lactate threshold in some well-trained ischemic heart disease patients. *J. Appl. Physiol.* 54: 18–23.
7. **Cunningham D. A., McGrimmon D., Wach L. F.** (1979) Cardiovascular response to interval and continuous training in women. *Eur. J. Appl. Physiol.* 41: 187–197.
8. **Essen-Gustavsson B., Henriksson J.** (1984) Enzyme levels in pools of microdissected human muscle fibres of identified type. Adaptive response to exercise. *Acta Physiol. Scand.* 120: 505–515.

9. **Evertsen F., Medbø J. I., Jebens E., Gjøvaag T. F.** (2000) Effect of training on the activity of five muscle enzymes studied on elite cross-country skiers. *Acta Physiol. Scand.* 167: 247–257.
10. **Evertsen F., Medbø J. I., Jebens E., Nicolaysen K.** (1997) Hard training for 5 mo increases $\text{Na}^+\text{-K}^+$ pump concentration in skeletal muscle of cross-country skiers. *Am. J. Physiol.* 272: R1417–1424
11. **Farrell P. A., Wilmore J. H., Coyle E. F., Billing J. E., Costill D. L.** (1993) Plasma lactate accumulation and distance running performance. *Med. Sci. Sports Exerc.* 25: 1091–1097.
12. **Gaskill S. E.** (1998) Cross-country skiing. *Human Kinetics, Champaign IL.*
13. **Gaskill S. E., Serfass R., Bacharach D. W., Kelly J. M.** (1999) Responses to training in cross-country skiers. *Med. Sci. Sports Exerc.* 31: 1211–1217.
14. **Hagberg J. M., Coyle E. F.** (1983) Physiological determinants of endurance performance as studied in competitive racewalkers. *Med. Sci. Sports Exerc.* 15: 287–289.
15. **Helgerud J., Ingjer F., Strømme S. B.** (1990) Sex differences in performance-matched marathon runners. *Eur. J. Appl. Physiol.* 61: 433–439.
16. **Henriksson J., Reitman J. S.** (1976) Quantitative measures of enzyme activities in type I and type II muscle fibres of man after training. *Acta Physiol. Scand.* 97: 392–397.
17. **Henritze J., Weltman A., Schurrer R. L., Barlow K.** (1985) Effects of training at and above the lactate threshold on the lactate threshold and maximal oxygen uptake. *Eur. J. Appl. Physiol.* 54: 84–88.
18. **Hickson R. C., Hagberg J. M., Ehsani A. A., Holloszy J. O.** (1981) Time course of the adaptive responses of aerobic power and heart rate to training. *Med. Sci. Sports Exerc.* 13: 17–20.
19. **Hurley B. F., Hagberg J. M., Allen W. K., Seals D. R., Young J. C., Cuddihee R. W., Holloszy J. O.** (1984) Effect of training on blood lactate levels during submaximal exercise. *J. Appl. Physiol.* 56: 1260–1264.
20. **Ingjer F.** (1991) Factors influencing assessment of maximal heart rate. *Scand. J. Med. Sci. Sports.* 1: 134–140
21. **Keul J., Dickhuth H. H., Lehman M., Staiger J.** (1982) The athlete's heart — haemodynamics and structure. *Int. J. Sports Med.* 3: 33–43.

22. **McArdle W. D., Katch F. I., Katch V. L.** (1991) *Exercise Physiology*. 3rd ed. Lea & Febiger, London.
23. **Mikeseil K. A., Dudley G. A.** (1984) Influence of intense endurance training on aerobic power of competitive distance runners. *Med. Sci. Sports Exerc.* 16: 371–375.
24. **Pierce E. F., Weltman A., Seip R. L., Snead D.** (1990) Effects of specificity of training on the lactate threshold and VO_2 peak. *Int. J. Sports Med.* 11: 267–272.
25. **Poole D. C., Gaesser G. A.** (1985) Response of ventilatory and lactate thresholds to continuous and interval training. *J. Appl. Physiol.* 58: 1115–1121.
26. **Rusko H.** (1987) The effect of training on aerobic power characteristics of young cross-country skiers. *J. Sports Sci.* 5: 273–286.
27. **Rusko H., Havu M., Karvinen E.** (1978) Aerobic performance capacity in athletes. *Eur. J. Appl. Physiol.* 38: 151–159.
28. **Rusko H., Rakkila P.** (1981) Effect of increased intensity of training on maximum oxygen uptake and muscular performance of young female cross-country skiers. In: *Medicine and Sport, Vol 14 (Women and Sport)*. S. Karger, Basel.
29. **Saltin B., Åstrand P. O.** (1967) Maximal oxygen uptake in athletes. *J. Appl. Physiol.* 23: 353–358.
30. **Sjödin B., Jacobs I., Svedenhag J.** (1982) Changes in the onset of blood lactate accumulation (OBLA) and muscle enzymes after training at OBLA. *Eur. J. Appl. Physiol.* 49: 45–57.
31. **Strømme S. B., Ingjer F., Meen H. D.** (1977) Assessment of maximal aerobic power in specifically trained athletes. *J. Appl. Physiol.* 42: 833–837.
32. **Tanaka K., Matsuura Y., Matsuzaka A., Hirakoba K., Kumagai S., Sun S. O., Asano K.** (1984) A longitudinal assesment of anaerobic threshold and distance running performance. *Med. Sci. Sports Exerc.* 16: 278–282.
33. **Weltman A., Seip R. L., Snead D., Weltman J. Y., Haskvitz E. M., Evans W. S., Veldhuis J. D., Rogol A. D.** (1992) Exercise training at and above the lactate threshold in previously untrained women. *Int. J. Sports Med.* 13: 257–263.
34. **Wenger H. A., Bell G. J.** (1986) The interactions of intensity, frequency and duration of exercise training in altering cardiorespiratory fitness. *Sports Med.* 3: 346–356.

35. **Yoshida T., Suda Y., Takeuchi N.** (1982) Endurance training regimen based on arterial blood lactate: effects on anaerobic threshold. *Eur. J. Appl. Physiol.* 49: 223-230.

Correspondence to:

Boye Welde

North-Troendelag University College

Department of Physical Education, Roestad

NO-7600 Levanger

NORWAY

THE EFFECTS OF TWO STYLES OF TEACHING AND TEACHERS QUALIFICATION ON MOTOR SKILL PERFORMANCE OF THE CARTWHEEL

V. Hein, M. Kivimets

Institute of Sport Pedagogy, University of Tartu, Tartu, Estonia

ABSTRACT

This study tested the effects of direct and indirect teaching on the motor skill acquisition of the fifth grade children. Boys (n=14) and girls (n=14) from two classes were taught a cartwheel with different teaching styles by experienced and inexperienced teachers under school condition during the four lessons. The motor skill of the cartwheel performance were videotaped and estimated prior and post treatment. The results were analysed within groups to determine if the learning was evident, and across groups, to examine the relative effectiveness of two styles of teaching. The interaction of two factors — the teacher's qualification and used methods — on learning outcome was evaluated using two way analysis of ANOVA. The learning outcome of the observed four groups revealed that the direct method was more acceptable for teaching complicated motor skill like cartwheel.

The results of study indicated that learning outcome depends more from the teacher experience than from used methods.

Key words: teaching styles, teacher's qualification, motor skill performance, schoolchildren

INTRODUCTION

Mosston's Spectrum of Teaching Styles has been widely applied in the field of physical education, and has been significantly refined since its inception in 1950's. Originally, the spectrum was a theoretical framework of eight interconnected styles, Style A through H, all derived from the same decision-making scheme [10]. Later, the spectrum was extended to 10 styles [11]. This schema divides decision about the teaching learning process into three sets: planning decisions, execution decisions, and assessment decisions. Each style has a unique theoretical structure determined by who, teacher or learner, makes which decisions. The value of any particular style of teaching lies in the conditions leads to conjecture about each style's probable effect on learner outcomes.

Styles A (command style), B (practice style), C (reciprocal style) and etc from one end of the spectrum are generally qualified as the direct style and the styles from the other end of spectrum as the indirect style of teaching (J-self teaching, I-learner initiated, H-individual program, G-divergent and F-guided discovery) [3, 8, 11, 12].

A good specification of the nature of the direct and indirect teaching styles has been presented by Chen [2]. According to it direct teaching provides security for both teachers and learners, because the program is arranged with a clear structure and progression, so it is easier for teachers and learners to follow and manage. However, disadvantage is that it does not allow creativity, originality, and individual differences. The major advantage of the indirect teaching is that it provides the learners with choice, flexibility, and responsibility for their own independent learning at their own level. The program is for all individuals. The teachers act as facilitators. The limitation of this approach is that it takes time for learners to get used to the concept of independent learning and establish the good habit of self-management.

The selection of an appropriate teaching styles may depends on several factors; such as the goal of the task, learner's previous experience in activity, the content of the activity, the availability of

facilities and equipment, personal preference and skills of the teacher etc [14].

Several studies have been attempted to establish relationships between particular styles of teaching and learning outcomes. Goldberger *et al.* [3] who examined the effects of teaching styles B, C and E (inclusion style) on learning outcome in a hockey accuracy task indicated that all three styles appeared to be beneficial. However, the best results were gained by applying the styles B and C. Additionally, the positive effect of style C on social skill development was established. In the investigation about the effectiveness of the teaching styles in respect of different children aptitudes was found that for children with average aptitude the style B was the most effective [4].

The direct styles are useful and appropriate, particularly when learner's safety or precision performance are required. Goldberger & Howarth [5] and Al-Mulla [1] have noted that if a teacher wants to teach a new and basic skill, then the direct method might be an appropriate choice. Divergent problem solving and guided discovery styles may be suitable for working towards curriculum objectives in dance, gymnastics and games [17].

From above presented statements arise the question, what style of teaching (direct or indirect) is more suitable to teach cartwheel performance as one of the gymnastics movement in curriculum? Does it require safety and precision performance? Is this movement qualified as a complicated movement at school gymnastic level? If yes, then the direct style may be more appropriate for teaching. This opinion is in contradiction with assignment reported by Williams [17].

Al-Mulla [1] summarised the findings of a study designed to investigate teacher's perceptions of different teaching methods and how often they are used during daily teaching. He noted that teachers' knowledge and use of different teaching methods, particularly those that provide more interactive and independent learning strategies for pupils, are limited. Additionally, it was concluded that male teachers more frequently used a direct method while female teachers gave more emphasis to guided discovery method.

Several researchers and scholars have outlined differences between experienced and inexperienced teachers in teaching skills such as in class time management, in teacher instructional and feedback behaviour, in student motor engagement [6, 7, 8, 15]. One of the essential characteristics of the effective/experience teacher is to have large repertoire of teaching styles [9, 13, 14]. However, based on the review of the literature, no studies have compared the effectiveness of different teaching styles in respect of use these by experienced and inexperienced teachers.

Therefore, the objectives of this study were: 1) to determine which teaching style — direct or indirect — is more appropriate to teach cartwheel performance; and 2) to compare the effectiveness of different teaching styles applied by experienced and inexperienced teachers.

MATERIAL AND METHODS

Subjects

Subjects for this study were 28 fifth grade children of one public school. A sample of 14 boys and 14 girls was randomly selected from a population of 64 volunteers. Seven children from each of these subgroups were then randomly assigned to form four treatment groups (two boys' and two girls' groups). In this study 4 groups of learners practised cartwheel under one of two teaching method.

The two female teachers with different experiences taught cartwheel using direct and indirect teaching methods. One of them had more than 15 years teaching experiences and another was the student teacher. The experienced teacher taught the cartwheel in boys and student teacher in girls' groups. Both of them had undergone extensive training in the theory and utilisation of the spectrum of the Teaching Styles. For each method a basic script was prepared. The content of the four lessons applying the indirect and direct method used by both teachers is presented in appendix.

The experts of gymnastics estimated the cartwheel performance from the videotype recordings of the pretest performances of 28 subjects independently. The scale score estimation of the cartwheel performance ranged from 1 to 5 with 0.5 interval. The internal consistency, calculated by Cronbach's alpha was 0.97 between two experienced experts of gymnastics. Variance coefficient of estimation not exceeded 12.5%.

Data analysis

Data are means and standard deviation. Mann-Whitney test was used to test the differences between the observed groups before the experience and paired t-test to evaluate the differences before and after the treatment in each group. To examine the overall effects of these treatments on learning outcome two way interaction analysis of ANOVA was employed. A level of $p < 0.05$ was selected to indicate statistical significance.

RESULTS

Looking first at entry performance it can be seen in Table 1 that significant differences calculated by Mann-Whitney test were not evident among of the treatments groups at the pretest. Post hoc analyses indicated significant differences between the group taught by experienced teacher and the group taught by an inexperienced teacher when the direct method was used. No differences in learning outcome followed when the indirect method was applied. However, in boys' group taught by experienced teacher the cartwheel performance improved more than in the group of girls taught by inexperienced teacher. The main statistics for the interactive effects of teacher qualification and teaching method on learning outcome is showed in Table 2. Figure 1 represents the interaction of two factors; the teacher's qualification and used methods on learning outcome. From those two factors the teacher's qualifica-

tion had more impact on learning outcome than the used method. For both teachers the direct method was more effective in teaching the cartwheel performance than the indirect method.

Table 1. Mean and SD of the test score before and after the treatment procedure of the boys and girls groups.

Methods/ subjects	Pre-treatment		Post-treatment		t-value	p-level
	Mean	SD	Mean	SD		
Indirect method						
Boys (n=7)	3.5*	0.6	3.9	0.8	2.3	NS
Girls (n=7)	3.7*	1.3	3.7	1.3	0.5	NS
Direct method						
Boys (n=7)	2.8*	1.0	3.6	0.9	3.0	0.05
Girls (n=7)	3.7*	1.2	3.9	1.2	1.3	NS

* — notes the absent of differences between all observed groups calculated by Mann Withney test.

Table 2. Two-way interaction of teaching methods and teachers' qualifications on learning outcome.

Effect	MS Effect	df Error	MS Error	F	p-level
Teacher qualifications	1.42	24	0.27	5.2	0.05
Methods	0.57	24	0.27	2.1	NS
Common effect	0.14	24	0.27	0.5	NS

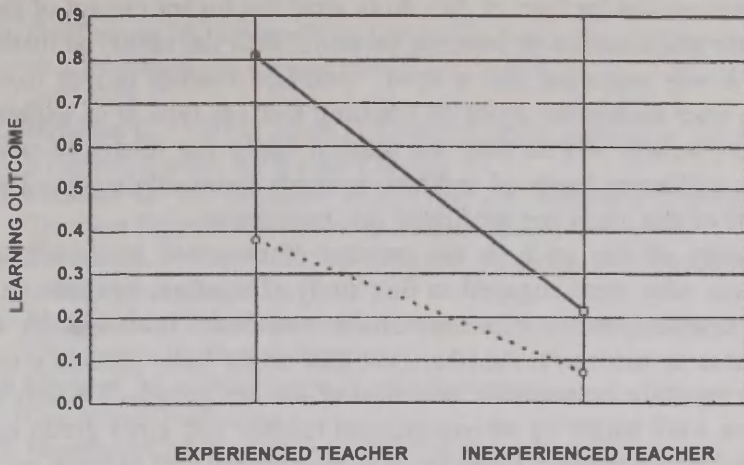


Figure 1. The interaction of teacher's experiences and teaching methods on learning outcome.

- indirect method
- direct method

DISCUSSION

This study represented an attempt to describe the impact of the teacher's qualification and teachers styles on learning outcome in respect of the cartwheel performance. The findings allowed to suggest that to teach cartwheel among schoolchildren by the direct method is more productive than indirect method. So the advice to use the styles of indirect method in gymnastics [17] may be more suitable for such kind of basic skills as travelling, balancing, jumping, rotation etc. but not for teaching cartwheel. Although cartwheel is considered to be a quite simple movement among gymnasts, then at school level in contrast it may be qualified as one of the complicated movements. The preference to use direct style of teaching in case of complicated movement has been also supported by several investigators [3, 4]. Sweeting and Rink [16] have noted that when the direct method is used, the learners receive more instant feedback about performance.

Unexpected findings of this study were the higher impact of the teacher qualification on learning outcome than the observed methods. It was supposed that a newly qualified teacher is free from often used traditional styles of teaching that are typical of experienced teachers and so they are able to apply the relatively new styles (different kinds of indirect method) more efficiently. The results of this study not confirmed this hypothesis.

Limits of this study is the number of observed teachers and children who were engaged in this study. Therefore, to make extensive generalisation is not reasonable and future investigation in this area is necessary. Another fact that might have influence on study results concerned the selection of the sex groups. The boy's groups were taught by an experienced teacher and girl's group by inexperienced teacher. However, it is well known that to teach girls in such kind of activity and at this age is easier than boys.

In conclusion, the learning outcome of the observed four groups revealed that the direct method was more acceptable for teaching complicated motor skill like cartwheel. The results of the study indicated that learning outcome depends more on teacher's experience than on used methods.

REFERENCES

1. **Al-Mulla F. H.** (1998) Teacher perceptions about different teaching methods. *BAALPE*. 34: 91-99.
2. **Chen P.** (1999) Effective teaching leadership in physical education beyond 2000. *Proceeding of World Congress of ICHPER & SD*, Cairo.
3. **Goldberger M., Gerney P., Chamberlain J.** (1982) The Effects of Three Styles of Teaching on the Psychomotor and Social Skill Development of Fifth Grade Children. *Res. Quart. Exerc. Sport*. 53: 116-124.
4. **Goldberger M., Gerney P.** (1986) The effects of direct teaching styles on motor skill acquisition of fifth grade children. *Res. Quart. Exerc. Sport*. 57: 215-219.

5. **Goldberger M., Howarth K.** [1993] The national curriculum in physical education and the spectrum of teaching styles. *Br. J. Phys. Educ.* 24: 23–28.
6. **Graham G.** (1992) *Teaching Children Physical Education. Becoming a Master Teacher.* IL, Human Kinetics.
7. **Hellison D. R., Templin T. J.** (1991) *A Reflective Approach to Teaching Physical Education.* IL, Human Kinetics.
8. **Mawer M.** (1998) 4 Ed. *The Effective Teaching of Physical Education.* London, Longman.
9. **Metzler M.** (1990) *Instructional Supervision in Physical Education.* Champaign, IL, Human Kinetics.
10. **Mosston M.** (1981) *Teaching physical education.* 2 ed., Columbus, OH, Merrill Publishing.
11. **Mosston M., Ashworth S.** (1986) *Teaching physical education.* 3 ed., Columbus, OH, Merrill Publishing.
12. **Peake L.** (1994) An overview of the spectrum of teaching styles. Biological and pedagogical problems of physical education and sport. *Acta et Commentationes Universitatis Tartuensis*, 967, 5–10.
13. **Rink J. E.** (1985) *Teaching Physical education for Learning.* St Louis, Miss., C. V. Mosby.
14. **Siedentop D.** (1991) *Developing Teaching skills in physical Education.* 3 ED. California, Mayfield Pub. Co.
15. **Tan S. K. S.** (1996) Differences Between Experienced and Inexperienced Physical Education Teachers' Augmented Feedback and Interactive Teaching decisions. *J. Teach. Phys. Educ.* 15: 151–170.
16. **Sweeting T., Rink J. E.** (1999) Effects of Direct Instruction and Environmentally Designed Instruction on the Process and Product Characteristics of a Fundamental Skill. *J. Teach. Phys. Educ.* 18: 216–233.
17. **Williams A.** (1993) Aspects of teaching and learning in gymnastics. *Br. J. Phys. Educ.* 24: 29–32.

APPENDIX

The content of lessons

The time for teaching the cartwheel was 20–25 minutes in each lesson

Lessons activity in case of use the direct method

Lesson 1

1. Exercises for practising the ability to strength muscles and keep body under control.
 - a) In supine position with the arms outstretched forwards for 5 to 10 sec. stretch as long as possible while at the same time squeezing the legs, buttocks and arms as tightly as possible (3 time)
 - b) The same exercise in prone position
 - c) Using the assistant who raises up the tight body from supine position supporting subjects under the heels or legs (3 times)
2. Exercises for practising overturned position (handstand). Handstand was included due to its' similarity of movement with cartwheel performance at the first phase.
 - a) From tuck position, back to the wall or to the Swedish ladder, climbing up by feet (to the handstand) and down to the initial position.
 - b) From lying support, legs on the first step of the Swedish ladder climbing up by feet (to the handstand) and down to the initial position.

Lesson 2

1. The repetition the exercises from the first lesson (1 time)
2. Exercises for handstand
 - a) From standing position the arms upwards, forward step by the take off leg with trunk pressing downward.
 - b) The same as the previous exercise, but with trunk pressing downward to hand support on floor.
 - c) Pushing off with support leg from squat support position on one leg.
 - d) Swinging up with backward leg in squat support position on one leg.

- e) Linking the exercise a,b,c to coherent movement (From standing position forward step by take off leg and swing with another leg to perform handstand)

Lesson 3

- a) The performing handstand against the wall with an assistant.
- b) The straddling the legs on the headstand against the wall or with assistant.
- c) The same as in previous, but on the handstand

Lesson 4

- a) The practising the exercises from lesson 3.
- b) The practising the whole cartwheel
- c) The practising the cartwheel performance across the mat only with hand support (to contact mat with leg was not permitted).
- d) The practising the cartwheel along the line.

Indirect method

From the spectrum of indirect method the guided discovery was used to lead the learner through a series of small steps to discover the solution to perform the cartwheel (divergent problem solving).

Lesson 1

The aim is to exercise the support phase on the hands.

Divergent questions:

1. What are the possible ways to balance with your two hands and one leg.
2. What are three possible ways to perform the balance with two hands on floor and one leg on an apparatus, which is high than the support space of hands.
3. Try to balance with one hand and one leg.
4. Show three possible ways to move only on two hands.

Lesson 2

1. The repetition of more complicated exercises performed by pupils in lesson 1 and to enhance the time of balance.
2. The pupils were asked to straddle legs in different positions and in different space levels.

Lesson 3

To practise the cartwheel by using a circle on the floor. The children stood on the circle facing the middle of the circle. Then they tried to place hands and feet on the line of circle. As they become proficient, the circle was made progressively larger until it was a straight line.

Lesson 4

To practise the cartwheel over a gymnast bench.

To practise the cartwheel over the carton boxes of different height which were placed on the gymnast bench. It allows children to choose the place to practice cartwheel according to their performance ability.

Correspondence to:

Vello Hein
Institute of Sport Pedagogy
University of Tartu
18 Ülikooli Street
50090 Tartu
Estonia

RATE OF LACTATE PRODUCTION DURING 10 AND 30 S BICYCLE SPRINTS VERSUS PHOSPHOFRUKTOKINASE ACTIVITY

J. I. Medbø*, E. Jebens*, P. Gramvik**

*National Institute of Occupational Health, Oslo, Norway

**Agder College, Faculty of Health and Sports, Kristiansand, Norway

ABSTRACT

The ATP synthesis is regulated by rate-limited enzymatic processes. It could be that the maximum activity of for example phosphofruktokinase (PFK) limits the performance during high-intensity exercise. To examine this possibility seven healthy young men cycled for 9–15 s to exhaustion. Muscle biopsies were taken before and immediately after exercise and analyzed for lactate and phosphocreatine concentrations. The muscle lactate concentration rose by $10.4 \pm 1.6 \text{ mmol} \times \text{kg}^{-1}$ wet muscle mass (mean \pm SEM), corresponding to a mean glycolytic flux expressed in units of fructose-6-phosphate (F-6-P) of $0.43 \text{ mmol F-6-P s}^{-1} \text{ kg}^{-1}$. The phosphocreatine and ATP concentrations fell by 6.9 ± 0.9 and $0.5 \pm 0.2 \text{ mmol} \times \text{kg}^{-1}$, respectively, during the sprint. The postexercise phosphocreatine concentration was $8.9 \pm 0.8 \text{ mmol} \times \text{kg}^{-1}$, suggesting that there was a considerable energy reserve at exhaustion. PFK's catalytic activity expressed in units of F-6-P was $0.91 \pm 0.06 \text{ mkat} \times \text{kg}^{-1}$ wet muscle mass at 38°C , which is more than twice the observed glycolytic flux. PFK thus seemed to work at substrate concentrations less than its K_m . This observation together with the high phosphocreatine concentration at exhaustion suggest that inadequate ATP-supply because of an inadequate catalytic activity of PFK was not a cause of fatigue during these experiments.

Key words: energy release, exercise, muscle metabolism, muscle energetics, phosphocreatine, phosphofruktokinase, EC 2.7.1.11, power output.

INTRODUCTION

During intense exercise working muscles use ATP at a high rate. The muscle ATP-concentration does, however, not change much during exercise [4, 9, 15, 16], showing that ATP is resynthesized as fast as it is being used. Aerobic and anaerobic enzymatic processes do the resynthesis, and the two quantitatively most important anaerobic processes are linked to phosphocreatine breakdown and to the glycolytic production of lactate.

All enzymatic processes have rate limits. The rate of aerobic ATP production may be set by mitochondrial enzymes or by the O₂ supply, and aerobic processes can therefore not provide ATP fast enough to provide all the ATP needed during high intensity exercise. It is also well established that the glycolytic enzyme phosphofructokinase (PFK) has a clear rate limit [1], but it is not known whether this limit is ever reached during exercise in man. It has been shown that for exercise intensities that can be maintained for at least 30 s, the higher the exercise intensity, the higher is the rate of lactate production [16, 23]. Thus, rate limits were not apparent in those studies, but limits may perhaps be found during exercise at higher intensities and of shorter durations where the ATP turnover rate is even higher.

To study the energy release and possible enzymatic rate limits during very high exercise intensities, we let subjects cycle at constant power for ≈ 10 and 30 s to exhaustion. Muscle biopsies were taken before and immediately after exercise, and the anaerobic energy release was determined from measured changes in muscle metabolites. The catalytic activity of phosphofructokinase was measured and compared with the observed glycolytic flux.

SUBJECTS AND METHODS

Subjects

Seven men 27 ± 2 yr old (mean \pm SD), 1.80 ± 0.06 m tall, weighing 78 ± 9 kg and with a maximal O_2 uptake of $39 \pm 3 \mu\text{mol} \times \text{s}^{-1} \times \text{kg}^{-1}$ ($52 \pm 4 \text{ ml}_{\text{STPD}} \times \text{min}^{-1} \times \text{kg}^{-1}$) gave their written consent to serve as subjects in the experiments after thorough information about the experiments and procedures. The Regional Ethics Committee approved the experimental protocol.

Procedures

Exercise was carried out on an electrically braked Krogh-type bicycle ergometer [10] at 2.0 Hz pedaling frequency. For the 10 s sprints described below the frequency was set to 2.1 Hz to compensate for the delay at the onset of exercise. The frequency was continuously shown to the subjects on an analog instrument. The ergometer was also equipped with a work meter counting the number of revolutions of the flywheel (6 rev s^{-1} at 2 Hz) and thus recording the work done during each experiment accurately. The reported power is the ratio between the recorded work done and the exercise duration recorded by a stop watch. Only negligible deviations between the preset and the actual power were found in all experiments.

The bicycle ergometer was equipped with flat pedals without straps around the foot. Thus, work was done on the ergometer only during the downward push.

Pretests

Each subject went through several pretests during the weeks before the experiments for familiarization with the equipment, exercise, and the experiments. The maximal O_2 uptake was determined by

the leveling off criterion [21]. Finally, the highest power that could be maintained for ≈ 10 s and ≈ 30 s was established for each subject.

Experiments

On the day of the experiment the subject arrived at the laboratory in the morning after an overnight fast. Local anesthesia (Xylocain, $10 \text{ g} \times \text{l}^{-1}$) was given before four incisions in the skin and muscle fascia were made over the lateral portion of the quadriceps muscle of each thigh. Thereafter the subject warmed up for 10 min at 50% of his maximal O_2 uptake before muscle biopsies were taken. The subjects cycled for about 10 s at the preset power to exhaustion while expired air was collected in a Douglas bag for later measurement of the O_2 uptake. As soon as possible after exercise (≈ 10 s) and while the subjects were still sitting on the bike, muscle biopsies were taken from both legs and immediately frozen in freon-22 cooled by liquid N_2 . For each biopsy taken we recorded the exact time from the end of exercise to the biopsy was sampled and frozen.

In the second experiment carried out after a 1 h break the subject carried out a 30 s ride to exhaustion. The same procedures as for the 10 s experiment were repeated apart for a lower power to allow for a longer exercise duration. Since the muscle and blood lactate concentrations were back to the resting value before the 30 s exercise, the 10 s sprints were assumed to have no important effect on the second performance [2]. The biopsy sites before and after the two exercises were randomized by a Latin square procedure.

Blood samples taken from a prewarmed finger as soon as possible after exercise, and 2, 5, 8, 11, 15, 20, and 30 min after exercise were analyzed for lactate.

Analyses

Biochemical analyses. Muscle and blood lactate concentrations were measured by enzymatic photofluorometry according to Passoneau and Lowry [19], using the increase of NADH-concentration in the LDH-reaction (lactate dehydrogenase, EC 1.1.1.27). Pyruvate produced was further processed by the glutamate-pyruvate transaminase reaction (l-alanine:2-oxoglutarate aminotransferase, EC 2.6.1.2), thus allowing a complete processing of all lactate in the sample. The muscle glucose-6-phosphate concentration was measured enzymatically according to Passoneau and Lowry [19]. Muscle phosphocreatine and ATP concentrations were measured in neutralized perchloric acid extracts of muscle biopsies by a luminescent method using firefly luciferase (EC 1.13.12.7; Bio-Orbit, Turku, Finland; Ref. 11). All values are expressed per kilogram wet muscle mass or per liter of blood. Since a working muscle probably does not take up fluid during exercise lasting less than 1 min [16], no correction was made for possible changes in muscle fluid content during the exercises.

Enzyme activity. The catalytic activity or ability of phosphofructokinase (PFK, EC 2.7.1.11) was measured at 340 nm on a Shimadzu MPS-2000 spectrophotometer according to a procedure by Passoneau and Lowry [19]. Muscle tissue dissected free from visible fat and connective tissue was homogenized in 50 mmol \times L⁻¹ Tris-HCl buffer at pH 8.1 and at a ratio of 20 g tissue per liter of buffer. The tissue was homogenized by hand using a tight fitting glass-glass homogenizer. The activity was measured at around 25 and 38°C within 4 h after homogenization to avoid reduction in the catalytic activity since separate pretests showed that some activity was lost upon subsequent freezing and thawing of the homogenate (data not shown). As little as 0.4 mg of tissue was enough for each analysis. The catalytic activities are expressed in katal (moles of fructose-6-phosphate [F-6-P] processed per second) at 25 and 38°C. The actual temperatures of measurements were 25.4–25.9 and 37.6–38.4°C (range), respectively, and the reported rates are

the measured rates corrected to 25.0 or 38.0°C, respectively, using a temperature coefficient of 1.07 K⁻¹.

O₂ uptake. The volume of expired air collected in Douglas bags was measured in a wet spirometer, while the fractions of CO₂ and O₂ were measured on an automatic system (CO₂ on an analyzer from Simrad Optronics, Oslo, Norway; O₂ on an S 3A/I analyzer from Ametek, Pittsburgh, PA, USA).

Fiber typing. The staining procedure involved different combinations of alkaline, formaldehyde, and acid preincubations in sequence before a routine myosin ATP-ase staining procedure. The muscle fibers were classified as type 1 or type 2 by a method described elsewhere [22].

All chemicals used were of analytical quality, and chemicals and enzymes used were bought from Boehringer Mannheim (Mannheim, Germany) and Sigma (St. Louis, MO, USA).

Data analysis and statistics

For phosphocreatine (PCr) the values in the biopsies frozen ≈12 s after exercise were 1.9±0.8 mmol × kg⁻¹ less than in the biopsies frozen ≈20 s later. Therefore only the values from the first biopsy taken from each subject are reported for phosphocreatine. For the other metabolites no differences were seen between the values from the two sets of biopsies, and the reported values are therefore the mean of the two sets of biopsies. The data are given as individual results or as mean ± SEM.

RESULTS

During the so called 10 s bouts the subjects cycled at 12.9±0.3 W × kg⁻¹ for 12.0±0.9 s to exhaustion. During the so called 30 s bouts the subjects cycled at 9.7±0.3 W × kg⁻¹ for 29±2 s to exhaustion.

Amounts of anaerobic energy release

The preexercise muscle metabolite concentrations were 1.9 ± 0.2 (lactate), 17.6 ± 0.9 (PCr) and 3.8 ± 0.2 $\text{mmol} \times \text{kg}^{-1}$ (ATP) before both exercises. The postexercise biopsies were taken and frozen ≈ 12 and 32 s after the end of the bout. During the 10 s sprints the muscle lactate concentration rose by 10.4 ± 1.6 $\text{mmol} \times \text{kg}^{-1}$ wet muscle mass, the phosphocreatine concentration fell by 6.9 ± 0.9 $\text{mmol} \times \text{kg}^{-1}$, while the glucose-6-phosphate concentration rose by 2.2 ± 0.3 $\text{mmol} \times \text{kg}^{-1}$. Lactate was thus produced at a mean rate of 0.86 $\text{mmol} \times \text{s}^{-1} \times \text{kg}^{-1}$, corresponding to 0.43 $\text{mmol F-6-P s}^{-1} \times \text{kg}^{-1}$. The blood lactate concentration rose by 1.3 mmol L^{-1} during the 10 s sprint as judged from blood samples taken around 40 s after the exercise. The peak postexercise blood lactate concentration found 5 min after the exercise was 8.5 ± 0.6 $\text{mmol} \times \text{L}^{-1}$.

During the 30 s rides the lactate concentration rose by 19.7 ± 1.2 $\text{mmol} \times \text{kg}^{-1}$, while the phosphocreatine concentration fell by 10.5 ± 0.9 $\text{mmol} \times \text{kg}^{-1}$ and the glucose-6-phosphate concentration rose by 2.7 ± 0.3 $\text{mmol} \times \text{kg}^{-1}$. The mean rate of lactate production was thus 0.70 ± 0.06 $\text{mmol} \times \text{s}^{-1} \times \text{kg}^{-1}$, corresponding to 0.35 $\text{mmol F-6-P s}^{-1} \times \text{kg}^{-1}$. During both rides the ATP concentration fell by 0.55 ± 0.14 $\text{mmol} \times \text{kg}^{-1}$. The blood lactate concentration rose by 4.2 $\text{mmol} \times \text{L}^{-1}$ during the 30 s ride. The peak postexercise blood lactate concentration was 11.6 ± 0.5 $\text{mmol} \times \text{L}^{-1}$.

The muscle phosphocreatine concentration at exhaustion was 8.9 ± 0.8 $\text{mmol} \times \text{kg}^{-1}$ after the 10 s sprint and 6.5 $\text{mmol} \times \text{kg}^{-1}$ after the 30 s ride. There was thus a considerable reserve at exhaustion in both cases.

In addition to the anaerobic energy release there was a considerable aerobic contribution as judged from the measured O_2 uptake and the use of stored O_2 (see Ref. 15 for details). Consequently, the calculated ATP-turnover rate during the 10 s sprint was 2.7 $\text{mmol} \times \text{s}^{-1} \times \text{kg}^{-1}$ wet muscle mass, which is 2.7 times the value corresponding to the subjects maximal O_2 uptake.

Catalytic activity of phosphofructokinase

The catalytic activity or ability of phosphofructokinase (PFK) expressed in units of fructose-6-phosphate (F-6-P) was 0.43 ± 0.02 mkat \times kg^{-1} wet muscle mass at 25.0°C . The activity measured at 38.0°C was 0.91 ± 0.06 mkat \times kg^{-1} wet muscle mass. The mean rate of lactate production of 0.43 mmol F-6-P $\text{s}^{-1} \times \text{kg}^{-1}$ wet muscle mass ($n=7$) was 47% of PFK's activity at 38°C (Figure 1). Moreover, the observed rates of lactate production averaged over the 10 s sprints were not correlated to the enzymatic activity (not shown, $P>0.15$).

The proportion of type 1 fibers was $47 \pm 4\%$ of all, and the proportion thus varied little between the seven subjects. There was no apparent relationship between the proportion of type 1 fibers, PFK's catalytic activity, or the observed glycolytic flux during the 10 s sprint.

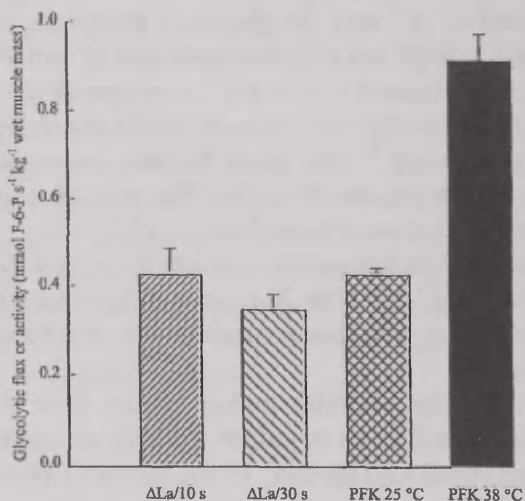


Figure 1. Comparison of the glycolytic flux and the catalytic activity of phosphofructokinase. The bars are (left to right) the mean rate of lactate production during 10 and 30 s exhausting bicycling, and the catalytic activity of phosphofructokinase at 25 and 38°C . All entities are expressed in units of fructose-6-phosphate. The data are mean \pm SEM of seven subjects.

DISCUSSION

The main finding in this study was that there was no sign of limited rates of lactate production as judged from the high phosphofructokinase activity relative to the rate of lactate production during the 10 and 30 s bicycle sprints as well as the high phosphocreatine and ATP reserves at exhaustion.

Inadequate supply of ATP can cause fatigue, but our data do not support that this has caused fatigue in our experiments. First, the ATP-concentration fell only moderately during exercise, suggesting that the muscle cells were able to regenerate ATP as fast as it was broken down. Second, there was a considerable phosphocreatine reserve at exhaustion that could produce more ATP if broken down. Third, the glycolytic flux was higher during the 10 s than during the 30 s sprint, suggesting that no limit in the glycolytic rate was met, at least not during the 30 s exercise. Fourth, phosphofructokinase's catalytic activity, the assumed rate limiting enzyme of the glycolysis, was more than twice the observed glycolytic flux during the 10 s sprint and nearly three times the flux during the 30 s exercise. It could be that other enzymes, for example glycogen phosphorylase, could limit the glycogenolysis. However, we found an accumulation of glucose-6-phosphate, and as pointed out by others [4], there is an accumulation of other metabolites upstream of phosphofructokinase but little accumulation of triose phosphates downstream of this enzyme, suggesting that phosphofructokinase's activity limits the glycolytic rate during exercise. Thus, there is a number of reasons to assume that the muscle cell's ability to re-synthesize ATP was not a limiting factor in our study.

It could be that in real life there is a regulation of phosphofructokinase's activity that is absent in the test tube, resulting in higher rates in the test tube than in live muscles. If so, we might have overestimated the maximum activity of our subjects. However, we are not aware of any evidence supporting that possibility. On the other hand, Peters and Spriet [20] found that there is a negative effect of diluting the sample, and they proposed that the catalytic activities of PFK measured in test tubes underestimates the value in live muscles. That conclusion is in line with several former studies.

First, Mansour [12] showed that the enzyme exists in either an inactive, low molecular-mass form (a dimer) or an active, high molecular-mass form (probably a tetramer) as judged from the sedimentation rates. Since most assays in the test tubes are carried out at much lower concentrations than in real life, test tube experiments probably to a large extent measure the low-mass form, while in real life the high-mass form dominates. Bosca *et al.* [3] showed that the concentrated enzyme (that is the active tetramer) was not inhibited by ATP in the physiological range of concentration, while their diluted enzyme (dimer form) was. In line with this Dobson *et al.* [5] who showed that if fructose-2,6-diphosphate is present, pH has little effect on the activity of PFK in the physiologic range, and 5 mmol L⁻¹ ATP did not inhibit the enzyme. Thus, there is little reason to assume that we have overestimated the true catalytic activity of PFK in our subjects' muscles.

We did not include release of lactate from muscle to blood during exercise in our calculations. The error is quite small even for exercise lasting several minutes [13, 17, Medbø *et al.*, submitted], and for exercise lasting some seconds there may be little time for releasing lactate. Our low blood lactate concentrations at the end of the exercises support that conclusion. Our calculations of the glycolytic flux also disregard accumulation of pyruvate and triose phosphates. As pointed out above the concentration of these muscle metabolites is low even after exercise [4, 18], and the error introduced is therefore of little importance.

The muscle metabolites and catalytic activities are measured in muscle biopsies containing mixtures of both fiber types, and our reported values do not reflect possible differences between the two main muscle fiber types. Our subjects appeared to have nearly equal proportions of each of the two main fiber types, which is typical for this muscle. Blomstrand and colleagues measured the phosphofructokinase activity in the knee extensors of untrained and trained subjects and reported values between 0.35 mkat × kg⁻¹ wet muscle mass for their welltrained subjects and 0.55 mkat × kg⁻¹ for their untrained subjects at 25°C [1]. Our observed values are thus similar to the values of their subjects.

The reported glycolytic flux is an average over a ≈ 12 s period. If there was a delay from the onset of exercise to the onset of glycolysis, the flux near the end would be higher than the reported value while the average value for shorter exercise periods would be less. This is most likely not a big problem. First, if there was a delay, phosphocreatine breakdown would have to cover the ATP-turnover during that period. Since lactate production provided more than twice as much ATP as phosphocreatine breakdown did and since there was a considerable aerobic energy release, a possible delay can at most be a second or two [15]. Second, Hirvonen et al. [8] reported an average rate of lactate production of $0.8 \text{ mmol} \times \text{s}^{-1} \times \text{kg}^{-1}$ wet muscle mass during a 40 m sprint lasting around 5 s. That value is similar to our value for the 10 s sprint. Third, during 2 min exercise to exhaustion lactate seems to be produced at a constant rate [14], further suggesting that there is no important delay in the lactate production at the onset of exercise.

Our subjects exercised at very high powers for 12 s. Higher powers can be maintained for 3–5 s [4, 9 18]. Separate tests on our subjects suggest that they were able to develop a 10–20% higher power for a 3–5 s period than that found for the 10 s sprints (unpublished observations). Since the glycolytic flux seems roughly proportional to the power, it is conceivable that even during these very intense bursts of exercise the glycolytic rate would probably not be more than around half of PFK's catalytic activity. In line with this Gaitanos et al. [7] reported a mean power during 6 s bicycling of $12.5 \text{ W} \times \text{kg}^{-1}$ and an anaerobic ATP turnover rate of $14 \text{ mmol} \times \text{s}^{-1} \times \text{kg}^{-1}$ dry muscle mass or $3.2 \text{ mmol} \times \text{s}^{-1} \times \text{kg}^{-1}$ wet muscle mass.

It is often held that enzymatic systems may reach their maximum rate in real life and thus limit the metabolic flux. This did not seem to be the case for phosphofructokinase even in our extreme situation. We have recently suggested that in healthy humans the Na,K pump's maximum power is never reached even during extreme exercises [6, Medbø *et al.*, in review]. Other factors than activities of enzymes producing ATP may limit the performance even when the metabolic fluxes are very high. It could be that the muscle's ability to use ATP, for example the myosin ATP-ase activity,

limits the performance. We are not aware of any data on the myosin ATP-ase activity in human muscle that can test this hypothesis.

To sum up, the glycolytic flux does not seem to be limited by phosphofructokinase's catalytic activity even during a 10 s sprint. A limited supply of energy in the form of ATP may therefore not be the limiting factor during this kind of exercise.

ACKNOWLEDGEMENTS

We are grateful to Bo Veiersted for medical assistance during the experiments, and to Astrid Bolling, Per Holmstad, Ada Ingvaldsen, Bjørg I. Selberg, and Jorid T. Stuenæs for their technical assistance.

REFERENCES

1. **Blomstrand E., Ekblom B., Newsholme E. A.** (1986) Maximum activities of key glycolytic and oxidative enzymes in human muscle from differently trained individuals. *J. Physiol.* 381: 111–118.
2. **Bogdanis G. C., Nevill M. E., Lakomy H. K. A.** (1994) Effects of previous dynamic exercise on power output during repeated maximal sprint cycling. *J. Sport Sci.* 12: 363–370.
3. **Boscá L., Aragón J. J., Sols A.** (1985) Modulation of muscle phosphofructokinase at physiological concentrations of enzyme. *J. Biol. Chem.* 260: 2100–2107.
4. **Cheetham M. E., Boobis L. H., Brooks S., Williams C.** (1986). Human muscle metabolism during sprinting. *J. Appl. Physiol.* 61: 54–60.
5. **Dobson G. P., Yamamoto E., Hochachka P. W.** (1986) Phosphofructokinase control in muscle: nature and reversal of pH-dependent ATP inhibition. *Am. J. Physiol.* 250: R71–R76.
6. **Evertsen F., Medbø J. I., Jebens E., Nicolaysen K.** (1997) Hard endurance-training increases the Na,K pump concentration in elite cross-country skiers. *Am. J. Physiol.* 272: R1417–R1424.

7. **Gaitanos G. C., Williams C., Boobis L. H., Brooks S.** (1993) Human muscle metabolism during intermittent maximal exercise. *J. Appl. Physiol.* 75: 712–719.
8. **Hirvonen J., Rehunen S., Rusko H., Härkönen M.** (1987) Break-down of high-energy phosphate compounds and lactate accumulation during short supramaximal exercise. *Eur. J. Appl. Physiol.* 56: 253–259.
9. **Jones N. L., McCartney N., Graham T., Spriet L. L., Kowalchuk J. M., Heigenhauser G. J. F., Sutton J. R.** (1985) Muscle performance and metabolism in maximal isokinetic cycling at slow and fast speeds. *J. Appl. Physiol.* 59: 132–136.
10. **Krogh A.** (1913) A bicycle ergometer and respiration apparatus for the experimental study of muscular work. *Skand. Archiv Physiol.* 30: 375–394.
11. **Lundin A., Rickardsson A., Thore A.** (1976) Continuous monitoring of ATP-converting reactions by purified firefly luciferase. *Analyt. Biochem.* 75: 611–620.
12. **Mansour T. E.** (1970) Kinetic and physical properties of phosphofructokinase. *Adv. Enzyme Regul.* 8: 37–51.
13. **Medbø J. I.** (1993) Glycogen breakdown and lactate accumulation during high intensity cycling. *Acta Physiol. Scand.* 149: 85–89.
14. **Medbø J. I., Gramvik P., Jebens E.** (1996) Anaerobic energy release during 2 min bicycling to exhaustion. *The Physiologist* 39: A-82.
15. **Medbø J. I., Gramvik P., Jebens E.** (1999) Aerobic and anaerobic energy release during 10 s and 30 s bicycle sprints. *Acta Kinesiol. Univ. Tartuensis* 4: 122–146.
16. **Medbø J. I., Tabata I.** (1993) Anaerobic energy release in working muscle during 30 s to 3 min exhausting bicycling. *J. Appl. Physiol.* 75: 1654–1660.
17. **Medbø J. I., Tabata I., Mohn A.-C., Gramvik P., Jebens E.** (1998) Does blood lactate measure the anaerobic energy release during intense exercise? *Acta Physiol. Scand.* 163: A10–A11.
18. **Nevill M. E., Boobis L. E., Brooks S., Williams C.** (1989) Effect of training on muscle metabolism during treadmill sprinting. *J. Appl. Physiol.* 67: 2376–2382.
19. **Passoneau J. V., Lowry O. H.** (1993) *Enzymatic Analysis. A Practical Guide.* Humana Press, Totowa, New Jersey, USA. pp 188–193, 252–254, 292–293.

20. **Peters S. J., Spriet L. L.** (1995) Skeletal muscle phosphofructokinase activity examined under physiological conditions in vitro. *J. Appl. Physiol.* 78: 1853–1858.
21. **Taylor H. L., Buskirk E., Henschel A.** (1955) Maximal oxygen intake as an objective measure of cardio-respiratory performance. *J. Appl. Physiol.* 8: 73–80.
22. **Vøllestad N. K., Vaage O., Hermansen L.** (1984) Muscle glycogen depletion patterns in type I and subgroups of type II fibres during prolonged severe exercise in man. *Acta Physiol. Scand.* 122: 433–441.
23. **Withers R. T., Sherman W. M., Clark D. G., Esselbach P. C., Notan S. R., Mackay M. H., Brinkman M.** (1991) Muscle metabolism during 30, 60 and 90 s of maximal bicycling on an air-braked ergometer. *Eur. J. Appl. Physiol.* 63: 354–362.

Correspondence to:

Jon Ingulf Medbø
National Institute of Occupational Health
PO Box 8149 dep.
N-0033-8149 Oslo
Norway

AGE-RELATED DIFFERENCES IN VERTICAL JUMPING PERFORMANCE IN WOMEN

M. Pääsuke, J. Ereline, H. Gapeyeva, P. Sander

Institute of Exercise Biology, University of Tartu, Tartu, Estonia

ABSTRACT

This study compared vertical jumping performance characteristics in young (20–23-year-old), middle-aged (52–58-year-old) and elderly (66–77-year-old) women. Two types of maximal vertical jumps were performed on force platform: squat jumps (SJ) from the static semi-squatting position and counter-movement jumps (CMJ) from the standing position. The jumping height, vertical velocity of take-off and mechanical power output of the whole body during take-off both in SJ and CMJ was significantly greater in the young women compared to the older groups, and in the middle aged women greater compared to the elderly women. The jumping height, vertical velocity of take-off and mechanical power output of the whole body during take-off in CMJ were greater compared to SJ only in young women. The young women had significantly greater absolute and body mass-related values of peak vertical ground reaction force in SJ than the other groups while the differences between the middle-aged and the elderly women were not significant. This study indicated that middle-aged and elderly women have a reduced ability to use potentiating effect of the stretch-shortening cycle to vertical jumping performance during counter-movement jump.

Key words: vertical jumping performance, leg extensor muscles, aging, women

INTRODUCTION

Muscle strength and the ability of the leg extensor muscles to develop force rapidly (explosive strength) are important performance characteristics which have been shown to contribute to several of the tasks of daily life as climbing stairs, walking or even the prevention of falls and trips [3, 13]. Aging is known to be associated with decreased maximal strength and explosive force production, especially at the onset of the sixth decade [12, 13, 22, 27].

Age-related decreases in maximal and explosive strength are associated with decline in muscle mass thought to be mediated by a reduction in the size and a loss of individual muscle fibres, especially of fast twitch fibres [18, 19]. Moreover, maximal voluntary activation of the agonist muscles and/or changes in the degree of agonist/antagonist co-activation may also differ in relation to age depending on the type of motion and the time or velocity characteristics of actions [12].

The vertical jumps can be used as a model to study explosive force-generating capacity of the leg extensor muscles. It has been shown that explosive muscle strength characteristics may decline with increasing age even more than maximal strength [5, 17]. However, the most of these measures are performed in male subjects.

Natural dynamic human locomotion is characterized by stretch-shortening cycle (SSC) actions, in which an eccentric muscle action precedes a concentric muscle action. It is well known that the force outcome in the maximal SSC actions is greater than force production during a concentric muscle action [2, 14, 15]. This is true also in aging subjects, although some studies have reported the mechanical performance enhancement after prestretching is somewhat diminished with aging [5], while some others have shown no age-related differences in performance enhancement with SSC [25]. Vertical jump height has been used as a standard measure to assess the efficiency of SSC. It has been shown that vertical jumps preceded by an eccentric contraction (counter-movement and drop jump) result in greater vertical jump heights [2, 24]. However, age-

related differences in the ability to use SSC in vertical jumping in women are poorly understood.

The purpose of this study was to compare the vertical jumping performance in young, middle-aged and elderly women. More specifically, we were interested in examining the biomechanical characteristics of squat and counter-movement jump, i.e. jump without and with SSC.

MATERIAL AND METHODS

Subjects

The total of thirty seven female subjects agreed to participate in the present study. The subjects were distributed into three age groups: young (20–23 year old, $n=13$), middle-aged (52–58 year old, $n=11$) and elderly (66–77 year old, $n=13$). The subjects were screened by questionnaire to exclude those with diagnosed musculoskeletal disorders. All the subjects were informed of the procedures to be utilized as well as the purpose of the study and their written informed consent was obtained. The study carried the approval of the University Ethics Committee. The physical characteristics of the subjects are presented in Table 1.

Table 1. The physical characteristics of the subjects. Mean \pm SEM.

Variables	Age groups		
	Young women ($n=13$)	Middle-aged women ($n=11$)	Elderly women ($n=13$)
Age (years)	20.6 \pm 0.2	55.5 \pm 0.8	70.8 \pm 0.8
Height (cm)	169.5 \pm 1.3	164.6 \pm 1.2	158.8 \pm 1.4
Body mass (kg)	60.5 \pm 1.7	70.6 \pm 1.3	65.6 \pm 2.1

Note: all variables were statistically significant ($p<0.05$) among the age groups.

Apparatus and Experimental Procedure

The vertical jumping tests were performed on force platform (PD-3A, Visti, Russia) with the measures of 0.75×0.75 m and natural frequency of 150 Hz. Two types of maximal vertical jumps were performed: squat jumps and counter-movement jumps. Squat jump (SJ) was begun from a static semi-squatting position with a knee angle of 90° of the knee flexion, followed by subsequent action, during which the leg and hip extensor muscles contracted concentrically. Counter-movement jump (CMJ) started from the upright standing position immediately after a fast preparatory counter-movement that stretched the leg extensor muscles (eccentric contraction). This was followed by an explosive maximal extension in the opposite direction (concentric contraction). The subjects were instructed to jump with their hands on the hips to eliminate the influence of the arm swing impulse. The movement amplitude of the knee joint during each jump was measured with electrogoniometer (Elgon) attached to the lateral side of the subject's right knee. Twenty-four to 48 hours before data collection the subjects were given instructions and the vertical jump tests were demonstrated. This was followed by a practice session to familiarize the subjects with the procedures. Prior to testing, each subject underwent a ten-minute warming-up period, that consisted of submaximal leg ergometry followed by stretch exercises, and the subjects performed several preliminary trials. The testing jumps had to be performed reactively with maximal effort. The following characteristics were recorded by a vertical force-time curve for each jump [2]:

Jumping height by the height of rise of the body centre of gravity:

$$H = g t_f^2 / 8,$$

where t_f^2 is flight time.

Peak vertical ground reaction force ($R_{v, \text{peak}}$).

Vertical velocity of take-off: $V_v = g t_f / 2$, where g is acceleration of gravity (9.81 m/s^2) and t_f flight time.

Power output during take-off: $P = W t_f^2 / 8T$, where W is body weight of the subjects, t_f flight time and T time of vertical ground reaction force production.

Three maximal SJ and CMJ were recorded and the trial with best jumping height in both cases was used for further analysis. A rest period of 1 min was allowed between the trials. The peak vertical ground reaction force in relation to body mass was calculated for both types of jumps.

Data Analysis

Data are means and standard errors of the mean (\pm SEM). One-way analysis of variance (ANOVA) following by Tukey post hoc comparisons were used to test for differences between age groups and for different types of vertical jumps. A level of $p < 0.05$ was selected to indicate statistical significance.

RESULTS

The young women had significantly greater height and less body mass than other groups, while the middle-aged women had significantly greater height and body mass than the elderly women (Table 1).

The jumping height of both SJ and CMJ was significantly greater in the young women compared to other groups, and in the middle-aged women greater compared to the elderly women (Figure 1A). The jumping height in CMJ was significantly greater compared to SJ in the young women, however, no significant differences in jumping height between CMJ and SJ in the middle-aged and elderly women were observed.

The young women had significantly greater peak vertical ground reaction force in SJ than the other groups while the differences between the middle-aged and the elderly women were not significant (Figure 1B). The young women had also significantly greater peak

vertical ground reaction force in CMJ than the middle-aged women, while the differences between the young and elderly women, and the middle-aged and elderly women were not significant.

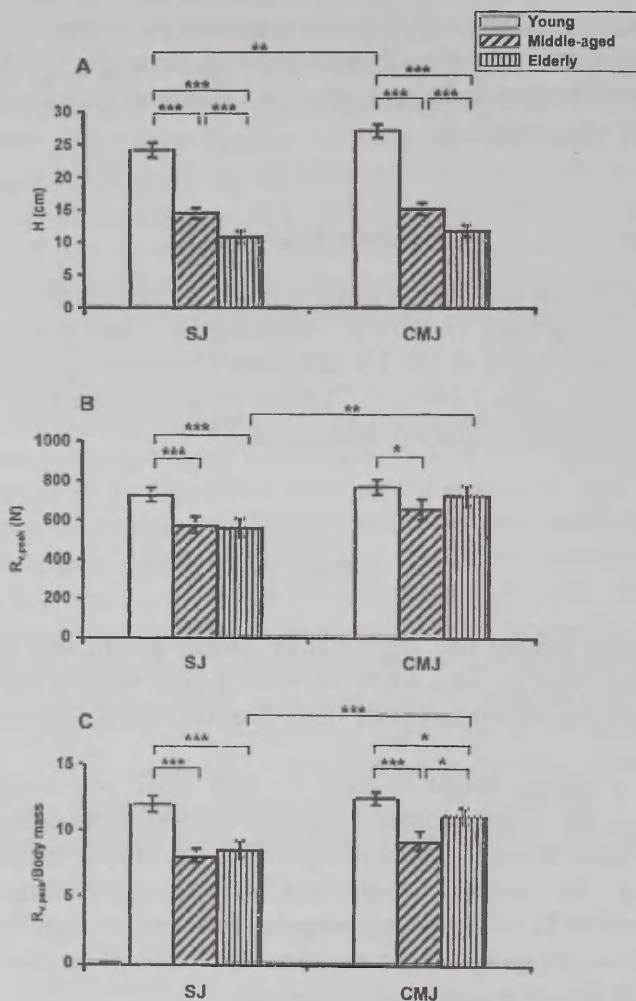


Figure 1. Mean (\pm SEM) jumping height (H) **A**, peak vertical ground reaction force ($R_{v,peak}$) **B** and ground reaction force relative to body mass ($R_{v,peak}/\text{Body mass}$) **C** in squat jump (SJ) and counter-movement jump (CMJ) in women at different age. *p<0.05; **p<0.01; ***p<0.001

The peak vertical ground reaction force in SJ in relation to body mass was significantly greater in the young women compared to the older groups, while differences between the middle-aged and elderly women were not significant (Figure 1C). The peak vertical ground reaction force in CMJ in relation to body mass differed between the groups so that the young women had greater values than two older groups, and the elderly women had greater values than the middle-aged women. In the elderly women, the peak vertical ground reaction force in relation to body mass was significantly greater in CMJ compared to SJ.

The young women had significantly greater vertical velocity of take-off (Figure 2A) and mechanical power output of the whole body during take-off (Figure 2B) both in SJ and CMJ compared to

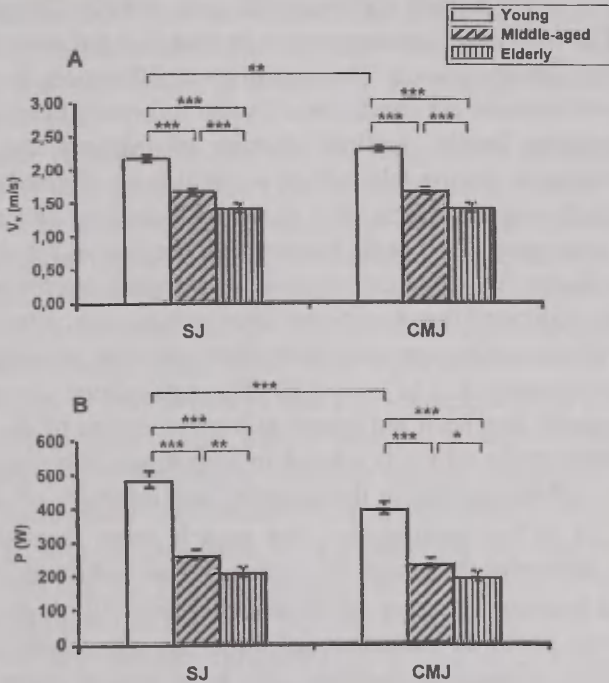


Figure 2. Mean (\pm SEM) vertical velocity of take-off (V_v) **A** and power output during take-off (P) **B** in squat jump (SJ) and counter-movement jump (CMJ) in women at different age. *p<0.05; **p<0.01; ***p<0.001

the older groups, and the middle-aged women's respective values were greater compared to the elderly women. The vertical velocity of take-off and mechanical power output during take-off in CMJ were greater than in SJ in the young women, while no significant differences in these characteristics between CMJ and SJ were observed in the middle-aged and elderly women.

DISCUSSION

This study indicated markedly greater jumping height, vertical velocity of take-off and mechanical power output of the whole body during take-off both in SJ and CMJ in young (20-year-old) women compared to middle-aged (50-year-old) and elderly (70-year-old) women. The mentioned characteristics in middle-aged women were greater than elderly women. The age-related differences in vertical jumping performance are conditioned by the following factors.

The jumping height, vertical velocity of take-off and power output production during take-off of vertical jump depends on the physiological process which take place in muscular and nervous systems with aging. It is well known that explosive force is one important factor limiting performance in jumping exercises. Several studies indicated that explosive force production capacity may decline with increasing age even more than maximal strength of the same muscle group [5, 11, 13, 17]. The age-related decrease in muscle strength has been attributed to a great extent to the reduction in muscle mass which is related to alterations in hormone balance [10], and the decline in the quantity and intensity of physical activity [21]. It has been shown that muscle mass is reduced by about one third over the age of 50, with a further reduction of about 15 percent between the ages of 70 and 80 years [9]. A decline in muscle mass could be indicative of either an age-related loss of muscle fibres, a decrease in fibre size, or a loss of entire motor units [1]. Lexell *et al.* [19] indicated that in subjects aged between

20 and 80 years in addition to a loss of muscle fibres, aging atrophy may be caused by a reduction in fast-twitch fibre size.

An important factor is intramuscular co-ordination and co-activation of activity of the agonist-antagonist muscles involved in performing jump [23]. Age-related changes in the spinal cord have been shown by electrophysiological and morphological methods. A decline in the numbers of myelinated fibres in human spinal roots [8] and in peripheral nerves [26], and progressive loss of motoneurons [7] with increasing age has been indicated. Demyelination of spinal and peripheral nerves may result in denervation of some muscle fibres with subsequent atrophy and weakness of the motor responses. A selective loss of large motoneurons which innervate fast-twitch muscle fibres would be correlated with a loss of explosive strength, since these fibres are recruited during rapid force production [16]. Thus, the age-related decrease in vertical jumping performance in women can be partly explained by decline in the ability for rapid neural activation of the extensor muscles of lower extremities.

In the present study the vertical jumps were performed without (SJ) and with preliminary counter-movement. Counter-movement jump is an exercise characterized by the so-called SSC, in which the action of muscles during the eccentric phase influences the subsequent concentric phase [2, 24]. Several mechanisms have been proposed to explain the positive effect of a counter-movement on the performance in the subsequent concentric action: (1) it allows for storage of elastic energy that can subsequently be re-utilized [2, 6], (2) it triggers spinal stretch reflexes as well as longer-latency responses [20] that help to increase muscle stimulation during the concentric phase, (3) it allows for greater joint movements at the start of push-off and more work production [4]. The present study indicated that the jumping height, vertical velocity of take-off, mechanical power and work performed by whole body during take-off in CMJ were greater compared to SJ only in young women. No significant differences in jumping height and biomechanical characteristics of take-off between SJ and CMJ were found either in middle-aged and elderly women. This study indicated, conse-

quently, that middle-aged or elderly female subjects cannot effectively use potentating effect of counter-movement (and SSC) to vertical jumping performance

In summary, the present results are in line with the earlier finding that the capacity for explosive force production of the leg extensor muscles declines drastically with increasing age. Middle-aged and elderly women showed a reduced ability to use the potentating effect of SSC to vertical jumping performance during counter-movement jump.

REFERENCES

1. **Aniansson A., Grimby G., Hedberg M., Krotkiewski M.** (1981) Muscle morphology, enzyme activity and muscle strength in elderly men and women. *Clin. Physiol.* 1: 73–86.
2. **Asmussen E., Bonde-Petersen F.** (1974) Storage of elastic energy in skeletal muscles in man. *Acta Physiol. Scand.* 91: 385–392.
3. **Bassey E. J., Fiatarone M. A., O'Neil E. F., Kelly M., Evans W. J., Lipsitz L. A.** (1992) Leg extension power and functional performance in very old men and women. *Clin. Sci.* 82: 321–327.
4. **Bobbert M. F., Gerritsen K. G., Litjens M. C. A., van Soest A. J.** (1996) Why is countermovement jump height greater than squat jump height. *Med. Sci. Sports Exerc.* 28: 1402–1412.
5. **Bosco C., Komi P. V.** (1980) Influence of aging on the mechanical behaviour of leg extensor muscles. *Eur. J. Appl. Physiol.* 45: 209–215.
6. **Bosco C., Ito A., Komi P. V., Luhtanen P., Rahkila P., Rusko H., Viitasalo J.** (1982) Neuromuscular function and mechanical efficiency of human leg extensor muscles during jumping exercises. *Acta Physiol. Scand.* 114: 543–550.
7. **Brown W. F., Strong M. J., Snow R.** (1988) Methods for estimating numbers of motor units in biceps-brachialis muscles and losses of motor units with aging. *Muscle Nerve* 11: 423–432.
8. **Gardner E.** (1940) Decrease in human neurones with age. *Anat. Rec.* 77: 529–536.
9. **Grimby G., Saltin B.** (1983). The aging muscle. *Clin. Physiol.* 3: 209–218.

10. **Häkkinen K., Pakarinen A.** (1993) Muscle strength and serum testosterone, cortisol and SHBG concentrations on middle-aged and elderly men and women. *Acta Physiol. Scand.* 148: 199–207.
11. **Häkkinen K., Pastinen U.-M., Karsikas R., Linnamo V.** (1995) Neuromuscular performance in voluntary bilateral and unilateral contractions and during electrical stimulation in men at different ages. *Eur. J. Appl. Physiol.* 70: 518–527.
12. **Häkkinen K., Alen M., Kallinen M., Izquierdo M., Jokelainen K., Lassila H., Mälkiä E., Kraemer W. J., Newton R.U.** (1998) Muscle cross-sectional area, force production, and neural activation of leg extensor muscles during isometric and dynamic actions in middle-aged and elderly men and women. *J. Aging Phys. Activity* 6: 232–247.
13. **Izquierdo M., Aguado X., Gonzalez R., Lopez J. L., Häkkinen K.** (1999) Maximal and explosive force production capacity and balance performance in men of different ages. *Eur. J. Appl. Physiol.* 79: 260–267.
14. **Komi P. V.** (1984) Physiological and biomechanical correlates of muscle function: Effects of muscle structure and stretch-shortening cycle. *Exerc. Sport Sci. Rev.* 12: 81–121.
15. **Komi P. V., Bosco C.** (1978) Utilization of stored elastic energy in leg extensor muscles by men and women. *Med. Sci. Sports* 10: 261–265.
16. **Larsson L.** (1982) Physical training effects on muscle morphology in sedentary males at different ages. *Med. Sci. Sports Exerc.* 14: 203–206.
17. **Larsson L., Grimby G., Karlsson J.** (1979) Muscle strength and speed of movement in relation to age and muscle morphology. *J. Appl. Physiol.* 46: 451–456.
18. **Lexell J., Henriksson-Larsen K., Winblad B., Sjöström M.** (1983) Distribution of different fiber types in human skeletal muscles. 3. Effects of aging studied in whole muscle cross sections. *Muscle Nerve* 6: 588–595.
19. **Lexell J., Taylor C., Sjöström M.** (1988) What is the cause of the ageing atrophy? *J. Neurol. Sci.* 84: 275–294.
20. **Melvill-Jones G., Watt D. G. D.** (1971) Observations on the control of stepping and hopping movements in man. *J. Physiol. (Lond.)* 219: 709–727.
21. **Mälkiä E., Impivaara O., Heliövaara M., Maatela J.** (1994) The physical activity of healthy and cronicly ill adults in Finland at

work, at leisure and during commuting. *Scand. J. Med. Sci. Sports* 4: 82–87.

22. **Narici M., Bordini M., Cerretelli P.** (1991) Effect of aging on human adductor pollicis muscle function. *J. Appl. Physiol.* 71: 1227–1281.
23. **Rack P. M. H., Westbury D. R.** (1974) The short range stiffness of active mammalian muscle and its effect on mechanical properties. *J. Physiol. (Lond.)* 240: 331–350.
24. **Riera J., Drobnic F., Galilea P. A., Pons V.** (1994) Comparison of two methods for the measurement of the extensor muscle dynamic force of the inferior limb: isokinetic dynamometry and vertical jump tests. *Sports. Med. Training Rehabil.* 5: 37–43.
25. **Svantesson U., Grimby G.** (1995) Stretch-shortening cycle during plantar flexion in young and elderly women and men. *Eur. J. Appl. Physiol.* 71: 381–385.
26. **Swallow M.** (1966) Fibre size and content of anterior tibial nerve of the foot. *J. Neurol. Neurosurg. Psychiat.* 29: 205–213.
27. **Vandervoort A. A., McComas, A. J.** (1986) Contractile changes in opposing muscles of the human ankle joint with aging. *J. Appl. Physiol.* 61: 361–367.

Correspondence to:

Mati Pääsuke
Institute of Exercise Biology
University of Tartu
5 Jakobi Str.
Tartu 51014
Estonia

EFFECTS OF A HEALTH-RELATED PHYSICAL EDUCATION CLASSES ON PHYSICAL ACTIVITY AND FITNESS IN ELEMENTARY SCHOOLCHILDREN

L. Raudsepp, P. Päll

Institute of Sport Pedagogy, University of Tartu, Tartu, Estonia

ABSTRACT

This study evaluated effects of intensified physical education lessons for 42 first and second grade students on health-related fitness and physical activity during lessons. Two schools were assigned to two conditions (intensified or health-related and usual programs). During two school years, intensified physical education classes were taught by physical education specialist and control classes were taught by classroom teachers. Measures of health-related fitness and heart rate data during lessons were collected. Mean heart rates during intensified physical education classes (157.2 ± 17.7 bpm) were significantly higher when compared to heart rate of control class students (134.8 ± 15.6 bpm, $p < 0.05$). After 2 years, students from intensified physical education classes were superior to students in control condition on agility, speed, cardiovascular endurance, functional and explosive strength ($p < 0.05$). Our results suggest that intensified or health-related elementary classes have had a significant positive effect on health-related fitness and physical activity during lessons.

Key words: elementary physical education, physical activity, health-related fitness

INTRODUCTION

It is widely acknowledged that children and youth need regular physical activity for normal growth and development, maintenance of good health and fitness, and development of physical activity skills and behaviors that will carry into adulthood [19, 21]. School physical education have many important goals, such as the development of motor skills, creative and artistic expression, self-realization and social development [3]. However, an important function of school physical education is to engage students in moderate-to-vigorous physical activity (MVPA), a requirement for experiencing both health and motor skill development benefits [11, 19]. Moreover, children's cardiorespiratory fitness will only be promoted in physical education lessons if they spent an appropriate amount of time in MVPA. When considering the frequency of MVPA, it is clear that the majority of physical education curricula do not have the frequency of lessons (three per week) to increase cardiorespiratory fitness in children [20].

There is general agreement on how physical education programs can be improved. Classes need to be taught more frequently and by more qualified instructors [1, 9, 15]. However, the frequency of physical education lessons and the amount of moderate-to-vigorous physical activity children obtain during physical education may be less than recommended [21]. Many physical education programs are available and the dominant curricula have emphasized skill-related fitness of movement education, though developmental, humanistic, and personal meaning curricula have been promoted [7]. In the past 10 to 15 years, the emphasis has begun to shift to the effects of physical education on the health-related physical activity and fitness, as the important health effects of physical education have been documented [2, 8, 11]. Several studies have demonstrated that health-related physical education programs in elementary school can increase physical activity during class and improve cardiovascular fitness [5, 9, 17, 18]. Sallis *et al.* [17] evaluated the effects of a health-related physical education program on physical activity and fitness in four- and fifth-grade students. It was concluded that health-related physical education

program can provide students with substantially more physical activity during physical education classes. Simons-Morton *et al.* [20] revealed that modification of the school physical education program increased the time children engaged in moderate-to-vigorous physical activity from less than 10% of class time at baseline to about 40% of the class time at posttest. In addition, Shephard and Lavallee [18] concluded that the enhanced physical education program (one hour of required physical education daily) improved several indices of physical performance in elementary schoolchildren.

In Estonia, like in many other countries, classroom teachers are responsible for teaching elementary physical education classes. However, classroom teachers are frequently untrained to conduct quality physical education in which lessons are likely to produce optimal skill gains and sufficient MVPA level of children [9]. Several studies have shown that physical education specialists usually provide better physical education than nonspecialists both in process and outcome measures [5, 10]. Results also show that curriculum-based interventions, such as the CATCH and SPARK programs (coupled with in-service training for classroom teachers) can substantially increase students' physical activity levels [8, 17].

Previous studies with elementary schoolchildren have mainly examined the effects of different physical education programs on measures of physical fitness or physical activity separately. The present study, however, focuses on the effects of intensified or health-related physical education on both physical activity during classes and health-related physical fitness of elementary school children.

METHODS

Setting and schools

Two elementary schools were randomly assigned to either health-related physical education program (experimental) or usual program (control) conditions. Two first grade classes from the experi-

mental and two from the control conditions were recruited to the study. Forty two students remained for the entire period of measurements from initial forty eight.

Experimental program. During 2 year period, children in the experimental group received 3 h of physical education each week, under the supervision of professional physical educator. The program aimed at maximizing physical activity of all students during the assigned time, with the intent of increasing each child's aerobic and muscular capacity. Activities were carefully choosed according to the motor development of the children and included indoor and outdoor activities (track and field, various games etc.). Children from the control group received only the standard physical education program for primary students and lessons (3 times per week) were supervised by classroom teacher. At the start of the program, teacher conducting experimental classes had 4 year and teacher supervising control classes 11 years teaching experiences.

Physical fitness

Four test from EUROFIT test battery were used to evaluate the physical fitness of children: standing long jump, 10x5 m shuttle run, bent arm hang and sit ups. 3-minute run according to Kaneko and Fushimoto [6] was used to measure the cardiovascular endurance of the students. Four measurement sessions were carried out in september 1997, april 1998, september 1998 and april 1999 during one-week school period. All measurements were taken by 3 experienced instructors.

Heart rate monitoring

Heart rate of every child was measured during health-related and usual physical education classes (control class) in two testing sessions (september 1997 and april 1998). Heart rates were recorded with Sport tester (Polar Electro, Finland) at 15 sec intervals. The device consisted of a sensing/transmitter unit strapped to the chest and a receiver/memory unit worn on the wrist. The data were transmitted to the computer using a special interface. The following heart rate variables were calculated: mean heart rate during

physical education classes (MHR), mean % of MVPA (150 bpm) during classes.

Statistical analysis

Data were analyzed using repeated measures analysis of variance (ANOVA). For each change measure, an effect size was calculated to quantify the gains achieved by the experimental students relative to controls. Effect size was the difference between two group means, divided by the underlying standard deviation. Effect sizes greater than 0.4 are considered large; 0.3 is moderate; and 0.1 is small [4]. Significance was set at $p < 0.05$.

RESULTS

Descriptive data of the subjects at baseline and effect sizes after two-year intervention are presented in Table 1. Multivariate ANOVA revealed no differences on physical fitness and anthropometrical measures at baseline between two classes.

Table 1. Descriptive baseline data and effect sizes for experimental and control classes.

Measures	Experimental (n=20)		Control (n= 22)		p	Effect Size vs. Control
	x	SD	x	SD		
Age (yrs)	7.2	0.4	7.1	0.3	ns	0.122
Height (cm)	133.6	6.4	134.5	5.6	ns	0.217
Weight (kg)	29.3	4.2	29.7	3.8	ns	0.228
10x5 m shuttle run (sec)	21.7	1.1	22.0	1.2	ns	0.232
3 minute run (m)	412.4	19.6	415.3	18.4	ns	0.344
Bent arm hang (sec)	7.8	3.2	7.3	2.7	ns	0.217
Standing long jump (cm)	132.5	10.6	133.2	9.5	ns	0.222

ns — not significant ($p > 0.05$)

The effects of condition on health-related fitness measurements after intervention period were medium.

Figure 1 shows changes in 10×5 m shuttle run across four measurement sessions. The results of the 4×2 ANOVA with the factors of age and condition revealed statistically significant differences between teaching conditions on 2nd, 3rd and 4th measurement session. Figure 2 presents changes in aerobic fitness across measurements. Significant differences between two classes are apparent in 3rd ja 4th measurements. Changes in bent arm hang are presented in Figure 3. Multivariate ANOVA revealed significant differences between two groups in measurement sessions 3 and 4. Figure 4. shows changes in explosive strength across measurements. Results revealed higher result of experimental group in second year measurements. Heart rate data (Figure 5) shows a significantly higher mean HR of children during experimental lessons.

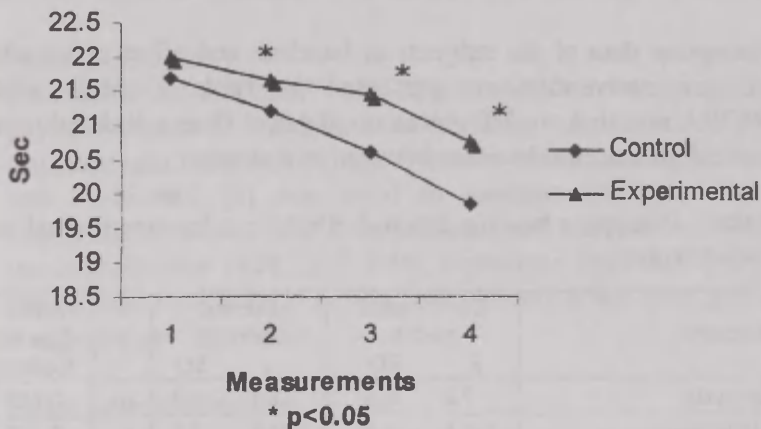


Figure 1. Changes in 10×5 m shuttle run.

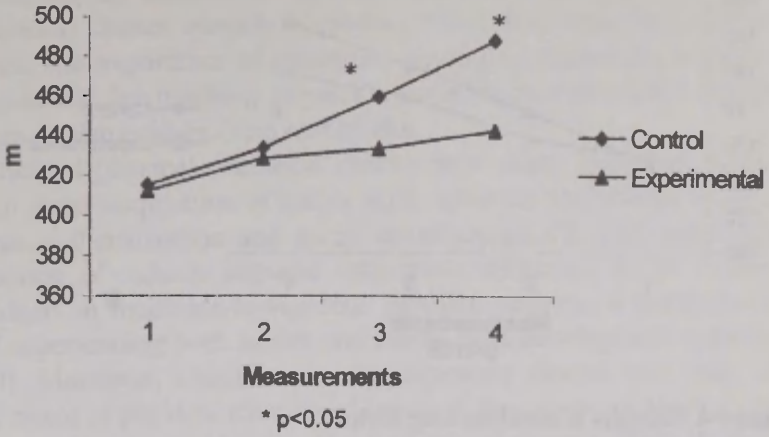


Figure 2. Changes in 3 minute run.

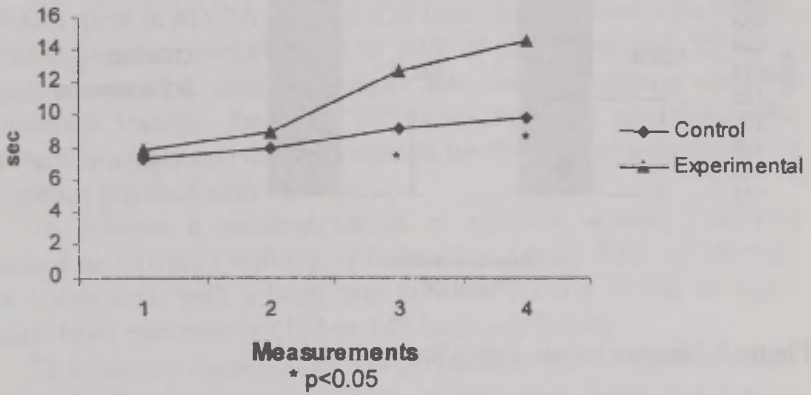


Figure 3. Changes in bent arm hang.

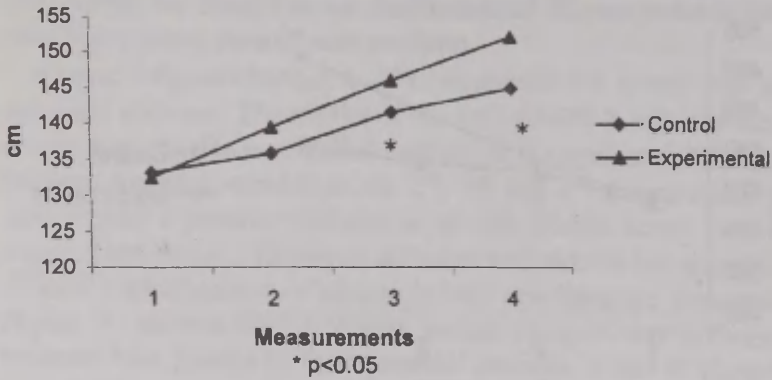


Figure 4. Changes in standing long jump.

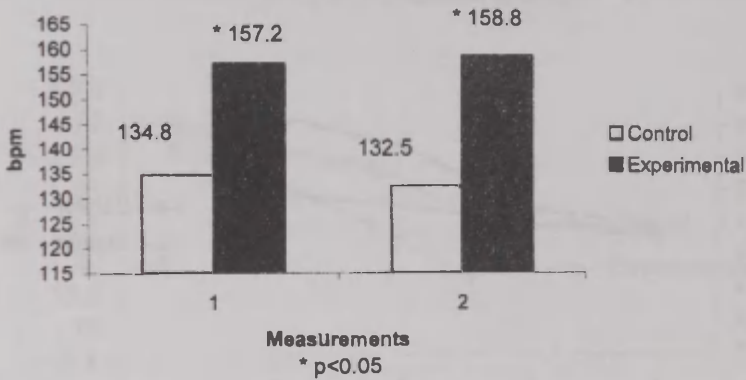


Figure 5. Mean heart rate during lessons.

DISCUSSION

Significant changes in health-related fitness measures and higher levels of physical activity were achieved using intensified or health-related primary physical education classes over the 2-year experimental period. These results are supportive for further

increasing the amount of health-related activities during physical education classes already beginning from the elementary school years. The importance of preservice training of classroom teachers, responsible for teaching physical education in elementary school years, is also evident from our results.

School physical education classes have many important goals, such as the acquisition of motor skills, creative and artistic expressions, self-realization and social development [3]. One important function of school physical education, however, is to engage students in moderate-to-vigorous physical activity, a requirement for experiencing both health and motor skill development benefits [19]. Moreover, children's cardiorespiratory fitness will only be promoted in physical education lessons if they spent an appropriate amount of time in MVPA. The findings of present study showed that mean heart rate of children during intensified physical education lessons was significantly higher compared with students of control classes. Different recommendations related to time children should spent in MVPA (at least 150 bpm) during classes for health-related purposes included 30 to 50% of class time [14, 25]. In a recent review of children's heart rate research during physical education lessons, Stratton [25] established two guidelines that include intensity and duration criteria for first moderate and second vigorous physical activity:

To achieve a moderate level of physical activity, children should be moving (walking or faster) for at least 50% (or 20 min) of lesson time with a heart rate between 50 and 59.9% of maximum heart rate reserve (135 to 148 beats per minute).

To achieve a vigorous level of physical activity, children's heart rates should be in excess of 60% of maximum heart rate reserve (148 beats per minute) for at least 50% (or 20 min) of lesson time.

Therefore, moderate-to-vigorous physical activity intensity between 50 and 74.9% (135 and 173 beats per minute) are appropriate for physical education curriculum goals for promoting physical activity and cardiorespiratory fitness [25].

Heart rate data of the present investigation showed that heart rate of children from experimental classes is meeting both recommendations established by Stratton [25] whereas physical activity

levels of students receiving standard physical education program met only first recommendation. Therefore, it is clearly evident that time spent in MVPA during physical education classes using intensified program is sufficient to promote health-related physical activity and fitness of elementary children. Consistent with our results, several previous studies have also demonstrated that health-related physical education program in elementary school can increase physical activity during lessons [8, 11, 17, 22]. Simons-Morton [20], for example, revealed that modification of the school physical education program increased the time children engaged in MVPA from less than 10% of class time at baseline to about 40% of the class time at posttest. However, as several authors [3, 11, 13] stated, the goals of primary physical education should not be limited for promotion of sufficient physical activity for health-related purposes, but also include development of motor skills, self-realization and social development. Thus, the implementation of health-related physical education lessons during elementary school years should be combined with alternative programs (motor learning, social and personal development etc.) for fulfilling broader goals of elementary school physical education.

The effects of intensified physical education lessons on health-related fitness of the children from experimental classes were evident after one year implementation period of this program. Multivariate analysis of variance showed that gains of cardiovascular fitness, muscular endurance, speed, agility and explosive strength were statistically significant. Effect sizes calculated for different fitness measures ranged from 0.21 to 0.34, which, according to Cohen [4] were moderate. Thus, the intervention led to improvements on health-related fitness components that were emphasized most in the experimental lessons. These results are in agreement with previous intervention studies [8, 17, 18] of elementary school students. Shephard [18] assessed the changes of physical fitness as a result of enhanced physical education program in 7 to 12 year old children. It was concluded that an enhanced program can increase fitness in primary school students, but the size of gains is not a strong argument for such programs. The effects of a 2-year physical education program on physical activity and fitness in elemen-

tary school students was assessed by Sallis *et al.* [17]. Consistent with results of present study, Sallis *et al.* [17] revealed that the largest fitness gains were found in experimental classes where lessons were taught by specialist-led conditions. In addition, interestingly, results showed that this effect was more marked in girls. Authors stated that the reason for that may be related to the fact that girls seem to be more responsive to physical activity and fitness change programs. Similar results were also reported by Vandongen *et al.* [26] and Stone *et al.* [24].

One weakness of present study, in our opinion, was the fact that the influence of possible confounding factors (habitual physical activity, somatic growth, social influences etc.) were not taken into consideration. However, previous investigations involving elementary school children have identified parental support, enjoyment of physical activity, time spent outdoors and attitudes toward exercise behavior as factors associated with physical activity behavior [16]. According to the recently developed ecological models [23], behavior (e.g. physical activity) can be influenced by multiple intra-personal, environmental and social variables [12]. Clearly, further work is needed to address the effects of school physical education, organized and non-organized exercise behavior outside of schools, as well as possible environmental and personal factors on physical fitness and health-related behavior during childhood.

In conclusion, significant effects of intensified physical education classes on health-related fitness measures as well as on physical activity during elementary lessons were apparent. These results are clearly supportive to orient elementary school physical education curriculum toward health-related fitness and activity content.

REFERENCES

1. **American Alliance for Health, Physical Education, Recreation and Dance.** (1994) Guidelines for elementary school physical education. A position statement of NASPE. Reston, VA: AAHPERD

2. **Blair S. N., Clark D. G., Cureton K. J., Powell K. E.** (1989) Exercise and fitness implications for a lifetime of health. In: *Perspective in Exercise in Sports and Exercise*, C. V. Gisolfi, D. R. Lamb (eds). Indianapolis, IN: Benchmark Press, Inc.
3. **Buschner C. A.** (1994) Teaching children movement concepts and skills: becoming a master teacher. *Human Kinetics*, Champaign, IL.
4. **Cohen J.** (1977) *Statistical power analysis for the behavioral sciences*. Academic Press, New York.
5. **Faucette N., McKenzie T. L., Patterson P.** (1990) Descriptive analysis of nonspecialist elementary physical education teachers' curricular choices and class organization. *J. Teach. Phys. Educ.* 9: 284–293.
6. **Kaneko M., Fuchimoto T.** (1993) Endurance performance capacity of 7 to 18 year boys and girls assessed by the "shuttle stamina test (SST)". In: *World-Wide Variation in Physical Fitness*, A. Claessens, J. Lefevre, B. van den Eynde (eds). Leuven: Institute of Physical Education, Catholic University of Leuven, 80–86.
7. **Koslow R. E.** (1988) Can physical fitness be a primary objective in a balanced physical education program? *J. Phys. Educ. Rec. Dance.* 59: 75–77.
8. **McKenzie T. L., Nader P. R., Strikmiller P. K., Yang M., Stone E. J., Perry C. L., Taylor W. C., Epping J., Feldman H., Luepker R. V., Kelder S. H.** (1996) School physical education: Effect of the Child and Adolescent Trial for Cardiovascular Health (CATCH). *Prev. Med.* 25: 423–431.
9. **McKenzie T. L., Sallis J. F., Kolody B., Faucetter N. F.** (1997) Long-term effects of a physical education curriculum and staff development program: SPARK. *Res. Q. Exerc. Sport.* 68: 280–291.
10. **McKenzie T. L., Sallis J. F., Faucetter N. F., Kolody B., Roby J.** (1993) Effects of an in-service intervention on the quality and quantity of elementary classroom teachers' physical education classes. *Res. Q. Exerc. Sport.* 64: 178–187.
11. **McKenzie T. L., Sallis J. F., Faucetter N. F., Roby J. J., Kolody B.** (1993) Effects of a curriculum and inservice program on the quantity and quality of elementary physical education classes. *Res. Q. Exerc. Sport.* 64: 25–31.
12. **McLeroy K. R., Bibeau D., Steckler A., Glanz K.** (1988) An ecological perspective on health promotion programmes. *Health Educ. Q.* 15: 351–377.

13. **Metzler M.** (1989) A review of research on time in sport pedagogy. *J. Teaching Phys. Educ.* 8: 87–103.
14. **Morrow J. R., Freedson P. S.** (1994) Relationship between habitual physical activity and physical fitness in adolescents. *Ped. Exerc. Sci.* 6: 315–329.
15. **Pate R. R., Small M. L., Ross J. G., Young J. C., Flint K. H., Warren C. W.** (1995) School physical education. *J. School Health.* 65: 312–318.
16. **Sallis J. F., Johnson M. F., Calfas K. J., Caparose S., Nichols J. F.** (1997) Assessing perceived physical environmental variables that may influence physical activity. *Res. Q. Exerc. Sport.* 68: 345–351.
17. **Sallis J. F., McKenzie T. L., Alcaraz J., Kolody B., Fauchette N., Roby J., Howell M. F.** (1997) The effects of a 2-year physical education program (SPARK) on physical activity and fitness in elementary school students. *Am. J. Publ. Health.* 87: 1328–1334.
18. **Shephard R. J., Lavallee H.** (1994) Changes of physical performance as indicators of the response to enhanced physical education. *J. Sports Med. Phys. Fitness.* 34: 323–335.
19. **Simons-Morton B. G., O'Hara N. M., Simons-Morton D. G., Parcel G. S.** (1987) Children and fitness: A public health perspective. *Res. Q. Exerc. Sport.* 58: 295–302.
20. **Simons-Morton B. G., Parcel G. S., Baranowski T., Forthofer R., O'Hara N. M.** (1991) Promoting physical activity and a healthful diet among children; Results of a school-based intervention study. *Am. J. Publ. Health.* 81: 986–991.
21. **Simons-Morton B. G., Parcel G. S., O'Hara N. M., Blair S. N., Pate R. R.** (1988) Health-related physical fitness in childhood: Status and recommendation. *Ann. Rev. Public Health.* 9: 403–425.
22. **Simons-Morton B. G., Taylor W. C., Snider S. A., Huang I. W.** (1993) Physical activity of fifth-grade students during physical education. *Am. J. Publ. Health.* 83: 262–265.
23. **Stokols D.** (1992) Establishing and maintaining healthy environments: Toward a social ecology and health promotion. *Am. Psychologist.* 47: 6–22.
24. **Stone E. J., Baranowski T., Sallis J. F.** (1995) Review of behavioral research for cardiopulmonary health: Emphasis on youth, gender, and ethnicity. *J. Health Educ.* 26 (suppl): S9–S17.
25. **Stratton G.** (1996) Children's heart rates during physical education lessons: A review. *Ped. Exerc. Sci.* 8: 215–233.

26. **Vandongen R., Jenner D. A., Thompson C.** (1995) A controlled evaluation of a fitness and nutrition intervention program on cardiovascular health in 10- to 12-year-old children. *Prev. Med.* 24: 9-22.

Correspondence to:

Lennart Raudsepp
Institute of Sport Pedagogy
University of Tartu
5 Jakobi Street Tartu 51014
Estonia

DEPENDENCE OF MUSCLE WORKING CAPACITY ON ARTERIAL BLOOD FLOW INTENSITY AT ONSET OF CALF EXERCISE

J. Poderys

Department of Physical Education and Sports Medicine,
Kaunas Medical University, Kaunas, Lithuania

ABSTRACT

The influence of short-term preliminary occlusion manoeuvre to the calf blood flow (CBF) and working capacity of the plantarflexor (PF) muscles was investigated in 14 male distance runners. CBF was measured by venous occlusion plethysmography. Working capacity (total work output) of the plantarflexor muscles was recorded before and after this manoeuvre. The results indicated that CBF in the reactive hyperaemia phase depended on the character and duration of the occlusion and on the degree of filling with blood of the primary segment under occlusion. The highest CBF was observed after occlusions, conducted when the amount of blood in the limb decreased before the occlusion and the lower CBF was recorded after occlusion done with the increased filling of blood vessels. Having a goal to maximally activate limb muscle arterial blood flow using occlusion of minimal duration the most optimal would be to conduct a 1 min occlusion to the sitting person after first decreasing blood vessel filling. A short-term occlusion improves muscle arterial blood flow and as a result muscle working capacity is improved.

Key words: arterial blood flow, occlusion plethysmography, muscle working capacity

INTRODUCTION

Muscle working capacity during long lasting physical activity is determined by blood supply [2, 3, 5, 8, 14, 17]. Changes in muscle blood flow directly influence intensity of oxidative metabolic processes in the muscle, and at the same time, they indicate their working capacity [2, 5, 9]. Thus, it is important to take into account the factors influencing blood circulation in solving a problem of how to increase muscle working capacity. For this purpose, a number of methods aimed at the increase of muscle working capacity were suggested. In this area it is suggested to use special regimes of muscle electrostimulation for the improving of muscle blood flow, various types of pressure chambers creating in them a negative pressure which facilitates an increased blood flow into the limbs [13]. In addition, there were suggested a various combinations of some methods for the improvement of muscle working capacity, and it was shown how these combinations improve muscle working capacity more substantially than any other stimulation method taken separately. For example, one of the variants of such combination of stimulation methods is a combination of so called "needle application" and negative pressure [6, 9].

A stable blood flow intensity in the working muscle settles only after 40–60 s or even later depending on the kind of work [8, 9]. Accordingly, if the arterial blood flow were intensified before the workout it would be possible to decrease the so called "blood debt" which appears at the beginning of the intensive work, and in this way to increase muscle working capacity.

The aim of this study was to measure the application of the preliminary occlusion to calf blood flow (CBF) and working capacity of the plantarflexor muscles. The following objectives were pursued. First, the peculiarities of reactive hyperemia depending on the time of the occlusion are investigated as well as it's character and preliminary filling with blood of the occluded limb blood vessels. Second, the influence of the preliminary occlusion on the working capacity of the plantarflexor muscles at onset of exercise.

MATERIAL AND METHODS

Fourteen male middle distance runners after written consent participated in the study. CBF (ml/min/100 cm³) was measured by venous occlusion plethysmography in sitting position, while electroplethysmograph EMPR-01 and Witney type sensors were used [9]. Three research series were conducted.

1. The influence of a short-term occlusion on the CBF. This research series consisted of three parts. In the 1st part, the changes in CBF was measured after 1, 2 and 3 min of occlusion under the resting condition, i.e. when the occlusion cuff was quickly inflated with air up to 250–260 mmHg of pressure in it. In this way, we have considered that the amount of blood in the calf's blood vessels during the occlusion was close to physiologically normal condition. In the 2nd part, the changes in CBF was measured after lifting the leg above the heart level and sustaining it in that position for 30 s. The amount of blood in the calf decreased by this manoeuvre and after a quick occlusion the leg was let down. The readings of the CBF after the end of the occlusion was registered in the sitting position. In the 3rd part, the changes in CBF were measured during a slowly blowing the air into the occlusion cuff (5 mmHg/s or slower), filling of the calf blood vessels with blood increases before the blood flow is arrested. There is larger amount of blood in the calf blood vessels than under conditions of physiological norm.

2. The influence of the short-term preliminary occlusion on the working capacity of the plantarflexor muscles. The maximal voluntary contraction (MVC) force of the plantarflexor muscles and total work output (ergogramma) during plantarflexion exercise were registered with specially designed ergodynamograph [9]. A total work output registered during plantarflexion movements by lifting a weight of 70% MVC force with intensity of 30 movements per min till exhaustion. Working capacity of the plantarflexor muscles was measured before and after application of short term preliminary occlusion.

3. Influence of a short-term preliminary occlusion on the CBF at onset of plantarflexion exercise. The CBF was measured during 2 exercises of 90 s duration performed with constant total work output (intensity) for the plantarflexor muscles: control exercise and exercise after exposure of short term preliminary occlusion. A short intervals of rest of 4 s duration was used periodically for measure the CBF.

Statistical Analysis

Data are means and standard errors of mean (\pm SEM). One-way analysis of variance (ANOVA) following by Tukey post hoc comparisons was used. A level of $p < 0.05$ was selected to indicate statistical significance.

RESULTS

1. The influence of a short-term occlusion on the CBF. The results of this part of study indicated that the increase of CBF the reactive hyperaemia phase depends on the character and duration of the occlusion and on the degree of filling with blood the calf's vessels before the arrest of blood flow (Tables 1 and 2). The highest CBF was observed after the occlusion conducted when the amount of blood in the limb was decreased before the occlusion and the lower CBF was registered after the occlusion done when there was increased filling with blood of blood vessels. The duration of the occlusion had direct influence on the arterial blood flow curves which were registered immediately after the end of occlusion. In all the cases the highest amount of CBF observed after the 3 min occlusions.

Table 1. CBF (ml/min/100 cm³) in the reactive hyperaemia phase depending on the duration of occlusion and on the degree of filling with blood of the segment (mean \pm SE).

Parts of experiment	Duration of occlusion		
	1 min.	2 min.	3 min.
I Physiological norm	13.94 \pm 0.40	17.04 \pm 1.21	19.74 \pm 0.59
II Preliminary decreased amount of blood	20.79 \pm 1.51	23.18 \pm 0.69	25.84 \pm 0.48
III Increased amount of blood before occlusion	11.71 \pm 0.57	17.05 \pm 0.29	18.04 \pm 0.38

Table 2. The dynamics of CBF (ml/min/100 cm³) in the reactive hyperaemia phase after 1 minute occlusions (mean \pm SE).

Parts of experiment	Before occlusion	Time of measurement			
		After 1-5s	After 15s	After 30s	After 45s
I Physiological norm	1.63 \pm 0.16	13.94 \pm 0.40	3.43 \pm 0.13	1.88 \pm 0.14	1.62 \pm 0.11
II Preliminary decreased amount of blood	1.66 \pm 0.10	20.79 \pm 1.51	5.71 \pm 0.37	2.61 \pm 0.17	1.90 \pm 0.12
III Increased amount of blood before occlusion	1.64 \pm 0.10	11.71 \pm 0.57	2.78 \pm 0.16	1.65 \pm 0.10	1.63 \pm 0.11

One aim of our experiments was to find the shortest possible duration of the occlusion and conditions under which the highest possible CBF could be obtained. When the subject was sitting, after conducting the occlusion and there was a preliminary decreased amount of peak arterial blood in the limb, relatively high level of CBF was observed even after occlusion of 1 min duration. It justified itself in all the subjects without exception. The CBF was higher ($p < 0.05$) obtained after 1 min occlusion in normal conditions than the amount of blood was increased before the occlusion. The CBF was decreased in the exponent-like manner and fully returned to the preliminary level after 1 min occlusion application 45 or 60 s after the end of the occlusion. The dynamics of the CBF

after arrest of 1 min duration, presented in the Table 2. After the occlusion, conducted when the blood vessel filling with blood was decreased, in the measurement made after 45 s the amount of flowing blood was higher than preliminary amount and in the measurement made after 60 s the amount of blood had returned to the preliminary level.

2. The influence of a short-term preliminary occlusion on the working capacity of the plantarflexor muscles. The results of this series of study are presented in Table 3. Total work output of the plantarflexor muscles registered during the control exercise and before the exposure to the experimental influence showed good stability of the method applied. The mean total work output produced during these two exercises was 1877.1 ± 27.2 J and 1880.6 ± 24.7 J, respectively. The muscle working capacity after the exposure of 1 min preliminary occlusion was improved. Subjects were able to produce more total work after a preliminary short-term occlusion, when a total work was in average 2100.6 ± 31.2 J. It was 11.7% higher ($p < 0.05$) than during the control exercise.

Table 3. Total work output of the plantarflexor muscles under the influence of 1 minute duration of CBF (mean \pm SE).

Parts of experiment	First exercise test (J)	Second exercise test (J)	Increase (%)
Control exercise	1877.1 ± 22.7	1814.6 ± 31.2	-3.3 ± 0.28
Exercise after preliminary occlusion	1880.6 ± 24.8	2100.6 ± 31.2	11.7 ± 0.31
Difference	$p > 0.05$	$p < 0.05$	$p < 0.05$

3. Influence of a short-term preliminary occlusion on the CBF at onset of plantarflexion exercise. The results of this series of study are presented in Table 4. During the control exercise till to 60s the CBF rised up to the steady state. The preliminary occlusion made a significant influence to the CBF at the beginning of exercise. The CBF even during the first seconds was significantly

($p < 0.001$) higher (in average 40.54 ± 4.87 ml/min/100 cm³) in comparison to the control exercise (13.89 ± 2.84 ml/min/100 cm³) and reached the steady state up to 30 s. There was no significant differences ($p < 0.05$) in the CBF readings during both of exercise.

Table 4. The dynamics of CBF (ml/min/100 cm³) during the two exercises (mean \pm SE).

Parts of experiment	Time of measurement								
	5s	10s	15s	20s	30s	45s	60s	75s	90s
Control exercise	13.89 ± 2.84	21.97 ± 4.48	27.41 ± 3.86	35.32 ± 5.84	47.32 ± 7.32	59.43 ± 5.17	62.78 ± 7.68	62.96 ± 8.69	63.80 ± 8.21
Exercise after pre- liminary occlusion	40.54 ± 4.87	46.94 ± 6.35	57.28 ± 6.10	60.85 ± 7.95	62.79 ± 7.75	63.78 ± 7.96	62.83 ± 6.71	62.27 ± 7.53	62.18 ± 6.96

DISCUSSION

The phenomenon of reactive hyperaemia — dilatation of the blood vessels after stopping of the blood circulation in the limb is well known regulatory mechanism of peripheral blood flow. This phenomenon has been used as a physiological test evaluating potentialities of the blood circulatory system and clinical test for diagnosing limb blood circulation disorders [7, 9]. Temporary stop of blood circulation has a single reaction response and is precisely dosed according to the length of the influence. After the long-term occlusions, the reactive hyperaemia takes place changing from aerobic metabolism to anaerobic. Metabolites of the anaerobic origin are accumulated in the muscles, which is one of the main factors determining the decrease in blood vessel tone after long lasting of blood arrests [7, 12]. A short-term arrest of blood flow does not have any major influence on the metabolic processes in the muscle. It was found [7] that considerable increase of anorganic phosphor and lactate is noticeable only after of arrests of blood flow longer than 7 min. The decrease of the blood vessels tone under the influence of short-term arrests of blood flow is determined by myogenic regulation mechanism [9, 12]. Under such conditions a given intra-

vascular pressure gives less deformation of the wall than when the smooth muscles are less active and, therefore, the discharges from the mechanoreceptors are reduced [12]. Experimental influence, applied in our experiment, probably did not have any considerable influence on metabolic processes in the muscles and regulatory mechanisms of blood circulation.

Regulatory mechanisms of the systemic blood circulation is oriented to sustain a gradient of pressure, necessary to insure needed blood circulation intensity in working muscles. It happens in the combination of heart work indexes and changes of total peripheral resistance [1, 8, 12, 16]. The regulations of local blood circulation is done mostly by changing hydrodynamic resistance of blood vessels, i.e. by changing their diameter [11, 16]. As hydrodynamic resistance is oppositely proportional to the blood vessel diameter in fourth degree, because the changes in their diameter is more important to the intensity of blood circulation in the organs, than changes in the arterial pressure [12].

The results of the present study indicated, that activity increase of the CBF in the reactive hyperaemia phase depends on the character and duration of the occlusion and on the degree of filling with blood of the primary segment under occlusion. Highest CBF was registered after the occlusion, conducted when the amount of blood in the limb was decreased before the occlusion and the lowest blood circulation readings were registered after occlusion done when there was increased filling with blood of blood vessels. This shows the importance of miogenic regulatory mechanisms. The duration of the occlusion had not direct influence on the CBF amounts registered after the end of the occlusion. Fast decrease in arterial blood flow intensity indicates that physical load should be applied as soon as possible after occlusion.

The goal of our studies was to find the shortest possible duration of the occlusion and conditions, under which the highest possible arterial blood circulation activity could be obtained. This is important since after longer blood flow arrests, the reactive hyperaemia takes place going from aerobic metabolism to anaerobic, and anaerobic metabolites start to accumulate in the muscles [7]. We have found in that, when the subject was sitting, after conducting

the occlusion when there was a decreased amount of blood in the limb, relatively high peak arterial blood flow was registered even after 1 min of occlusion. It was true for all the subjects without exception. This means, that having a goal to maximally activate limb muscle blood flow using occlusion of minimal duration the most optimal would be to conduct a 1 min occlusion to the sitting person, after first decreasing blood vessel filling.

In the intensifying of local arterial blood flow two stages can be distinguished. The 1st stage — the blood circulation intensifies, this intensifying levels off afterwards, but soon the next stage of the blood circulation intensifying starts — 2nd stage. These stages reflect blood vessel — small arteries, arterioles, precapillary sphincters, capillaries and big arteries — behaviour during the intensifying work phase. This was proven in the experimental research [4]. The first blood circulation increase stage reflects vasodilatation of capillaries, precapillary sphincters, arterioles and small arteries, and the second stage shows a vasodilatation of big, so called highway arteries. As shown in the study mentioned above [4] and in the work on physiological mechanisms of reactive hyperaemia [7] the second stage of blood vessel vasodilatation develops only after longer lasting occlusions i.e. when the artery walls react to increased speed of blood flow. In our research the figures of maximal blood flow in the reactive hyperemia phase were not maximal possible so they do not reflect maximal possibilities of the blood circulation system. In this case the intensifying of blood circulation after short-term arrests of blood flow was related to the changes in functional state of the vessels beds. The results of this study have shown that a exposure of short term preliminary occlusion decreased so called “blood debt” which appears at the beginning of the intensive work, and in the same way increased muscle working capacity.

Functional vasodilatation cannot be attributed to changes in the local concentration of any single metabolic factor [6]. Mechanical factors, such as vascular compression and increases in perfusion pressure also affect vascular resistance and skeletal muscle blood flow. However, the specific manner in which these mechanical factors interact is not well understood [6, 8]. The recent determina-

tion that arterial vessels, upstream from the active tissue and microcirculation, also dilate during reactive hyperaemia and during voluntary muscle contractions has led to the consideration that the vessels are primarily responsible for the regulation of bulk flow to the tissue. Consideration of the interrelations of the factors affecting vasomotor tone may lead to a better understanding of the regulation of muscle blood flow.

The arterial blood flow in the reactive hyperaemia phase depends on the character and duration of the occlusion and on the degree of filling with blood of the primary segment under occlusion. Having a goal to maximally activate limb muscle blood circulation using occlusion of minimal duration the most optimal would be to conduct a 1 min occlusion to the sitting person after first decreasing blood vessel filling.

In conclusion, a short-term preliminary occlusion improves muscle blood flow and as a result muscle working capacity is improved.

REFERENCES

1. **Ahlborg G., Ottosson-Seeberger A., Hemsén A., Lundberg J. M.** (1996) Central and regional hemodynamic effects during infusion of big endothelin-1 in healthy humans. *J. Appl. Physiol.* 80: 1921–1927.
2. **Depairon M., Zicot M.** (1996) The quantitation of blood flow/metabolism coupling at rest and after exercise in peripheral arterial insufficiency, using PET and ^{15}O labelled tracers. *Angiol.* 47: 991–999.
3. **Fitzpatrick R., Taylor J. L., McCloskey D. I.** (1996) Effects of arterial perfusion pressure on force production in working human hand muscles. *J. Physiol. (Lond.)* 495: 885–891.
4. **Hajutin V. M., Meštšerski E. L., Veselova E. S.** (1980) Work-induced hyperaemia in skeletal muscles. *Bull. Acad. Med.* 80: 54–60 (in Russian).
5. **Hughson R. L., Shoemaker J. K., Tschakovsky M. E., Kowalchuk J. M.** (1996) Dependence of muscle VO_2 on blood flow dynamics at onset of forearm exercise. *J. Appl. Physiol.* 81: 1619–1626.
6. **Lash J. M.** (1996) Regulation of skeletal muscle blood flow during contractions. *Proc. Soc. Exp. Biol. Med.* 211: 218–235.

7. **Matizone D. P., Skards J. V.** (1980) Comparison of production of the anaerobic metabolites to forearm blood flow during reactive hyperemia. In: *Regulation of Blood Flow in Muscles*. Riga, pp. 64–69 (in Russian).
8. **Ozoliņš P. P.** (1984) *Adaptation of the Circulatory System to Sports Activities*. Riga: Zinātne (in Russian).
9. **Poderys J.** (1998) Influence of the short-term preliminary occlusion on the dynamics of arterial blood flow and working capacity of the calf muscles. *J. Educ. Phys Training Sport*. 1: 49–57.
10. **Poderys J., Trinkunas E.** (1999) Venous occlusion plethysmography for the assessment of the microcirculation under influence of calf's stretch exercise. *Proceedings of International Conference on Biomedical Engineering*. Kaunas: Technologija, pp. 125–129.
11. **Saltin B.** (1988) Capacity of blood flow delivery to exercising skeletal muscle in humans. *Am. J. Cardiol.* 62: 30–35.
12. **Schmidt R. F., Thews G.** (1989) *Human Physiology*. 2nd Ed. Berlin.
13. **Sundberg C. J.** (1994) Exercise and training during graded leg ischemia in healthy men. *Acta Physiol. Scand.* 150: 615–650.
14. **Terjung R. L., Mathien G. M., Erney T. P., Ogilvie R. W.** (1988) Peripheral adaptations to low blood flow in muscle during exercise. *Am. J. Cardiol.* 62: 15–19.
15. **Tkatchenko B. I., Kuprjanov V. V., Orlov, R. S., Gurevich M. I., Banin V. V., Maisbin B. I.** (1989) *Physiology of Blood Vessels*. *Perf. Physiol. Sci.* 20: 3–26 (in Russian).
16. **Tschakovsky M. E., Shoemaker J. K., Hughson R. L.** (1996) Vasodilation and muscle pump contribution to immediate exercise hyperemia. *Am. J. Physiol.* 271: 1697–1701.
17. **Vasiljeva V. V.** (1989) Blood flow to muscles — the main factor of performance in athletes. *Theor. Pract. Phys. Cult.* 8: 35–36 (in Russian).

Correspondence to:

Jonas Poderys
Department of Physical Education and Sports Medicine
Kaunas Medical University
Jankaus 2
LT-3026 Kaunas
Lithuania

ANTHROPOMETRICAL AND PHYSIOLOGICAL FACTORS OF ROWING PERFORMANCE: A REVIEW

J. Mäestu, J. Jürimäe

Institute of Sport Pedagogy, University of Tartu, Tartu, Estonia

SUMMARY

The anthropometry and physiology of rowing performance is briefly reviewed. Rowing is a strength-endurance type of sport, and body size and body mass are undoubtedly performance related factors. Most of the successful rowers are very tall, with a low body fat percent and high muscle mass, with approximately 75 percent of slow-twitch muscle fibers in working muscles. The lungs of oarsmen reflect their large body dimensions and ventilation is also very high in trained rowers. Effort in a 2000 metre rowing race is about 70% aerobic. In world class oarsmen, maximal oxygen consumption reaches 6.0–6.6 $\text{l}\cdot\text{min}^{-1}$. Aerobic power and muscle endurance often change by 10% over the season. The high maximal oxygen consumption values are reached by training volumes of approximately 5000–6000 km per year. As the 2000 metre rowing race is about 30% anaerobic, anaerobic energy sources play also an important role in competition success. Average peak lactate values of 15 $\text{mmol}\cdot\text{l}^{-1}$ have been measured after national and 17 $\text{mmol}\cdot\text{l}^{-1}$ after international regattas. These values reflect that oarsmen have extremely large anaerobic capacities. The development of performance prediction models based on laboratory data in rowers has practical importance for talent identification and for the development and assessment of rowing performance. Rowing performance could be assessed using specific anthropometric and metabolic variables.

Key words: rowing performance, anthropometrical parameters, physiological parameters

1. INTRODUCTION

The typical rowing competition takes place on a 2000 metre course and lasts 6–7 min. During the competition, anaerobic alactic and lactic as well as aerobic capacities are stressed to their maximum [55]. In rowing, which is a strength-endurance type of sport, body size and body mass are undoubtedly performance related factors [6, 53]. This is obvious when analyzing the energetic profile of a 2000 metre race. A rower has to develop more than 200 times a stroke with a peak force of 800 to more than 1000 Newton [6, 53, 55]. Maximal strength and strength-endurance are therefore basic components of their year round training program [6, 56]. Both components are related to body size and body mass [6]. Maximum oxygen consumption of rowers is among the largest values recorded. Rowing involves approximately 70% of the muscle mass because all extremities and the trunk participate in the propulsion of the boat. Rowing is a cyclic movement, where both legs and arms work synchronized, using one (sweep rowing) or two (sculling) oars [55]. There are some differences in the biological demands of sweep and sculling boats and the need for massive body build is increased if the craft carries a cox [51, 53].

During training 15–40 strokes per minute are used and 32–38 strokes per minute during a race in the single scull corresponding to a stroke duration of 0.6–2.2 seconds. Between 210 and 230 strokes are performed during the 2000 metre race. Mainstays of the rowing technique are the turning points at the start and at the end of the stroke and its force time characteristics. The balance is also important and may be a challenge especially in the smaller racing shells. The coordination with team members is another prerequisite for rowing success [55].

In this article the anthropometric and physiological factors of rowing performance are briefly reviewed.

2. ANTHROPOMETRIC PROFILE

Rowing is a weight-supported sport [53]. Except in competitions with a specific weight limitation (i.e., the lightweight categories, where a maximum body mass is 72.5 kg for males and 59.0 kg for females), it is advantageous to recruit rowers with massive body build, thereby ensuring that a high proportion of the total mass transported is active muscle rather than the "dead-weight" of the cox and the vessel itself [51].

Anthropometric profiles of rowers have extensively been studied (Table 1). It is an advantage for the rower to be tall [52, 53]. Anthropometric data for adult rowers emphasize the importance of body mass and body size for rowing performance [7]. Usually, elite rowers are more than 190 cm tall and weigh 90–95 kg. Long arms are particularly helpful in giving extra leverage [53]. Ideally, the body mass of rowers should contain a high proportion of muscle [6, 53]. For example, muscle mass of Estonian national level heavyweight rowers was found to be 49.5 ± 6.1 kg, which corresponded to $62.3 \pm 3.1\%$ of their body mass [28]. Bourgois *et al.* [6] found that the muscle mass of 168 male junior rowing world championship competitors was 50.2 kg and 62.4% of the whole body mass. A large muscle mass does not penalize rowers, whose body mass is supported in the boat [9]. Significant correlations between muscle mass and 2000 metre rowing ergometer time trial ($r \geq -0.85$) have been found [9, 27]. Furthermore, Jürimäe *et al.* [27] reported significant relationships between muscle mass and 2000 metre rowing performance on single sculls ($r = -0.64$). The percentage of body fat seems to have been decreasing in recent years [53]. Carter [8] found means of 7.8% in men participating in the 1976 Montreal Olympics and McKensie *et al.* [38] reported a mean of 9.6% for the Canadian 1980 Olympic team. Hagerman *et al.* [16] found no changes for body composition parameters between in-season and off-season among 1984 USA Los Angeles Olympic team. However, lightweight rowers tend to reduce their body fat% for competitive season. Morris *et al.* [42] reported that male lightweight rowers reduced their body fat % from 10.0% to 7.8% while no changes were observed in fat free mass.

Table 1. The anthropometric parameters of male rowers.

	Height (cm)	Weight (kg)	BMI (kg×m ⁻²)	Body fat %	Study
Heavyweight					
1997 junior WC sculling rowers (n=168)	186.5±6.3	80.4±6.9	23.1±1.6	11.5±2.2	Bourgeois <i>et al.</i> [6]
1997 junior WC sweep rowers (n=214)	188.2±5.3	83.6±7.5	23.6±1.8	11.8±2.6	Bourgeois <i>et al.</i> [6]
WC and OG gold medal winners (n=2)	197	99	25.5	—	Larsson and Forsberg [31]
International level German rowers (n=28)	194	89	23.6	—	Hagerman and Hagerman [18]
International level German rowers (n=9)	187.2±4.9	81.1±6.3	23.2±5.6	—	Beneke [3]
International level French rowers (n=12)	182.0±5.0	77.0±7.0	23.2±6.0	—	Messonnier <i>et al.</i> [40]
National level Estonian rowers (n=10)	191.7±4.1	85.4±4.2	23.2±1.3	7.6±1.9	Jürimäe <i>et al.</i> [27]
National level Danish rowers (n=139)	183.5±2.7	74.8±4.1	22.3±4.3	11.8±2.6	Jensen [26]
Dutch national team 1988 (n=18)	190.0	79.3	22.0	—	Rienks <i>et al.</i> [44]
Lightweight					
1985 WC rowers (n=144)	180.7	70.3	20.1	—	Rodriguez [45]
USA olympic team (n=12)	183.0±3.0	72.2±1.4	21.5±2.1	—	Hagerman and Staron [16]
National level Aus- tralian rowers (n=18)	180.5±2.7	69.8±1.6	21.8±2.1	7.8±0.8	Morris and Payne [42]
National level Danish rowers (n=68)	183.0±4.5	74.5±3.0	22.5±3.8	11.1±1.9	Jensen [26]

BMI — body mass index; WC — world championships; OG — olympic games.

In the men, the world champions were 10.0% taller and 27.2% heavier than the general Canadian population [51]. Hirata [21] also pointed out that gold medal winners were consistently taller and heavier than the average for national champions. In the case of single sculls, the respective differences were a substantial 0.12 metre and 9.6 kg. Hahn [19] suggested that more successful rowers are tall, heavy and possess a low skinfold reading. Malina [37] noted that promising rowers were already taller than the general population during childhood and they maintained their relative advantage throughout adolescence. Carter [8] suggested that the body dimensions of national level contestants were increasing by about 0.02 metre and 5.0 kg per decade.

Comparing different boat classes, Hirata [21] found that sweep rowers were consistently taller and heavier than sculling rowers, the difference amounting to some 0.02 metre and 3.8 kg for men. In competitions where a cox was carried, there were further small increases in the height and body mass of the successful rowers, 0.03 metre and 5.0 kg in the case of male pairs [21]. Bourgois *et al.* [6] investigated the participants of the 1997 World Junior Championships and found that the limbs of sweep rowers were taller than scullers. Sweep rowers had also bigger girths than scullers, but no differences were found in skinfold thicknesses [6]. Furthermore, no differences were found in bone diameters between sweep and sculling rowers [6].

The anthropometric characteristics of lightweight rowers differ radically from those of their heavier peers [33, 53]. In male competitors, de Rose *et al.* [10] found no significant differences in anthropometric characteristics between lightweight rowers and untrained student controls. The advantage of heavyweight rowers in smaller distances (up to 500 metres) has been calculated to be about 4% which is reduced to 2.5% over the 2000 metre distance and remains about the same as the distance gets longer [55].

In summary, rowers are tall and have relatively high muscle mass. Sweep rowers are usually taller and heavier and have longer limbs than the scullers. In recent years, the height and body mass of rowers has increased, while the percentage of body fat has decreased.

3. PHYSIOLOGICAL PROFILE

Rowers are strong, reflecting their large body dimensions, but their maximal muscle strength is not correlated in any simple way to their rowing strength [52]. Their body mass is supported while seated in the boat [55]. Rowing is a sport in which about 70% of the whole body muscle mass is involved [53, 55]. They are unique in their ability to develop a strength with the use of both legs which corresponds to the sum of strength of the right and left legs [52]. In untrained subjects and athletes trained in other disciplines, two-legged strength of the left and right legs determined separately corresponds approximately 80% of the sum of the left and right legs determined separately, but increases with specific training [52].

Corresponding to the relative low duty cycle (approximately 36 strokes per minute), the strength of rowers is more pronounced at low contraction velocities [52, 55]. For the muscles engaged in rowing, the percentage of slow-twitch oxidative muscle fibers is approximately 70% [52, 55]. Differences are also found between the structure of muscles in highly and less qualified rowers, although they trained similarly with respect to time and volume [55]. In internationally successful competitive rowers, slow-twitch oxidative muscle fiber content has been reported as high as 85%, with few fast-twitch glycolytic fibers in the *vastus lateralis* muscle [46, 55]. Muscle hypertrophy is evident in rowers [52, 53, 55]. Hypertrophy is found not only in fast-twitch glycolytic and oxidative glycolytic muscle fibers, but also in slow-twitch oxidative muscle fibers [22]. The hypertrophy is more evident in internationally successful rowers [46]. Muscle hypertrophy as a result of training is primarily caused by volume expansion of single fibers [46, 55]. The muscle structure also depends on the biomechanical requirements of different boat seats [46].

The lungs of rowers reflect their large body dimensions. Rowers have large vital capacities with a highest recorded value of 9.1 l, and the better rowers tend to have relatively large values of their vital capacity [52]. As with other types of exercise, ventilation increases during ergometer [41] and on water [49] rowing in

proportion to the increase in oxygen consumption to a certain point, after which a more marked increase takes place [52]. The highest recorded value has been reported to be $243 \text{ l} \times \text{min}^{-1}$ [52]. Hagerman *et al.* [15] found the highest ventilation value of 221 l in 310 competitive oarsmen, while maximal ventilation averaged $200 \text{ l} \times \text{min}^{-1}$ in world class championships winners ($n = 14$) [49]. For a given oxygen uptake, ventilation is low in trained rowers [49].

The position of the body and the use of respiratory muscles in rowing may limit ventilation and thereby reduce maximal aerobic power relative to that achieved in cycling or treadmill running [54]. The results of the investigations showed that ventilation at a given maximal oxygen consumption during intense submaximal exercise (higher than 75% of maximal oxygen consumption) was not significantly lower compared with that in cycling and treadmill running, which would suggest that submaximal rowing does not restrict ventilation [54]. At maximal effort, maximal oxygen consumption and ventilation for rowing were less than those for the other types of exercise, although the differences were not statistically significant in elite oarsmen [54]. These data are consistent with a ventilatory limitation to maximal performance in rowing that may have been partly overcome by training in elite rowers. Alternatively, a lower maximal ventilation in rowing might have been an effect rather than a cause of a lower rate of muscle activation in rowing [54]. Szal *et al.* [57] found that breathing rates are higher for a given intensity of submaximal effort during rowing than during cycling.

Hyperventilation during rowing is more marked than during cycling and is associated with a higher breathing frequency [57]. A high breathing frequency during rowing in trained rowers reflects that respiration is coupled to the rowing stroke [35]. Furthermore, inspiration is relatively short during the drive phase of the stroke [51]. As elite rowers make two inspirations during one drive cycle [34], then less qualified rowers were able to make only 1.48 inspirations during one drive cycle [55]. Therefore, some changes in breathing frequency take place as the qualification gets better in rowers.

In summary, elite rowers have more slow-twitch oxidative muscle fibers in their working muscles than less qualified rowers. As rowing is very intensive, rowers present high vital capacity and ventilation values in comparison with other sportlers.

3.1. Aerobic power

During competition, a rower depends mainly on his/her aerobic metabolism because energy stores and glycolysis are limited to cover the energy demand only for approximately 1.5–2.0 minutes [55]. Aerobic power can be defined as the maximal oxygen consumption as estimated during a performance that lasts two to 10 minutes [26]. Aerobic and anaerobic energy contributions on a 2000 metre rowing race in different studies are presented in Table 2. According to Roth *et al.* [46], the energy of the 2000 metre race was provided 67% aerobically and 33% anaerobically, 21% alactic and 12% lactic. While Secher *et al.* [48] found that the aerobic energy contribution may be up to 86%.

Table 2. Mean contribution of aerobic and anaerobic energy during rowing in different studies using elite heavyweight male rowers.

Study	n	Aerobic contribution %	Anaerobic contribution %
Russell <i>et al.</i> [47]	19	84	16
Droghetti <i>et al.</i> [12]	19	80	20
Hagerman <i>et al.</i> [16]	310	70	30
Hartmann [20]	17	82	18
Mickelson and Hagerman [41]	25	72	28
Roth <i>et al.</i> [46]	10	67	33
Secher <i>et al.</i> [48]	7	70–86	14–30
Messonnier <i>et al.</i> [40]	13	86	14

Maximal oxygen consumption of rowers has extensively been studied (Table 3). Values for maximal oxygen consumption have increased over the years from approximately $3.4 \text{ l} \times \text{min}^{-1}$ [52]. In world class rowers, maximal oxygen consumption reaches $6.0\text{--}6.6 \text{ l} \times \text{min}^{-1}$ [55]. While maximal oxygen consumption is often large in rowers, this finding reflects mainly their large body dimensions [52]. The relative oxygen consumption is relatively low in rowers compared to other endurance athletes because their high body mass [6, 17, 49, 53, 55] and those with the highest value expressed in litres per minute will tend to show the lowest relative value [24]. Only in some, mainly in lightweight rowers, relative oxygen consumption reaches $75 \text{ ml} \times \text{min}^{-1} \times \text{kg}^{-1}$ [50, 55].

Maximal oxygen consumption is an important predictor of competition success, although its predictive influence varies in different analyses [50, 55]. For example, the correlations of $r=-0.64$ to $r=-0.87$ between maximal oxygen consumption and on-water rowing performance have been found [9, 27, 50]. Maximal oxygen consumption depends on the content of slow-twitch oxidative muscle fibers as well as the level of aerobic and anaerobic threshold [53, 55].

Maximal oxygen consumption increases with training distance per year, but levels off at training volumes of approximately 5000–6000 km per year [55]. Seasonal changes have been described in maximal oxygen consumption. Maximal oxygen consumption may increase $5\text{--}15 \text{ ml} \times \text{min}^{-1} \times \text{kg}^{-1}$ during the competitive season [55]. Hagerman *et al.* [16] reported maximal oxygen consumption to increase from 5.09 to $6.01 \text{ l} \times \text{min}^{-1}$ from off-season to in-season among 1984 USA Los Angeles Olympic rowing team, while maximal heart rate showed no statistically significant changes. Pronounced changes in maximal oxygen consumption of highly trained rowers take place if during off-season the distance rowed is reduced below approximately 100 km per week [55]. Intensive endurance training increases not only mitochondrial volume in slow-twitch muscle fibers but also in fast-twitch muscle fibers [22]. There is a strong relation between maximal oxygen consumption and whole body mitochondrial volume [22].

Tabel 3. Maximal oxygen consumption in male rowers.

Subjects	$l \times \text{min}^{-1}$	$\text{ml} \times \text{min}^{-1} \times \text{kg}^{-1}$	Study
Heavyweight			
Winners of international regattas (n=14)	5.8	63.0	Secher <i>et al.</i> [50]
Participates of international regattas (n=13)	5.5	67.0	Secher <i>et al.</i> [50]
International level USA rowers (n=14)	6.0	69.1	Hagerman and Staron [16]
International level Italian and Danish rowers (n=18)	5.7±0.4	–	Jensen and Nielsen [25]
International level German rowers (n=310)	5.9	67.6	Hagerman <i>et al.</i> [15]
International level English rowers (n=13)	5.7±0.1	–	Doherty <i>et al.</i> [11]
International level Polish rowers (n=168)	5.15±0.4	60.5±3.2	Klusiewicz <i>et al.</i> [29]
International level French rowers (n=13)	4.9±0.3	64.8±5.5	Messonnier <i>et al.</i> [40]
National level German rowers (n=13)	4.6±0.6	59.8±8.1	Urhausen <i>et al.</i> [58]
National level English rowers (n=25)	4.7±0.4	–	Lakomy and Lakomy [30]
National level Finnish rowers (n=25)	5.1±0.4	–	Peltonen and Rusko [43]
National level Estonian rowers (n=10)	4.9±0.6	61.6±5.6	Jürimäe <i>et al.</i> [27]
Glasgow University male rowers (n=13)	4.5±0.4	–	Cosgrave <i>et al.</i> [9]
Virginia University male rowers (n=10)	4.6±1.5	–	Womack <i>et al.</i> [61]

Table 3. Maximal oxygen consumption in male rowers (continued).

Subjects	$l \times \text{min}^{-1}$	$\text{ml} \times \text{min}^{-1} \text{kg}^{-1}$	Study
Lightweight			
International level German rowers (n=10)	5.1	72.0	Secher <i>et al.</i> [50]
International level Danish rowers (n=68)	5.2 ± 0.1	–	Jensen [26]
International level Irish rowers (n=10)	5.6 ± 0.3	–	Mahony <i>et al.</i> [36]
Juniors			
English juniors (n=9)	4.5 ± 0.1	–	Doherty <i>et al.</i> [11]
Polish juniors (n=8)	4.9 ± 0.4	58.7 ± 5.1	Faff <i>et al.</i> [13]
Australian juniors (n=19)	4.5 ± 1.4	–	Russell <i>et al.</i> [47]
Belgian juniors (n=10)	4.1 ± 0.4	–	Bourgois <i>et al.</i> [6]

Muscle structure plays also an important role in submaximal performance [55]. With a higher percentage of slow-twitch muscle fibers, rowers are able to perform with more power per stroke at a blood lactate concentration of $4.0 \text{ mmol} \times \text{l}^{-1}$ [55]. However, specific endurance training increases the work rate per stroke at a given lactate level, without changing the slow-twitch muscle fiber content mainly due to higher oxidative capacities of fast-twitch muscle fibers [23]. As rowing training increases the oxidative capacity of both slow-twitch and also fast-twitch muscle fibers, the maximal oxygen consumption of highly trained rowers with different slow-twitch muscle fiber content may not be different [23, 55].

The submaximal endurance capacity measured as the power which elicits a blood lactate level of $4.0 \text{ mmol} \times \text{l}^{-1}$ (i.e., anaerobic threshold) has been reported to be the most predictive parameter for competition performance in trained rowers, especially in small boats such as singles and doubles [3, 52, 53, 55, 60]. In highly trained rowers, anaerobic threshold corresponds to approximately 80–85% of maximum performance [55]. The maximal oxygen consumption at $4.0 \text{ mmol} \times \text{l}^{-1}$ anaerobic threshold has also been reported to be approximately 85% of maximal oxygen consump-

tion [55]. When taking the individual anaerobic threshold into account, Bourgois *et al.* [5] found significantly lower individual anaerobic threshold values compared to anaerobic threshold values during the progressive incremental test on a rowing ergometer. The study concluded that the two anaerobic threshold concepts did give different information with respect to the capacity for submaximal exercise and further research is necessary to determine a real maximal aerobic steady-state concept [5]. Maximum lactate decreases with higher anaerobic threshold due to higher oxidative metabolic capacity [55].

In summary, the maximal oxygen consumption of rowers is high among other endurance athletes and significant changes in maximal oxygen consumption take place during competitive season. Relative oxygen consumption is lower due to high body mass. The relative oxygen consumption is higher in lightweight rowers.

3.2. Anaerobic power

Roth *et al.* [46] calculated from metabolic and bioptic measurements in tank rowing that the energy for the simulated rowing race of seven minutes was provided 67% aerobically and 33% anaerobically, 21% alactic and 12% lactic (Table 2). However, anaerobic capacity explains only 10–20% of the performance in competition in trained rowers [55, 60]. One reason could be that all trained rowers have a highly developed strength due to the hypertrophy of slow-twitch and fast-twitch muscle fibers as well as increased muscle mass [55]. The reported energy contribution from the anaerobic energy system suggests that it would significantly influence 2000 metre rowing performance [47]. Anaerobic power and capacity are physiological factors dominating the performance during the start and the finish of a rowing race [48, 55]. Lower glycolytic capacities may be of negative effect at the start spurt and the final spurt in the rowing race [56].

The lactate concentration in blood shows the power of the anaerobic energy process. Average team values of $15 \text{ mmol} \times \text{l}^{-1}$

have been measured after national regattas and $17 \text{ mmol} \times \text{l}^{-1}$ after international regattas [59]. For example, lactate concentration after 2000 metre single scull race was found to be $16.02 \pm 2.42 \text{ mmol} \times \text{l}^{-1}$ in Estonian national level rowers ($n=10$) [27]. These values reflect that rowers have extremely large anaerobic capacities [27, 52]. Bangsbo *et al.* [2] suggested that the anaerobic energy production during intense exercise is related to the muscle mass involved.

Anaerobic capacity can be estimated as the maximal oxygen deficit, i.e., the difference between the estimated total oxygen requirement and the oxygen consumption established during an "all-out" performance lasting from two to six minutes [39]. The accumulated oxygen deficit was 36% higher ($p < 0.05$) during maximal rowing compared to running [2]. Taking the different stroke frequencies into account, the maximal oxygen deficit has been calculated to be $95 \text{ ml} \times \text{min}^{-1} \times \text{kg}^{-1}$ for rowers [12] and thus substantially higher than the $64 \text{ ml} \times \text{min}^{-1} \times \text{kg}^{-1}$ in runners as reported by Bangsbo *et al.* [1]. Russell *et al.* [47] found accumulated oxygen deficit in 19 junior rowers to be $2.1 \pm 1.4 \text{ l} \times \text{min}^{-1}$. The total oxygen debt for 310 competitive oarsmen during six minute "all-out" rowing ergometer test averaged 13.4 litres [15]. The validity of this method of measuring anaerobic capacity is supported by a close relationship with the anaerobic energy produced in a single muscle group [1]. The oxygen deficit appears not to be related to blood lactate during submaximal exercise, muscle enzyme activity, number of muscle capillaries, percent of slow-twitch muscle fibers and/or muscle buffer capacity [2].

It is well established that the central and peripheral adaptations in rowers in response to exercise training result in an increase in maximal oxygen consumption as well as in a concomitant shift of the blood lactate versus work rate curve to the right, i.e., towards higher absolute and relative work rates [40]. The shift of the lactate curve towards higher relative work rate shows a possible dissociation between the underlying phenomena that lead to the shift and those involved in the concomitant increase in the aerobic capacity [40]. The shift indicates that the magnitude of the adaptive metabolic processes inducing the changes in lactate is proportionally greater than the corresponding changes in oxygen consumption

[40]. Under these conditions, differences may subsist in lactate kinetic parameters among highly trained rowers with approximately the same oxygen consumption and working at the same relative work rate [40].

Despite the importance of the lactate performance curve and the anaerobic threshold, this concept has some limitations [55]. In successful rowers of comparable competition level, the anaerobic threshold of $4.0 \text{ mmol} \times \text{l}^{-1}$, maximum lactate and maximal oxygen consumption may be very different. A lower anaerobic threshold of $4.0 \text{ mmol} \times \text{l}^{-1}$ may be compensated to some degree by higher lactate formation, by increased lactate tolerance, and also by higher work efficiency [55]. Maximum lactate decreases with higher anaerobic threshold due to higher oxidative metabolic capacity [52, 55]. Since lactate concentration is the dynamic resultant of both lactate appearance and disappearance, a shift in $4.0 \text{ mmol} \times \text{l}^{-1}$ blood lactate concentration can be a result of a change in one or both of these processes [40]. However, the measurement of $4.0 \text{ mmol} \times \text{l}^{-1}$ blood lactate concentration alone gives no information on any of these parameters in rowers [40].

Peak lactate levels should always be determined on well-rested and well-nourished competitors, since values may be influenced by both recent exercise and glycogen depletion [53]. The peak lactate concentrations appear to be substantially higher in male rowers, both adults and juniors (typically $11\text{--}19 \text{ mmol} \times \text{l}^{-1}$ and occasionally as high as $25 \text{ mmol} \times \text{l}^{-1}$) than in females ($8.6\text{--}10.5 \text{ mmol} \times \text{l}^{-1}$) [53]. The main explanation is likely to be that men have larger muscle mass relative to blood volume than women [53, 55]. The peak lactate concentrations reached by male competitors are inversely related to the proportion of slow-twitch muscle fibers in the active muscle groups [55].

Anaerobic power can be measured on a rowing ergometer. The anaerobic alactic power has been measured by five maximal strokes and anaerobic lactic power by 40 second maximal work [27, 55]. Anaerobic tests have not been sensitive enough to detect changes with training [32]. Although, strength and anaerobic capacity are important in rowing, they should not be increased above a "critical" value [32].

In summary, anaerobic capacity explains about 10–20% of the performance in rowing competition and is high in trained rowers. Average blood lactate values have been reported to be 15–17 mmol \times l⁻¹ after 2000 metre distance. Maximum blood lactate decreases with higher anaerobic threshold due to the higher oxidative metabolic capacity.

4. GENERAL CONSIDERATIONS

The development of performance prediction models based on laboratory data allows coaches and scientists to predict on-water rowing performance and to identify potentially talented rowers. Furthermore, it allows the development and assessment of different training programmes for rowers of different boat classes and weight categories. As observed in many studies, a typical heavyweight rowers morphological prototype is that of a tall, heavy and lean athlete with a large aerobic and anaerobic capacities [6, 7, 28, 52, 53, 55]. These morphological qualities occur directly from specific rowing training and genetic inheritance [47]. This is in accordance with the recent findings of Jürimäe *et al.* [28] who found significant relationships between 2000 metre rowing ergometer time and height, body mass and muscle mass ($-0.77 < r < -0.81$), while muscle mass was the only characteristic to predict competition results on 2000 metre single scull distance. Anthropometric parameters are more important in heavyweight than in lightweight rowers. It is advantageous for lightweight rowers when being not very tall and have high metabolic values. A large muscle mass is achieved by long hours of aerobic training combined with resistance training [28, 47, 56]. This type of training also results in a rower with a high aerobic capacity and metabolic efficiency [28, 47, 56].

When taking the metabolic characteristics into account, the most predictive parameter has been reported to be maximal oxygen consumption ($l \times \text{min}^{-1}$), although its predictive influence varies in different analyses [28, 47, 52, 55, 61]. On-water rowing perform-

ance has also been shown to be associated with the ability to maintain a high percentage of maximal aerobic power and high metabolic efficiency [18]. In support to this finding Jürimäe *et al.* [28] found significant relationships between maximal aerobic power and 2000 metre single scull distance ($r=-0.70$). On-water rowing is also significantly related to maximal aerobic power (i.e., power, where maximal oxygen consumption is reached) [28]. Competitive rowing means not only maximal aerobic but also maximal anaerobic effort. Significant correlations have been found between 40 second maximal work (i.e., anaerobic lactic power) and 2000 metre rowing ergometer time trials [28]. However, Russell *et al.* [47] found that anaerobic capacity, as measured by the accumulated oxygen deficit, did not correlate significantly with the 2000 metre rowing ergometer time-trial. These differences could be explained by the fact that the anaerobic energy contribution during 2000 metre ergometer time-trial was low (16%) in that study, whereas it has been reported to be 20–30% during 2000 metre on-water rowing [49, 53, 55].

In summary, the prediction of individual performance of rowers needs a complex testing battery, where specific anthropometric and physiological parameters are used.

REFERENCES

1. **Bangsbo J., Gollnick P. D., Graham T. E., Juel C., Kiens B., Mizuno M., Saltin B.** (1990) Anaerobic energy production and O_2 deficit-debt relationships during exhaustive exercise in humans. *J. Physiol.* 422: 539–559.
2. **Bangsbo J., Petersen A., Michalsik L.** (1993) Accumulated oxygen deficit during intense exercise and muscle characteristics of elite athletes. *Int. J. Sport Med.* 14: 207–213.
3. **Beneke R.** (1983) Anaerobic threshold, individual anaerobic threshold, and maximal lactate steady-state in rowing. *Med. Sci. Sport Exerc.* 27: 863–867.

4. **Bouckaert J., Pannier L., Vrijens J.** (1983) Cardiorespiratory response to bicycle and rowing ergometer exercise in oarsmen. *Eur. J. Appl. Physiol.* 51: 51–59.
5. **Bourgois J., Vrijens J.** (1998) Metabolic and cardiorespiratory responses in young oarsmen during prolonged exercise tests on a rowing ergometer at power outputs corresponding to two concepts of anaerobic threshold. *Eur. J. Appl. Physiol.* 77: 164–169.
6. **Bourgois J., Claessens A. L., Vrijens J.** (1998) Hazewinkel anthropometric project 1997. A study of world class male and female junior rowers. Vlaamse Trainerschool, BLOSO, Brussels.
7. **Bourgois J., Claessens A. L., Vrijens J., Phillipaerts R., Renterghem B., Thomis M., Janssens M., Loos R., Lefevre J.** (2000) Anthropometric characteristics of elite male junior rowers. *Br. J. Sports Med.* 34: 213–217.
8. **Carter J. E. L.** (1982) Body composition of athletes. In *Physiol Structure of Olympic Athletes. Part I* (ed. J. E. L. Carter). Basel. Karger: 107–116.
9. **Cosgrave M. J., Wilson J., Watt D., Grant S. F.** (1999) The relationship between selected physiological variables of rowers and rowing performance as determined by a 2000 m ergometer test. *J. Sport Sci.* 17: 845–852.
10. **de Rose E., Crawford S. M., Kerr D. A., Ward R., Ross W. D.** (1989) Physique characteristics of Pan-American lightweight rowers. *Int. J. Sport Med.* 10: 292–297.
11. **Doherty M., Hughes M. G., Godfrey R., Warrington G., Hawkins S.** (1999) A comparison of physical, physiological and performance parameters in Great Britain senior and junior oarsmen. *J. Sport Sci.* 17: 12–13.
12. **Droghetti P., Jensen K., Nielsen T. S.** (1991) The total estimated metabolic cost of rowing. *FISA Coach.* 2: 1–4.
13. **Faff J., Biensko A., Jagodinska T., Burkhard K., Borkovski L.** (1993) Diagnostic value of indices derived from the critical power test in assessing the anaerobic work capacity of rowers. *Biol. Sport.* 10: 9–14.
14. **Haber P., Fertlich A., Kummer F.** (1978) Einschätzung der Maximalen Aeroben Kapazität mittels einfacher Ergometrie bei unterschiedlichen Trainingszutäten. *Akta Med. Austria.* 4: 176–179.
15. **Hagerman F., Connors M., Gault J., Hagerman G.** (1978) Energy expenditure during simulated rowing. *J. Appl. Physiol.* 45: 87–93.

16. **Hagerman F., Staron R.** (1983) Seasonal variables among physiological variables in elite oarsmen. *Can. J. Appl. Sports Sci.* 8: 143–148.
17. **Hagerman F.** (1984) Applied physiology of rowing. *Sport Med.* 1: 303–326.
18. **Hagerman F., Hagerman M.** (1990) A comparison of energy output and input among elite rowers. *FISA Coach.* 2: 5–8.
19. **Hahn A.** (1990) Identification and selection of talent in Australian rowing. *Excel.* 6: 5–11.
20. **Hartmann U.** (1987) Querschnitten an Leistungsrudern im Flachland und Langsschnittuntersuchungen an Elite-rudern in der Höhe mittels eines zweistufigen Tests auf einem Gjessing-ruderergometer. Hartnung Görre Verlag, Konstanz.
21. **Hirata K.** (1979) Selections of Olympic Champions. Tokyo, Hirata Institute.
22. **Howald H.** (1982) Training-induced morphological and functional changes in skeletal muscle. *Int. J. Sport Med.* 3: 1–12.
23. **Howald H.** (1988) Leistungsphysiologische Grundlagen des Ruderns. In *Rudern* (ed J. Steinacker). Berlin, Heidelberg, Springer. 31–38.
24. **Jensen K., Secher N. H., Fiskestrand A., Christensen N. J., Lund J. O.** (1984) Influence of body weight on physiologic variables measured during maximal dynamic exercise. *Acta Physiol. Scand.* 212: 39.
25. **Jensen K., Nielsen T.** (1993) High altitude training does not increase maximum oxygen uptake or work capacity at sea level in rowers. *Scand. J. Med. Sci. Sport.* 3: 56–62.
26. **Jensen K.** (1994) Test procedures for rowing. *FISA Coach.* 4: 1–6.
27. **Jürimäe J., Mäestu J., Jürimäe T., Pihl E.** (1999) Relationship between rowing performance and different metabolic parameters in male rowers. *Med. Sport.* 52: 119–126.
28. **Jürimäe J., Mäestu J., Jürimäe T., Pihl E.** (2000) Prediction of rowing performance on single sculls from metabolic and anthropometric variables. *J. Hum. Mov. Stud.* 38: 123–136.
29. **Klusiewicz A., Faff J., Zdanowicz R.** (1997) The usefulness of PWC_{170} in assessing the performance determined on a rowing ergometer. *Biol. Sport.* 14: 127–132.
30. **Lakomy H., Lakomy J.** (1992) Estimation of maximum oxygen uptake from submaximal exercise on a Concept II rowing ergometer. *J. Sport Sci.* 11: 227–232.

31. Larsson L., Forsberg A. (1980) Morphological muscle characteristics in rowers. *Can. J. Appl. Sport Sci.* 5: 239–240.
32. Lormes W., Debatin H. J., Grünert-Fuchs M., Müller T., Steinacker J. M., Stauch M. (1990) Anaerobic rowing ergometer tests—test design, application and interpretation. In *Advances in Ergometry* (ed: Bachl N., Graham T., Löllgen H.), Berlin, Heidelberg, Springer: 477–482.
33. Mahler D., Nelson W., Hagerman F. (1984) Mechanical and physiological evaluations of exercise performance in elite national rowers. *JAMA.* 252: 496–499.
34. Mahler D., Parker H., Andersen D. (1985) Physiological changes in rowing performance associated with training in collegiate women rowers. *Int. J. Sport Med.* 6: 229–233.
35. Mahler D. A., Shuhart C. R., Brew E., Stukel T. A. (1991) Ventilatory responses and entertainment of breathing during rowing, *Med. Sci. Sport Exerc.* 23: 186–192.
36. Mahony N., Dome B., O'Brien M. (1999) A comparison of physiological responses to rowing on friction-loaded and air-braked ergometers. *J. Sport Sci.* 17: 143–149.
37. Malina R. M. (1994) Physical activity and training: effects on stature and the adolescent growth spurt. *Med. Sci. Sport Exerc.* 26: 759–766.
38. McKenzie D. C., Rhodes E. C. (1982) Cardiorespiratory and metabolic responses to exercise on a rowing ergometer. *Aus. J. Sport Med.* 14: 21–23.
39. Medbo J. L., Mohn A., Tabata I., Bahr J., Vaage O., Sejersted O. M. (1988) Anaerobic capacity determined by maximal oxygen deficit. *J. Appl. Physiol.* 64: 50–60.
40. Messonnier L., Freund H., Bourdin M., Belli A., Lacour J. (1997) Lactate exchange and removal abilities in rowing performance. *Med. Sci. Sport Exerc.* 29: 396–401.
41. Mickelson T. C., Hagerman F. C. (1982) Anaerobic threshold measurements of elite oarsmen. *Med. Sci. Sport Exerc.* 14: 440–444.
42. Morris F., Payne W. (1996) Seasonal variables in the body composition of lightweight rowers. *Br. J. Sport Med.* 30: 301–304.
43. Peltonen J., Rusko H. (1992) Interrelations between power, force production and energy metabolism in maximal leg work using a modified rowing ergometer. *J. Sport Sci.* 11: 233–240.
44. Rienks N. H., van der Pol A. J., Toussaint H. M. (1988) De evaluatie van conditie en techniek bij top- en subtoproeiers op een isokinetische roeergometer. *Gneeskunde en sport*, 2: 34–39.

45. **Rodriguez F. A.** (1986) Physical structure of international lightweight rowers. In: Reilly T., Watkins J., Borms J., eds. *Kinantropometry III*. London: E and FN Spon: 255–261.
46. **Roth W., Hasart E., Wolf W., Pansold B.** (1983) Untersuchungen zur Dynamic der Energiebereitstellung während maximaler Mittelzeitausdauerbelastung. *Med. Sport*. 23: 107–114.
47. **Russell A. P., le Rossignol P. F., Sparrow W. A.** (1998) Prediction of elite schoolboy 2000-m rowing ergometer performance from metabolic, anthropometric and strength variables. *J. Sport Sci.* 16: 749–754.
48. **Secher N. H., Vaage O., Jackson R.** (1982) Rowing performance and maximal aerobic power of oarsmen. *Scand. J. Sport Sci.* 4: 9–11.
49. **Secher N. H.** (1983) The physiology of rowing. *Int. J. Sport Med.* 1: 23–53.
50. **Secher N., Vaage O., Jensen K., Jackson R.** (1983) Maximal aerobic power in oarsmen. *J. Appl. Physiol.* 51: 155–162.
51. **Secher N. H.** (1990) Rowing. In: *Physiology of sports* (ed. Reilly T., Secher N., Snell P., Williams C.). 259–286.
52. **Secher N. H.** (1993) The physiology of rowing. *Sport Med.* 15: 23–53.
53. **Shephard R. J.** (1998) Science and medicine of rowing: A review. *J. Sport Sci.* 16: 603–620.
54. **Smith T. B. R. J., Hopkins W. G., Taylor N. A. S.** (1994) Respiratory responses of elite oarsmen, former oarsmen, and highly trained non-rowers during rowing, cycling and running. *Eur. J. Appl. Physiol.*, 69: 44–49.
55. **Steinacker J. M.** (1993) Physiological aspects of rowing. *Int. J. Sport Med.* 1: 3–10.
56. **Steinacker J. M., Lormes W., Lehmann M., Altenburg D.** (1998) Training of rowers before world championships. *Med. Sci. Sport Exerc.* 30: 1158–1163.
57. **Szal S. E., Schoene R. B.** (1989) Ventilatory response to rowing and cycling in elite oarsmen. *J. Appl. Physiol.* 67: 264–269.
58. **Urhausen A., Weiler B., Kinderman W.** (1993) Heart rate, blood lactate and catecholamines during ergometry and on-water rowing. *Int. J. Sport Med.* 14: 20–23.
59. **Vaage O.** (1986) *Textbook of work physiology*. McGraw-Hill, New-York: 673.
60. **Wolf W. V., Roth W.** (1987) Validität Spiroergometrischer Parameter für die Weltkampfleistung im Rudern. *Med. Sport*. 27: 162–166.

61. **Womack C. J., Davis S. E., Wood C. M., Sauer K., Alvarez A., Weltmann A., Gaesser G. A.** (1996) Effects of training on physical correlates of rowing ergometry performance. *J. Str. Cond. Res.* 10: 234-238.

Correspondence to:

Jaak Jürimäe
Institute of Sport Pedagogy
University of Tartu
18 Ülikooli Street
50090 Tartu
Estonia

RESPONSES OF BLOOD HORMONES TO TWO HOURS OF ENDURANCE ROWING ON SINGLE SCULLS IN MALE COMPETITIVE ROWERS

J. Jürimäe, T. Jürimäe, P. Purge

Institute of Sport Pedagogy, University of Tartu, Tartu, Estonia

ABSTRACT

This investigation studied hormonal responses to a single endurance rowing training session in 12 male competitive single scull rowers (23.3 ± 6.3 yrs; 188.7 ± 5.9 cm; 82.0 ± 10.8 kg; body fat %: $10.9 \pm 3.3\%$). Anaerobic threshold (AT_4) was determined during the first measurement session on a rowing ergometer. The second measurement session consisted of an endurance rowing training session on single sculls for about 2 hrs (7891 ± 761 sec; covered distance: 22.6 ± 2.5 km; mean heart rate: 136.2 ± 6.6 beats \times min⁻¹; intensity: $77.4 \pm 3.8\%$ from calculated AT_4). Venous blood samples were obtained before and immediately after and after 30, 60 and 120 min of exercise. Cortisol (C), total testosterone (T_{tot}) and sex-hormone-binding-globulin (SHBG) were measured and free testosterone (T_{free}) and the T_{free} :C ratio calculated. The lactate concentration in blood did not change significantly during training (1.7 ± 0.4 to 1.9 ± 0.4 mmol \times l⁻¹), while body mass was reduced (82.0 ± 10.8 to 80.6 ± 11.2 kg). The concentrations of C and T_{tot} were not significantly changed during rowing and in the first two hours of the recovery. T_{free} was reduced during the first 30 min of the recovery and a further reduction was established during the second hour. Body mass immediately after the training session was significantly related to the covered distance ($r = -0.75$). The concentrations of C ($r = 0.49$) and T_{free} ($r = -0.58$) were also significantly related to the covered distance. The results of this study indicated that a prolonged low intensity rowing training session poses a similar anabolic and catabolic stimulus for the trained rower.

Key words: testosterone, cortisol, endurance training, competitive rowers

INTRODUCTION

The anabolic hormone testosterone (T_{tot}) and the catabolic hormone cortisol (C) blood concentrations are influenced by the intensity and duration of endurance exercise [3, 13]. Circulating levels of T_{tot} and C in blood have been used to evaluate the metabolic balance in rowers [8, 14, 16, 19, 20, 21]. Free testosterone (T_{free}), the ratio between T_{tot} and the sex-hormone-binding-globulin (SHBG) levels, may be more specific indicator of anabolism as it represents the biologically available T_{tot} [8, 16, 20, 21]. The $T_{\text{free}}:C$ ratio is postulated to describe the balance between anabolic and catabolic metabolism. A decrease in $T_{\text{free}}:C$ ratio should then indicate a metabolic strain [8, 20, 21, 22]. Accordingly, the responses of anabolic and catabolic hormones can be used for assessment of the trainable effect of exercise session and for control of the recovery period. Changes in the anabolic and catabolic metabolism have been described during maximal rowing ergometer bout [8, 14, 19].

Rowing training is described in terms of extensive (below $2 \text{ mmol} \times \text{l}^{-1}$ blood lactate [LA]), intensive ($2\text{--}4 \text{ mmol} \times \text{l}^{-1}$ blood LA), highly intensive ($4\text{--}8 \text{ mmol} \times \text{l}^{-1}$ blood LA) endurance training and speed or tempo training [20, 21]. Since during a 2000 m rowing competition the aerobic energy supply makes of $\approx 70\text{--}86\%$ of the total energy, depending upon boat class and race tactics [15, 17, 20], prolonged extensive rowing on water makes up the largest part of the training programme [20, 21]. For example, Mader *et al.* [12] report that 86–94% of the training volume during winter and 70–77% during summer is extensive. This study was conducted to evaluate the adaptation of anabolic and catabolic hormones to a single endurance rowing training session below $2 \text{ mmol} \times \text{l}^{-1}$ blood lactate in male competitive single scull rowers.

METHODS

Subjects

Twelve male national level rowers volunteered to participate in the study (Table 1). The subjects had trained regularly for the last 7.1 ± 3.2 years. Measurements took place during the pre-competition period in May. The rowers were fully familiarised with the procedures before providing their consent to participate in the experiment as approved by the Medical Ethics Committee of University of Tartu. Each rower was tested on two separate sessions interspaced by at least one week. The rowers were asked not to participate in any physical activity in the 24 hrs before testing and to abstain from eating for 3 hrs before testing. Height (Martin metal anthropometer) and body mass (A&D Instruments Ltd, UK) of the subjects were measured to the nearest 0.1 cm and 0.05 kg, respectively. Body composition was measured using a multifrequency impedance analyzer (Multiscan 5000, UK).

Table 1. Physical and functional characteristics of rowers (n=12).

Variable	X \pm SD	Range
Age (yrs)	23.3 \pm 6.3	19.0–34.0
Height (cm)	188.7 \pm 5.9	178.0–197.0
Weight (kg)	82.0 \pm 10.8	67.10–97.0
Body fat (%)	10.9 \pm 3.3	4.7–16.5
AT ₄ (W)	280.0 \pm 39.4	225.0–373.0
AT ₄ (beats \times min ⁻¹)	176.1 \pm 9.7	156.0–191.0
Pa _{max} (W)	357.7 \pm 52.9	299.2–484.9

AT₄, anaerobic threshold; Pa_{max}, maximal aerobic power.

Heart rate (HR) was stored at five seconds intervals (Sporttester Polar Vantage NV, Kempele, Finland). Anaerobic threshold defined by HR (AT₄; beats \times min⁻¹) was determined during the first measurement session. A progressive incremental exercise test to maximal intensity was performed on a wind resistance braked

rowing ergometer (Concept II, Morrisville, USA) [9, 10]. The initial work load was 150 W and power was increased every third minute by 50 W until exhaustion. The stroke rate varied between 18 and 34 strokes \times min⁻¹. Capillary blood samples for enzymatic determination of LA (Lange, Germany) were taken from a fingertip during a 30 s interval at the end of each work intensity. The AT₄ was assessed by linear interpolation from the LA versus HR curve [9, 10].

The second measurement session consisted of anaerobic rowing on single sculls at the intensity of $\approx 75\%$ AT₄ for about two hours. The training took place on the river at air temperature of 20–22°C, with a humidity of 40–45% and the weather was not windy. After voiding, the subject's baseline (PRE) measurements were performed. Following the baseline measurements, the subject warmed-up with stretching and jogging for 15 min, and then rowed for an average of two hours and 17 minutes (7891 \pm 761 sec). The mean covered distance was 22.6 \pm 2.5 km. Exercise mean HR was 136 \pm 7 beats \times min⁻¹ (range 129–148 beats \times min⁻¹) or 77.4 \pm 3.8% of AT₄. Body weighing and venous blood sample were repeated immediately post-exercise (POST), and at 30 min (POST-30'), one hour (POST-60') and two hours (POST-120') from rowing. The LA before exercise was 1.7 \pm 0.4 mmol \times l⁻¹ and did not change significantly during the training session (after: 1.9 \pm 0.4 mmol \times l⁻¹). The subjects were not allowed to drink during exercise and for two hours recovery period.

Blood analysis

Blood sampling (10 ml) was obtained from the anticubital vein with the subjects in the upright position. The plasma was separated by centrifugation and frozen at -20°C until analyses were performed. The T_{tot}, C and SHBG were measured in duplicate by radioimmunoassays (RIAs) (Orion Diagnostica, Orion Corporation, Finland) [7]. Samples from one individual were run in the same assay. The inter- and intra-assay coefficients of variation were less than 5%. The T_{free} was calculated as the ratio of T_{tot} to SHBG and

$T_{free}:C$ as the ratio of T_{free} to C [8]. Aliquots of the whole blood were also analysed in quadruplicate for haematocrit (Hct) using 12000 rpm for five minutes and haemoglobin (Hgb) using a Lange (Germany) microanalyzer. Post-exercise percentage changes in blood volume (BV), red cell (CV) and plasma volume (PV) were calculated from Hgb and Hct in each subject using the formula of Dill and Costill [2].

Statistical methods

Mean \pm standard deviation (SD) for each of the dependent variables were determined following each measurement occasion. The Friedman analyses of variance (ANOVAs) by ranks were used to examine changes at each test point as the raw data and their logarithmic transformations were not normally distributed. An alpha level of 0.05 was adopted. The Wilcoxon matched-pairs signed-ranks test was used where post hoc analysis was necessary. Kendall Rank Correlation coefficient was used to determine the relationship between dependent variables at each time point. An alpha level of 0.05 was used.

RESULTS

Body mass was reduced after the training session (Table 2). Hct was increased immediately after the training session and decreased during the first 30 min of the recovery (Table 2). No other statistically significant changes were noted during the two hours recovery period. No significant changes were observed in the concentration of Hgb over the period of investigation. Accordingly, BV and CV did also not change significantly during the endurance training and two hours recovery period. Reduction in PV was found immediately after the training session ($-4.2 \pm 6.7\%$). During the first 30 min of recovery, PV increased almost to the preexercise level and remained unchanged during the two hours recovery period.

Table 2. Body mass and blood haematology before (PRE), immediately after (POST) and after 30 minutes (POST-30'), 1 hour (POST-60') and 2 hours (POST-120') of extensive endurance rowing training session ($\bar{X} \pm \text{SD}$).

Variable	PRE	POST	POST-30'	POST-60'	POST-120'
Body mass (kg)	82.0 \pm 10.8	80.6 \pm 11.2*	80.1 \pm 10.2*	79.9 \pm 10.0*#	80.0 \pm 10.0*
Haematocrit (%)	45.7 \pm 2.6	47.8 \pm 1.8*	46.2 \pm 1.9#	45.9 \pm 2.2#	45.8 \pm 2.7#
Haemoglobin (g \times dl ⁻¹)	14.9 \pm 1.0	15.2 \pm 0.8	15.0 \pm 0.8	14.9 \pm 0.7	15.2 \pm 0.9
Δ Blood volume (%)	0	-2.5 \pm 5.2	-1.0 \pm 3.5	-0.6 \pm 2.8	-2.1 \pm 3.7
Δ Plasma volume (%)	0	-4.2 \pm 6.7*	-1.2 \pm 5.4#	-0.2 \pm 5.7#	0.6 \pm 5.9#
Δ Cellular volume (%)	0	1.2 \pm 2.3	0.2 \pm 3.8	0.0 \pm 2.6	-1.9 \pm 4.3

* Significantly different from PRE; $p < 0.05$.

Significantly different from POST; $p < 0.05$.

The concentrations of C and T_{tot} were not significantly changed during the training session and during the first two hours recovery (Figure 1). The T_{free} was not significantly changed immediately following the training session. However, a reduction in T_{free} occurred during the first 30 min of recovery and further reduction occurred during the second hour of recovery. No significant changes were observed in the $T_{\text{free}}:C$ ratio. The SHBG increased immediately following training and remained elevated during the two hours of recovery (PRE: 35.29 \pm 8.45 nmol \times l⁻¹; POST: 39.81 \pm 10.22 nmol \times l⁻¹).

A relationship was established between the covered distance and the body mass measured immediately after the training session ($r = -0.75$). No significant relationships were observed between the covered distance and blood haematology parameters ($r < 0.49$). The covered distance was also related to the C ($r = 0.49$) and T_{free} ($r = -0.58$) immediately after rowing. Other relationships between the covered distance and hormone values were not significant ($r < 0.40$).

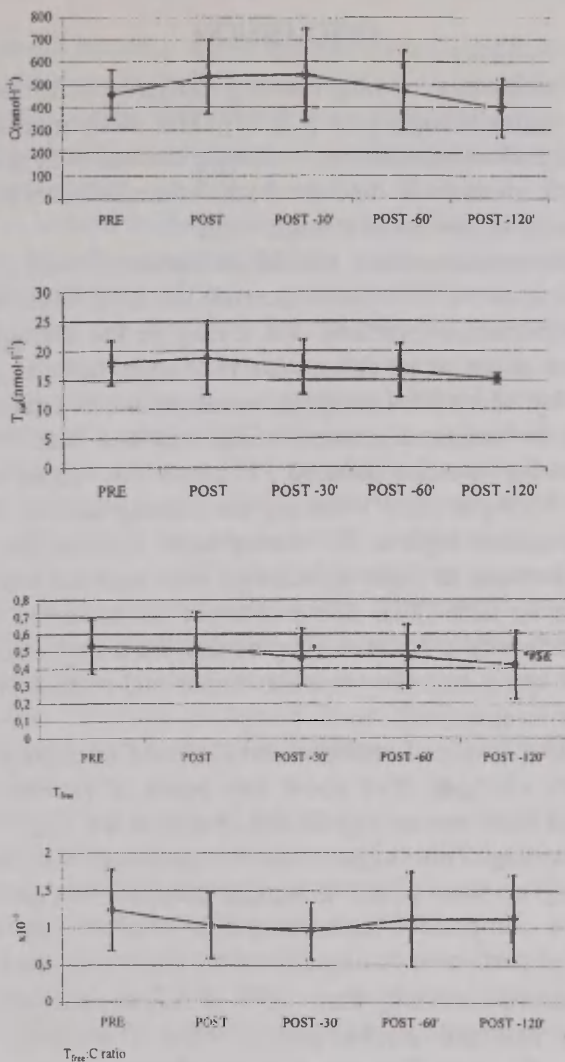


Figure 1. Cortisol (C), total testosterone (T_{tot}), free testosterone (T_{free}) and T_{free}:C ratio before (PRE), immediately after (POST) and after 30 minutes (POST-30'), 1 hour (POST-60') and 2 hours (POST-120') of extensive endurance rowing training session (X±SD). *Significantly different from PRE; p<0.05. #Significantly different from POST; p<0.05. \$Significantly different from POST-30'; p<0.05. & Significantly different from POST-60'; p<0.05.

DISCUSSION

Extensive endurance rowing training forms a main part of the training in competitive rowers [20, 21]. This study investigated the effects of a prolonged endurance training session with no elevation in blood LA on anabolic and catabolic hormone concentrations in male competitive rowers in a single scull.

Hormone concentrations should be corrected with changes in PV as a decrease in PV would increase the level even without an increase in hormone secretion. Yet it may be the concentration of the hormone at the target tissues that is of importance regardless of the means by which the change in concentration is established [5]. Therefore, the hormone concentrations reported here are used as uncorrected for exercise-induced PV alterations. There was a decrease (by 4.2%) in the PV during the training session. However, this change is not high as PV during acute exercise has been reported to decrease in close association with exercise intensity and duration up to 20% [18]. These changes are mediated by an increased intravascular hydrostatic pressure, fluid shifts from the intravascular space into the muscular tissue and especially by fluid losses via sweating [10].

Circulating levels of anabolic and catabolic hormones were not significantly changed after about two hours of extensive rowing training and there was no significant change in the $T_{\text{free}}:C$ ratio as a result of training. This suggests that the prolonged endurance rowing training session posed a similar anabolic versus catabolic stimulus for competitive male rowers. It could be argued that the high level of performance capacity of the rowers requires a greater absolute exercise intensity than $\approx 75\%$ of AT_4 to attain a given percentage of maximal aerobic power, what is necessary to elicit changes in T_{free} and C concentrations after the aerobic exercise (i.e., [1, 22]). Viru [22] has suggested that the threshold intensity for these hormone responses may be closely related to the AT_4 . When the exercise intensity is below that response threshold in trained individuals, a greater duration of exercise may be necessary to achieve a response [1, 22]. From these perspectives, a longer duration of training session at the intensity of $\approx 75\%$ of AT_4 , or a

higher exercise intensity might have provided a sufficient stimulus for the increases in hormone concentrations in competitive rowers. However, these kind of endurance training sessions are not frequent in the training programmes of competitive rowers [20, 21].

Immediately after the training session C ($r=0.49$) and T_{free} ($r=-0.58$) were related to the covered distance. This is in accordance with Viru [22] who suggests that the hormonal response depends on the total amount of work performed. However, the given amount of work that was done by our subjects was not enough to elicit an increase in the post-exercise hormone levels. In addition, Viru [22] proposed that the correlation between hormone levels and performance is established only after an earlier hormone effect on a muscle tissue. Usually, there appears to be a biphasic increase in the circulating level of C during a two hours of endurance exercise [22]. The blood C concentration increased slightly during rowing training session and achieved the highest value after 30 min of recovery. While C decreased rapidly after the 30 min of recovery. The increased tissue uptake of C is responsible for the early decline in serum C levels during the recovery [4, 11]. The plasma T_{free} responses in rowers are similar to the hormone results of other studies reported in response to a prolonged aerobic treadmill running [6].

In summary, the results of this study demonstrated that plasma T_{free} and C concentrations were not significantly changed in endurance-trained rowers in response to the prolonged rowing training session below the intensity of $2 \text{ mmol} \times \text{l}^{-1}$ blood LA. In addition, the lack of significant changes in $T_{\text{free}}:C$ ratio as a result of training session suggested that this kind of endurance training posed a similar anabolic versus catabolic stimulus for competitive rowers.

ACKNOWLEDGEMENTS

The authors are greatly indebted to researcher Dr. Eve Pihl for help with taking and analysing blood samples.

REFERENCES

1. **Banfi G.** (1998) Hormonal ratio (cortisol:testosterone) as a marker of exhaustion in athletes. *J. Sports Med. Phys. Fit.* 38: 91–92.
2. **Dill D. B., Costill D. L.** (1974) Calculation of percentage changes in volumes of blood, plasma and red cells in dehydration. *J. Appl. Physiol.* 37: 247–248.
3. **Fahrner C. L., Hackney A. C.** (1998) Effects of endurance exercise on free testosterone concentration and the binding affinity of sex hormone binding globulin (SHBG). *Int. J. Sports Med.* 19: 12–15.
4. **Few J. D.** (1974) Effect of exercise on the secretion and metabolism of cortisol in man. *J. Endocrinol.* 62: 341–353.
5. **Hackney A. C., Premo M. C., McMurray R. G.** (1995) Influence of aerobic versus anaerobic exercise on the relationship between reproductive hormones in men. *J. Sports Sci.* 13: 305–311.
6. **Hoffman J. R., Maresh C. M., Armstrong L. E., Gabaree C. L., Bergeron M. F., Kenefick R. W., Castellani J. W., Ahlquist L. E., Ward A.** (1994) Effects of hydration state on plasma testosterone, cortisol and catecholamine concentrations before and during mild exercise at elevated temperature. *Eur. J. Appl. Physiol.* 69: 294–300.
7. **Jaffe B. M., Behrman H. R.** (1979) *Methods of Hormone Radioimmunoassay.* New York: Academic Press.
8. **Jürimäe J., Jürimäe T.** (2000) Responses of blood hormones to the maximal rowing ergometer test in college rowers. *J. Sports Med. Phys. Fit.* (In Press)
9. **Jürimäe J., Mäestu J., Jürimäe T., Pihl E.** (2000) Prediction of rowing performance on single sculls from metabolic and anthropometric variables. *J. Hum. Mov. Studies* 38: 123–136.
10. **Jürimäe J., Jürimäe T., Pihl E.** (2000) Changes in the body fluids during endurance rowing training. *Ann. NY Acad. Sci.* 904: 353–358.
11. **Kuoppasalmi K., Naveri H., Rehunen S., Härkönen M., Adlercreutz H.** (1976) Effect of strenuous anaerobic running exercise on plasma growth hormone, cortisol, luteinizing hormone, testosterone, androstenedione, estrone and estradiol. *J. Steroid. Biochem.* 7: 823–829.
12. **Mader A., Hartmann A., Hollmann W.** (1986) Einfluss eines Höhentrainings auf die kardiopulmonale Leistungsfähigkeit in Meereshöhe, dargestellt am Beispiel der Deutschen Ruder-Nationalmannschaft. In: Hollmann W. (ed.), *Zentrale Themen der Sportmedizin*, pp. 276–290. Berlin, Heidelberg: Springer.

13. **Maresh C. M., Cook M. R., Cohen H. D., Graham C., Gunn, W. S.** (1988) Exercise testing in the evaluation of human responses to powerline fields. *Aviat. Space Environ. Med.* 59: 1139–1145.
14. **Obminski Z., Aniol-Strzyzewska K., Burkhard-Jagodzinska K., Krawczyk B., Malczewska U., Klusiewicz A., Hubner-Wozniak E.** (1997) Endocrine and metabolic responses to an indoor rowing competition in male and female elite athletes. *Biol. Sport* 14: 145–153.
15. **Russell A. P., LeRossignol P. F., Sparrow W. A.** (1998) Prediction of elite schoolboy 2000-m rowing ergometer performance from metabolic, anthropometric and strength variables. *J. Sports Sci.* 16: 749–754.
16. **Secher N. H.** (1993) Physiological and biomechanical aspects of rowing. *Sports Med.* 15: 24–42.
17. **Secher N. H., Vaage O., Jackson R. C.** (1982) Rowing performance and maximal aerobic power in oarsmen. *Scand. J. Sports Sci.* 4: 9–11.
18. **Schmidt W.** (1999) Blood volume and endurance performance. *Int. J. Sports Med.* 20: S20.
19. **Snegovskaya V., Viru A.** (1993) Elevation of cortisol and growth hormone levels in the course of further improvement of performance capacity in trained rowers. *Int. J. Sports Med.* 14: 202–207.
20. **Steinacker J. M.** (1993) Physiological aspects of training in rowing. *Int. J. Sports Med.* 14: S3–S10.
21. **Steinacker J. M., Lormes W., Lehmann M., Altenburg D.** (1998) Training of rowers before world championships. *Med. Sci. Sports Exerc.* 30: 1158–1163.
22. **Viru A.** (1995) Humoral functions and exercise. In: Torg JS, Shephard RJ (Eds) *Current Therapy in Sports Medicine*. Third Edition, pp. 501–506. Mosby-Year Book, Inc.

Correspondence to:

Jaak Jürimäe
Institute of Sport Pedagogy
University of Tartu
18 Ülikooli Street
50090 Tartu
Estonia

MUSCLE FATIGUE AND POST-TETANIC POTENTIATION IN BOYS AND ADULT MEN WHEN PERFORMING JUMPING EXERCISES OF MAXIMAL INTENSITY

A. Skurvydas, G. Mamkus

Laboratory of Human Motorics, Lithuanian Institute of Physical
Education, Kaunas, Lithuania,

ABSTRACT

The aim of the present study was to compare changes in twitch contractile properties of quadriceps femoris muscle in boys aged 12–14 years and adult men when performing jumping exercises of maximal intensity. The quadriceps femoris muscle was directly stimulated by single square-wave pulses in rest and after maximal voluntary isometric contraction (MVC) of 10 s duration (post-tetanic potentiation condition). Twitch peak force (Pt), contraction time (CT) and half-relaxation time (HRT), and MVC force of the quadriceps femoris muscle were measured before and after jumping test. The sum of CT and HRT (CT+HRT) was calculated. Bosco jumping test of 1 min duration performed on force platform. The power developed by leg muscles of adult men at the start of the Bosco jumping test and the decrease in power was greater ($p < 0.05$) than in boys. After the jumping test there was a greater decrease in Pt and MVC of adult men if compared with the corresponding values found in boys. CT+HRT values in boys increased to a greater ($p < 0.05$) extent than in adult men. Before jumping test twitch Pt potentiation was higher ($p < 0.05$) in men compared with boys. Besides there was actually hardly any change in Pt potentiation of adult men after the exercise while there was a considerable decrease in the corresponding values for boys if compared with the initial values though there was an increase in the CT+HRT potentiation of boys.

Key words: skeletal muscle, growth and maturation, contraction and relaxation, jumping, fatigue, post-tetanic potentiation

INTRODUCTION

There exists a great number of studies dealing with the peculiarities of growth and maturation of the skeletal muscles [4, 6, 8, 9, 10]. However, the phenomenon of the post-tetanic potentiation of matured muscles and muscle fatigue have been investigated quite thoroughly. Post-tetanic potentiation is characterised by an increase in peak twitch tension that occurs following tetanic tension development in the muscle and which rapidly decays following removal of the potentiating stimulus [14, 17]. Despite considerable research, the causes of muscle fatigue have yet to be clearly established [5, 14, 17]. The problem is complex, since multiple factors are clearly involved. It has been shown that the processes of post-tetanic potentiation and fatigue occur concurrently, beginning from the onset of activation [7, 14]. Still changes in the efficiency of an voluntary contraction and relaxation of the skeletal muscles induced by a single electrical stimulus as well as phenomena of muscle post-tetanic potentiation and their resistance to fatigue depending upon age are far from being clear and require further investigation.

The aim of the present study was to compare changes in twitch contractile properties of the quadriceps femoris muscle, including changes in post-tetanic potentiation in boys aged 12–14 years and adult men when performing jumping exercises of maximal intensity for 60 s (Bosco test).

METHODS

Subjects

The subjects were healthy adult men (aged 21–26 years, $n=15$) and 12–14 years-old boys ($n=12$) not engaged in sports. The boys attended physical education classes twice per week.

Apparatus and Experimental Procedure

Force measurements. The equipment and technique of measuring force of the quadriceps femoris muscle was described in our previous studies [12, 13]. Subjects sat upright on the experimental chair with a vertical back support provided. A strap secured the hips and thighs to minimise uncontrolled movements. The right leg was clamped in the force measuring device with the knee semi-flexed. A 6 cm wide plastic cuff, placed around the right leg just proximal to the malleoli, was tightly attached to a linear variable differential transducer. The output of the transducer, proportional to isometric knee extension force, was amplified and digitised at a sampling rate of 1 kHz by a 12-bit analogue-to-digital converter installed in an IBM-compatible personal computer. The digitised signal was stored on hard disk for subsequent analysis. The output from the force transducer was also displayed on voltmeter in front of the subject.

Electrical stimulation. Details of equipment and procedure for electrical stimulation were described previously [12, 13]. A high-voltage stimulator (MG 440, Medicor, Budapest, Hungary) was used. Electrical stimuli to the quadriceps muscle were delivered through surface electrodes (9×18 cm) padded with cotton cloth and soaked in saline solution. One stimulation electrode was placed just above the patella, while the other covered the large portion of the muscle belly in the proximal third part of the thigh. The electrical stimulation was always delivered in trains of square wave pulses of 1 ms duration (voltage 150 V). The subjects were introduced to electrical stimulation. The following data were measured: (1) twitch peak force (Pt), twitch contraction time (CT) and twitch half-relaxation time (HRT). Twitch post-tetanic potentiation (PTP) was evaluated as the percentage elevation in twitch parameters (Pt, CT and HRT) after 10-s maximal voluntary contraction (MVC) [13, 17]. The sum of CT and HRT (CT+HRT) was calculated.

Vertical jump performance. Each subject performed maximal voluntary vertical jumps on force platform PD-3A (Visti, Sankt-Peterburg, Russia). The force signal was registered and analysed with the help of personal computer IBM-compatible AT 486 per-

sonal computer. Heights of the jumps (H) were recorded by an earlier technique by applying the following formula [2]:

$$H = 1.1226 \times Tf^2$$

where Tf = flight time. Jumps with a preliminary counter-movement to 90 degrees angle in the knee (counter-movement jump, CMJ) were performed.

A Bosco jumping test for 1 min duration [1] was performed. To estimate the maximal mechanical power of the leg extensor muscles, the protocol required that the subject jumped continuously with maximal leg extension for a set period of 1 min. To standardise the knee angular displacement during contact phase, the subjects were required to bend the knee to about 60 degrees. Furthermore, to avoid unmeasurable work output, horizontal, and lateral displacements should be minimised and the hands must be kept on hips throughout. To calculate the average mechanical power (W) during 60 s the following formula was used [1]:

$$W = g2 \times Tf \times 60 / 4n \times (60 - Tf),$$

where W = mechanical power expressed in $W \times kg^{-1}$; g = acceleration of gravity; Tf = sum of the total flight time (s) of all jumps; n = number of jumps performed during the total 1 min work period. To follow the change in power output during the total jumping performance, power measurements were calculated for every 15 s period were extracted from the 1 min jumping test. Power output during first 15 s (W0-15) of jumping test and power output during last 15 s (W45-60) of jumping test were calculated.

The experimental protocol. Upon arriving at the laboratory, the subject was seated in the experimental chair and after 5 min muscle contractile properties were recorded in the following sequence: 1. Pt (3 times every 30 s), MVC force (top of the MVC was reached, held about 2 s and relaxation, MVC was reached 3 times every 1 min). 3. Post-tetanic twitch potentiation (PTP) was evaluated. Before testing the height of the vertical jump the subject performed warming-up which consisted of 10 min running on the spot with intensity that corresponded to heart rate 130-150 beats per minute. Then every 1 min the subjects performed 3 control jumps. After

testing the height of vertical jump jumping exercise of maximal intensity for 1 min were performed. One and 3 min following the end of the exercise the height of jump was established. After this control measuring of the height of jump the subjects were seated in the experimental chair again and the same muscle contraction and relaxation properties as before the exercise were tested. The testing of these properties was undertaken between 3 and 5 min following the end of jumping exercise. Besides, MVC force was measured two times.

Statistical analysis

Data are means and standard deviations (SD). The two-way analysis of variance (two-way ANOVA) for repeated measures was used to determine differences between the groups. One-way ANOVA for repeated measures were used to test statistical differences within each group (pre- vs. post fatigue). Student t test for paired data was used to determine any differences between separate measurements of parametric variables. Statistical significance was set at $p < 0.05$.

RESULTS

The power developed by leg muscles in adult men at the start of the jumping exercise (W0-15) and the rate of decrease in power was significantly ($p < 0.05$) greater than in 12-14-year-old boys (Table 1). The jumping height in boys was restored during 1 min significantly ($p < 0.05$) sooner than in adult men. After 3 min of the end of the test the height of the jump in boys and adult men was not differed from the control value ($p > 0.05$).

Table 1. Mean (SD) values of the vertical jumping characteristics of 12–14 year-old boys and adult men.

Groups	W0–15 (W/kg)	W0–15 – W45–60/ W0–15 × 100 (percent)	Percent of height of vertical jump 1 min after jumping to initial level	Percent of height of vertical jump 3 min after jumping to initial level
Boys	13.2 (3.2)	41.2 (6.5)	92.8 (6.1)	97.8 (6.4)
Adult men	19.8 (2.2)	51.8 (9.1)	83.1 (5.6)	96.4 (6.7)
P	<0.05	<0.05	<0.05	>0.05

Notes: W0–15 and W45–60 — power output during first 15 s of jumping test and power output during last 15 s of jumping test, respectively.

Before performing jumping test Pt and MVC force in adult men was greater ($p < 0.05$) while CT+HRT was smaller ($p < 0.05$) than in boys (Table 2). Besides post-tetanic potentiation of twitch force was greater ($p < 0.05$) in men than in boys. After retaining the MVC force for 10 s however there was a significantly ($p < 0.05$) expressed decrease CT+HRT in boys while in men this value remained unchanged ($p > 0.05$).

Table 2. Mean (SD) values of MVC force and twitch contractile properties of the quadriceps femoris muscle in boys (B) and men (A) before and after the jumping exercise.

	Pt (N)		PTP Pt (%)		CT+HRT (ms)		PTP CT+RT (%)		MVC force (N)	
	B	A	B	A	B	A	B	A	B	A
Before test	57 (8)	74* (10)	195 (44)	261* (47)	153* (15)	130 (18)	86 (11)	100* (10)	413 (50)	924* (105)
After test	46* (5)	25 (4)	154 (32)	292* (59)	179* (16)	124 (17)	68 (11)	110* (18)	352 (43)	669* (108)
P	<0.05	<0.05	<0.05	>0.05	<0.05	>0.05	<0.05	>0.05	<0.05	<0.05
After/ before (%)	84* (11)	36 (6)	83 (21)	120* (24)	118* (15)	97 (20)	78 (11)	109* (19)	86 (8)	74* (11)

Note. * $p < 0.05$ between the men and boys.

After performing jumping test there was a greater ($p < 0.05$) decrease in Pt and MVC force of adult men and boys but there was a

statistically significantly ($p < 0.05$) greater decrease in Pt and MVC force of adult men if compared with the corresponding values found in boys (Table 2). Besides, CT+HRT values in boys increased ($p < 0.05$) while in adult men CT+HRT was unchanged ($p > 0.05$) after jumping exercise. It is of interest to note that there was actually hardly any change in post-tetanic potentiation Pt of adult men after the exercise while there was a considerable decrease in the corresponding values for boys if compared with the initial values though there was an increase ($p < 0.05$) in the CT+HRT potentiation of boys.

DISCUSSION

The first finding of our study is that after performing jumping test there was a significantly greater decrease in Pt and MVC force of adult men compared to boys. CT+HRT in boys prolonged to a greater extent than the corresponding values found in adult men. The second finding of our study is that before jumping test post-tetanic twitch force potentiation was significantly greater in men than in boys. Besides there was actually hardly any change in Pt potentiation of adult men after the exercise while there was a considerable decrease in the corresponding values for boys if compared with the initial values though there was a prolongation of potentiated CT+HRT in boys.

It has been established that in the period of puberty a particularly intensive synthesis of testosterone takes place [10] and it especially contributes to the protein synthesis of muscle fibres of the fast-twitch type [6] though another opinion has also been voiced, namely, that in the process of the growth of the human organism the ratio of muscle fibres of fast-twitch type and the slow-twitch type remains unchanged [4]. The sarcoplasmic reticulum (SR), where the calcium ions are situated, is more developed in muscle fibres of the fast-twitch type. The more developed the SR the greater the ratio and amount of the calcium ions released, consequently, the greater should be Pt and the shorter should be CT and

HRT [5]. The results of the research carried out by these scientists account for the fact why the values Pt, MVC force and (W0-15) are smaller and the corresponding value of CT+HRT is longer in boys than in men.

It is believed that after maintaining the maximal contraction force of the muscle the release of Ca^{2+} from SR into the myoplasm takes place which in its turn activates the phosphorylation of the light chains (LC) of myosin [15]. Due to phosphorylation of the LC of myosin the transformation rate of the myosin cross-bridges from weak state to strong state improves [15]. With this mechanism in action during the same period of time there is attachment of greater number of myosin cross-bridges with actin which contributes to an increase in the muscle contraction force, the more so in the case of the small Ca^{2+} concentration [15]. It has been established that muscles of the fast-twitch type have greater post-tetanic potentiation [16]. If this is the case it becomes quite understandable why the Pt potentiation of men is greater than that of boys whose muscles do not contain yet fully matured muscle fibres of the fast-twitch type [8].

It is not quite clear why after 10 s of maintaining MVC, HRT and CT values for boys become shorter while in the case of men the corresponding values remained unchanged. Due to phosphorylation of the LC of myosin the detachment of myosin cross-bridges from actin becomes worse [15]. Therefore, after 10 s of maintaining MVC, RT should become longer but in our case RT for boys shortened. Muscle relaxation rate depends not only on the detachment rate of myosin cross-bridges from actin but on their amount as well. The greater the amount of Ca^{2+} the greater the number of myosin cross-bridges subjected to attachment and the longer it takes Ca^{2+} to transform into SR [5, 17]. After 10 s maintaining MVC some portion of calcium ions may be coupled up together with parvalbumin may cause the worsening of muscle relaxation [17]. It is believed that following the start of the exercise the amount of Ca^{2+} in the myoplasm should be increase [17] and because of this the RT should be longer. Therefore the fact that CT+HRT values become shorter in the case of the experiment with boys cannot be accounted for by phosphorylation of myosin LC

and Ca^{2+} kinetics alone, the more so that our knowledge about the changes taking place in the parvalbumin during the period of muscle growth and maturation is still rather scarce.

Phosphorylation of the myosin LC is not the only mechanism activating muscle work, the more so that scientists do not point to any direct relation between phosphorylation of the myosin LC and increase in Pt [7]. This relation could be distorted by a number of other factors. For example, with the increase in the temperature of muscles the rate of the muscle contraction and relaxation improves [5]. There is no doubt whatever that after 10 s of maintaining MVC the muscle temperature rises and this causes the shortening of HRT. Besides, after maximal muscular efforts the mechanisms of transmission of the muscle force signal can remain unchanged for some time. For examples, the smaller values of elasticity of the muscle, and the shorter the values of HRT and CT.

The subjects had to perform jumping exercise which cause the accumulation of metabolites in the muscles [1]. With an increase in the concentration of hydrogen ions and non-organic phosphate the number of myosin bridges attached to actin decreases and the force developed by them decreases [3, 5, 11]. Besides, due to the some reason the amount of the Ca^{2+} released from SR and the sensitivity of myofibrils to Ca^{2+} decreases [3, 11]. Therefore the curve of contraction force depending upon Ca^{2+} concentration moves to the right [11] and this circumstance calls forth a particular decrease in the muscle contraction force at low stimulation frequencies. It is quite understandable therefore why there was a greater decrease in Pt than MVC force in the case of adult men (Table 2). Besides, prolonged values of CT+HRT for boys after the exercise can be explained by accumulation of metabolites. The fact that the anaerobic way of ATP resynthesis predominates in the muscles of adult men if compared with the muscles of boys accounts for a greater decrease of MVC force and Pt in men. It is not clear at all however why the CT+HRT prolonged more in the case of boys than in the case of men.

Two main mechanisms, i.e. post-tetanic potentiation and muscle fatigue are in competition during physical exercise [7, 14]. Therefore the efficiency of muscle contraction and relaxation depends on

the interaction of the mechanisms of fatigue and potentiation [14]. The more intensive and the shorter the work performed by the muscles the more active are the mechanisms of potentiation [14]. Since our subjects have been performing a rather intensive work post-tetanic potentiation might have also had some influence on Pt registered after work. This is in accordance with the data received by some others authors [7]. It has been established that after maximal efforts of the quadriceps m. femoris for 60 s a great amount of myosin LC is phosphorylated [7]. Pt, however, is considerably decreased due to metabolic changes. The fact that post-tetanic potentiation was retained both in men and in boys during fatigue coincides with the results of the research carried out by other scientists [7, 14]. The subjects in those cases though were adult men. It is not clear however why because of fatigue there was a decrease of Pt in boys while PTP remained unchanged. Besides, it is difficult to explain why during fatigue there was a shortening of CT+HRT due to PTP only in boys while this value in adult men remained unchanged.

By way of summing up it can be asserted that after jumping exercise of maximal intensity MVC force and Pt were more reduced in adult men than in boys and, what is rather strange, the muscle CT+HRT was more prolonged in boys than in the case of adult men. Besides there was actually hardly any change in Pt potentiation of adult men after the exercise while there was a considerable decrease in the corresponding values for boys if compared with the initial values though there was a prolongation of potentiated CT+HRT in boys. Thus, it can be maintained that post-tetanic potentiation both in boys and in adult men can partially compensate the muscle fatigue generated while performing the jumping exercise.

REFERENCES

1. **Bosco C., Luhtanen P., Komi P. V.** (1983) A simple method for measurement of mechanical power in jumping. *Eur. J. Appl. Physiol.* 50: 273-282.
2. **Bosco C., Viitasalo J. T., Komi P. V., Luhtanen, P.** (1982) Combined effect of elastic energy and myoelectrical potentiation during stretch-shortening cycle exercise. *Acta Physiol. Scand.* 114: 557-565.
3. **Edman K. A. P., Lou F.** (1990) Change in force and stiffness induced by fatigue and intracellular acidification in frog muscle fibres. *J. Physiol. (Lond.)* 424: 133-149.
4. **Eriksson B. O., Golnick P. D., Saltin B.** (1973) Muscle metabolism and enzyme activities after training in boys 11-13 years old. *Acta Physiol. Scand.* 87: 485-497.
5. **Fitts R. H.** (1994) Cellular mechanisms of muscle fatigue. *Physiol. Rev.* 7: 49-95.
6. **Glenmark B., Hedberg G., Jansson E.** (1992) Changes in muscle fiber type from adolescence to adulthood in women and men. *Acta Physiol. Scand.* 146: 251-259.
7. **Grange R. W., Houston M. E.** (1991) Simultaneous potentiation and fatigue in quadriceps after 60-second maximal voluntary isometric contractions. *J. Appl. Physiol.* 70: 229-242.
8. **Jansson E.** (1996) Age-related fiber type changes in human skeletal muscle. In: *Biochemistry of Exercise IX* (eds. Maughan R.J.). Champaign, IL: Human Kinetics. 297-307.
9. **Kovanen V., Suominen H.** (1989) Age- and training-related changes in the collagen metabolism of rat skeletal muscle. *Eur. J. Appl. Physiol.* 58: 765-771.
10. **Malina R. M., Bouchard C.** (1991) *Growth, Maturation and Physical Activity*. Champaign, IL: Human Kinetics, 1991.
11. **Metzger J. M., Moss R. L.** (1987) Greater hydrogen ion-induced depression of tension and velocity in skinned single fibers of rat fast than slow muscles. *J. Physiol.* 393: 727-742.
12. **Ratkevicius A., Skurvydas A., Lexell J.** (1995) Submaximal-exercise-induced impairment of human muscle to develop and maintain force at low frequencies of electrical stimulation. *Eur. J. Appl. Physiol.* 70: 294-300.
13. **Ratkevicius A., Skurvydas A., Mamkus G.** (1997) The twitch contractile properties of quadriceps muscle in athletes. *Kinesiology* 29: 17-20.

14. **Skurvydas A., Stasiulis A., Jascaninas J.** (1997) Theoretical analysis of mechanisms determining skeletal muscle fatigue. *Antropomotoryka* 16: 155–161.
15. **Sweeney H. L., Stull J. T.** (1990) Alteration of cross-bridge kinetics by myosin light chain phosphorylation in rabbit skeletal muscle: implication for regulation of actin-myosin interaction. *Proc. Natl. Acad. Sci. USA.* 87: 414–418.
16. **Vandervoort A. A., Quinlan J., McComas A. J.** (1983) Twitch potentiation after voluntary contraction. *Exp. Neurol.* 81: 141–152.
17. **Westerblad H., Lee J. A., Lännergren J., Allen D. G.** (1991) Cellular mechanisms of fatigue in skeletal muscle. *Am. J. Physiol.* 261: 195–209.

Correspondence to:

Albertas Skurvydas
Laboratory of Human Motorics
Lithuanian Institute of Physical Education
Sporto 6
Kaunas 3029
Lithuania

GENDER DEPENDENT ASSOCIATION BETWEEN DIFFERENT ESTIMATES OF BODY FAT DISTRIBUTION AND BLOOD PRESSURE IN OBESE CHILDREN AND ADOLESCENTS

K. Sudi*, S. Gallistl**, R. Möller***, E. Tafait***,
M. H. Borkenstein****

* Institute of Sport Sciences, Karl-Franzens University

** Ludwig-Boltzmann Research Institute for Pediatric Hemostasis and Thrombosis, Department of Pediatrics, Karl-Franzens University

*** Institute for Medical Chemistry and Pregl-Laboratory, Karl-Franzens University

**** Division for Diabetes and Endocrinology, Department of Pediatrics, Karl-Franzens University, Graz, Austria

ABSTRACT

We measured different estimates of body fat distribution and explored their association with systolic (SBP) and diastolic blood pressure (DBP) in obese children and adolescents. 29 obese boys [mean \pm SD; age: 11.5 \pm 2.7 yrs., body mass index (BMI): 26.6 \pm 3 SBP: 121 \pm 14.8 mm Hg, DBP: 60.4 \pm 7.4 mm Hg] and 56 obese girls (age: 12 \pm 1.9 yrs., BMI: 27.7 \pm 5.25, SBP: 122.1 \pm 13.4 mm Hg, DBP: 62.3 \pm 10.8 mm Hg) were studied. Fat mass (FM) was estimated by means of bioelectrical impedance. Waist (Wc) and hip circumference (Hc) were measured and waist-to-hip ratio (WHR) was calculated. Subcutaneous adipose tissue layers (SAT-layers) were measured by means of the optical device Lipometer at 15 different body sites (from *1-neck* to *15-calf*) on the right side of the body. In boys, significant correlations were found between either SBP and age, body weight, BMI and FM. Wc and Hc but not WHR were correlated to SBP. DBP was associated with body weight, BMI and Hc. SAT-layers *12-lateral thigh* and *15-calf* were inversely correlated to SBP.

DBP was not associated with any of the measured SAT-layers. In girls, SBP and DBP were correlated to body weight, BMI, FM, Wc and Hc but not to WHR. SBP was correlated to SAT-layers *1-neck*, *3-biceps*, and *6-lateral chest* whereas DBP was correlated to SAT-layers *1-neck* and *4-upper back*. Multiple regression revealed that after control for body weight, SAT-layers *5-front chest* and *6-lateral chest* contributed to the variation in SBP in boys (adj. $R^2=0.696$, $p<0.0001$). In girls, body weight contributed independently to the variation in SBP (adj. $R^2=0.167$, $p=0.0012$). Body weight together with SAT-layers *4-upper back*, *5-front chest*, and *12-lateral thigh* contributed to the variation in DBP (adj. $R^2=0.346$, $p<0.0001$). The results imply that different adipose tissue depots might contribute in a gender dependent mechanism to the variation in blood pressure. Nevertheless, body weight is a main and independent determinant for blood pressure in childhood and juvenile obesity.

Key words: blood pressure, body fat distribution, obesity, children, Lipometer, gender

INTRODUCTION

Obesity in childhood often results in an increased morbidity and mortality in later life [15]. The increase in body weight and body fat mass is associated with worsening in cardiovascular risk [10]. An excess of adipose tissue might constitute the metabolic syndrome which can be described as a cluster of metabolic abnormalities [4] among which systolic blood pressure has been shown to be related to the extent of fibrous-plaque lesions and fatty streak [3].

Hypertension and being overweight were shown to exert additive effects in increasing insulinemia [8] and an impairment in insulin sensitivity contribute to the age-related elevation in blood pressure [14]. Fasting plasma insulin may be used as a marker for the development of obesity-associated metabolic disorders and elevated blood pressure in children [17]. However, there is also evidence to assume an independent association of body fat distribution with blood pressure [7]. Body fat distribution, either determined by the waist circumference [9], waist-to-hip ratio and skinfolds [13], dual-energy x-ray absorptiometry (DEXA) [24] or mag-

netic resonance imaging (MRI) [6], were shown to be independently and significantly linked with systolic and diastolic blood pressure. On the other hand, there are also data available which do not support an independent relationship of body fat distribution on blood pressure [5, 16, 22]. Whether this discrepancy can be explained by a gender-dependent difference [1] or by the different estimates of body fat distribution used so far, is not clear.

We have recently shown that the optical device Lipometer measures a subcutaneous monolayer in a rapid, safe, and highly precise manner [18, 19] and further enables. In this study, we addressed the question whether subcutaneous adipose tissue layers or commonly used anthropometric estimates of body fat distribution (waist- and hip circumference) were differently associated with blood pressure. A second aim was to find out whether any of the above mentioned estimates of body fat distribution is also an independent determinant for blood pressure in obese children and adolescents after adjustment for body weight and body fat mass.

SUBJECTS AND METHODS

Subjects

The whole study group comprised 29 boys (mean \pm SD; age: 11.5 \pm 2.7 yrs., body mass index, BMI: 26.6 \pm 3) and 56 girls (age: 12 \pm 1.9 yrs., BMI: 27.7 \pm 5.25). Obesity was defined as a BMI \geq 90th percentile for age and sex. Medical examination of children revealed no history of severe illness or other exclusion criteria.

Resting blood pressure was measured in sitting position after a 15-min rest using a mercury sphygmomanometer. Anthropometric characteristics and values of systolic (SBP) and diastolic blood pressure (DBP) are shown in table 1.

Table 1. Anthropometric characteristics, measures of adiposity, parameters of body fat distribution, and values of systolic and diastolic blood pressure for the whole study group and for boys and girls separately. P accounts for the gender difference between variables. Differences were tested by means of ANOVA.

Parameters	All children (n=85)	Boys (n=29)	Girls (n=54)	P Anova
Age	11.8±2.2	11.5±2.7	12±1.9	0.44*
Height (cm)	156.3±11.35	156.1±14.8	156.4±9.2	0.85*
Weight (kg)	68±18.1	66.15±17.7	68.9±18.4	0.51
BMI	27.3±4.6	26.6±3	27.7±5.25	0.335*
Systolic BP	121.9±13.8	121.4±14.8	122.1±13.4	0.84
Diastolic BP	61.7±9.8	60.4±7.4	62.3±10.8	0.59*
Waist circf. (cm)	87.1±13.5	88.4±11.2	86.4±14.6	0.53
Hip circf. (cm)	94.05±13.3	92.9±10.5	94.7±14.5	0.56
WHR	0.93±0.05	0.95±0.05	0.91±0.05	0.0007
FM (kg)	31.3±12	29.4±10	32.2±12.85	0.3
%FM	44.75±7.95	43.8±7.4	45.2±8.3	0.45

* differences were tested by means of Kruskal-Wallis test.

Anthropometric estimate of body fat distribution

Waist- (Wc) and hip circumference (Hc) were measured in triplicate to the nearest 0.5 cm and the median was taken. The waist-to-hip ratio (WHR) was calculated as Wc/Hc^{-1} (table 1).

Assessment of body composition

Fat free mass of children was estimated [21] by means of bioelectrical impedance (BIA Akern-RJL 101/S) with an applied current of 50 kHz in the fasting state after children were resting in supine position [2]. Fat mass (FM) was calculated as the difference between body weight and FFM. Percentage FM (%) was expressed as the relative amount of FM for a given body mass (table 1).

Determination of subcutaneous adipose tissue layers (SAT-layers)

SAT-layers were determined by means of the optical device Lipometer [19]. Briefly, the Lipometer uses light emitting diodes which illuminate a selected SAT-layer, forming certain geometrical patterns that vary in succession. A photodiode measures the light intensities that are backscattered in the subcutaneous adipose tissue. These light signals are amplified, digitised and stored in a computer. Calibration and evaluation were done using CT as the reference [18].

Measurement for the thickness of SAT-layers in mm were performed at 15 specified body sites, from *1-neck* to *15-calf*, on the right side of the body in standing position [18] (table 2). The coefficients of variation of SAT-layers are ranging between 1.9% for SAT-layer *5-front chest* and 12.2% for SAT-layer *13-rear thigh* [21].

Table 2. Values of the thickness of measured SAT-layers (from *1-neck* to *15-calf*). P accounts for the gender difference in measured SAT-layers (by means of ANOVA).

Measured SAT-layers (in mm)	All children	Boys	Girls	P Anova
1-neck	16.55±6.75	16.7±6.85	16.5±6.75	0.89
2-triceps	17.9±5.5	17.3±5.9	18.2±5.4	0.49
3-biceps	10.9±4.5	10.1±3.8	11.2±4.75	0.28
4-upper back	16.3±6.65	15.8±6.6	16.6±6.7	0.63
5-front chest	22.8±7.8	22.1±6.1	23.2±8.6	0.58*
6-lateral chest	19.2±7.4	17.8±6	20±8	0.198
7-upper abdomen	24.2±6.7	25.9±6.4	23.3±6.8	0.10
8-lower abdomen	18.1±5.3	19.4±5.2	17.4±5.3	0.105
9-lower back	19.3±6.5	19.5±5.65	19.2±7	0.83
10-hip	22.1±6	22.7±6.3	21.8±5.9	0.53
11-front thigh	10.4±3.7	11.5±4.2	9.8±3.3	0.044
12-lateral thigh	11.9±3.3	12.4±3.8	11.6±3	0.31
13-rear thigh	7.45±3.3	9.3±4.25	6.5±2.25	0.0037*
14-inner thigh	14.5±3.6	14.8±3.4	14.4±3.8	0.61
15-calf	6.9±2.1	7.9±2.4	6.4±1.8	0.0015

Statistics

Kolmogorov-Smirnov test and Chi-Square test were used to prove normality of data. Analysis of variance was used to compare parameters between groups where appropriate. In case of a significant difference, the *post-hoc* Bonferroni correction was employed. Kruskal-Wallis test was used if variances were not normally distributed. Correlations between variables of interest were calculated using Pearson's correlation coefficient and partial correlation was performed to adjust for the influence of confounding variables. Based on the results of the bivariate correlations, the independence and significance of variables was tested by stepwise, multiple regression analysis. The significance level of P-values was set at 5%. Values are shown as mean and standard deviation.

RESULTS

Most variables were not significantly different between boys and girls (table 1). However, WHR was significantly higher in boys (0.95 ± 0.05) than in girls (0.91 ± 0.05 , $p=0.0007$). Measured SAT-layers from the upper body and the trunk were not different between boys and girls (table 2). SAT-layers *11-front thigh* ($p=0.044$), *13-rear thigh* ($p=0.0037$), and *15-calf* ($p=0.0015$) showed significantly greater values in boys than in girls (Table 2).

Pearson correlation between estimates of adiposity and blood pressure:

A significant association was found between either SBP and DBP with body mass, BMI and FM in boys and girls (table 3). However, only in girls % FM was significantly associated with SBP and DBP (both $p<0.01$; table 3). When all children were combined, chronological age was correlated to SBP ($r=0.32$, $p=0.0016$) and DBP ($r=0.23$, $p=0.019$). After adjustment for age, all estimates of adiposity were significantly correlated to SBP and DBP in all children (all $p<0.01$, table 3).

Table 3. Pearson product moment correlation shows the association between anthropometric characteristics, different estimates of body fat distribution (Waist circf., Waist circumference; Hip circf., Hip circumference, 15 measured SAT-layers), and systolic and diastolic blood pressure in boys and girls. The first coefficient accounts for the association of the variable with systolic blood pressure, the second coefficient for diastolic blood pressure. Partial correlation was performed in all children with chronologic age as confounding variable.

Parameters	Boys	Girls	All children (adj. Age)
Body mass	0.71‡ / 0.39*	0.43† / 0.40†	0.43‡ / 0.34†
BMI	0.61† / 0.37*	0.43† / 0.44†	0.36† / 0.38†
FM	0.64† / 0.37*	0.41† / 0.39†	0.37† / 0.32†
%FM	0.22 / 0.20	0.35† / 0.35†	0.28† / 0.30†
Waist circf.	0.52† / 0.28	0.37† / 0.39†	0.30† / 0.29†
Hip circf.	0.56† / 0.36*	0.40† / 0.38†	0.33† / 0.31†
1-neck	-0.10 / 0.10	0.39† / 0.38†	0.19* / 0.28†
2-triceps	-0.24 / -0.29	0.09 / 0.11	-0.10 / -0.04
3-biceps	-0.19 / -0.14	0.31* / 0.19	0.08 / 0.07
4-upper back	-0.25 / -0.15	0.21 / 0.35†	0.04 / 0.22*
5-front chest	0.23 / 0.16	0.16 / 0.09	0.19* / 0.11
6-lateral chest	-0.21 / 0.03	0.29* / 0.15	0.09 / 0.09
7-upper abdomen	-0.20 / -0.04	-0.05 / 0.06	-0.02 / 0.09
8-lower abdomen	-0.10 / 0.06	-0.11 / -0.15	0.02 / -0.02
9-lower back	-0.31 / -0.12	0.03 / 0.20	-0.01 / 0.18
10-hip	-0.27 / -0.12	-0.08 / -0.02	-0.07 / 0.01
11-front thigh	-0.24 / -0.14	0.09 / 0.20	0.025 / 0.13
12-lateral thigh	-0.37* / 0.05	-0.07 / 0.175	-0.13 / 0.18
13-rear thigh	-0.23 / -0.24	-0.19 / -0.01	-0.13 / -0.07
14-inner thigh	-0.07 / -0.11	-0.06 / 0.05	-0.03 / -0.25*
15-calf	-0.58† / -0.27	-0.14 / -0.03	0.04 / -0.07

* $p < 0.05$, † $p < 0.01$ ‡ $p < 0.0001$

Pearson correlation between estimates of body fat distribution and blood pressure:

Waist circumference (Wc) and hip circumference (Hc) were significantly correlated to SBP in boys and girls (all $p < 0.01$, table 3).

However, the association between waist circumference and DBP was significant in girls but not in boys. WHR was not associated with SBP and DBP (data not shown).

After adjustment for age, Wc and Hc were significantly correlated to SBP and DBP at a p-level <0.01 in all children (table 3).

Only two measured SAT-layers (*12-lateral thigh*, $p<0.05$ and *15-calf*, $p<0.01$) were inversely correlated to SBP in boys (table 3). In girls, SAT-layer *1-neck* was correlated to SBP and DBP (both $p<0.01$), whereas SAT-layers *3-biceps* and *6-lateral chest* were correlated to SBP (both $p<0.05$). SAT-layer *4-upper back* was associated with DBP ($p<0.01$).

In all children, SAT-layer *1-neck* was correlated to SBP and DBP after adjustment for age (table 3). However, SAT-layer *5-front chest* was correlated to SBP ($p<0.05$) whereas SAT-layers *4-upper back* and *14-inner thigh* (negative association) were correlated to DBP.

Multiple regression analysis:

Body mass contributed significantly to the variation in SBP and DBP in boys and in girls (table 4). However, estimates of body fat distribution contributed significantly to the variation in SBP in boys but not in girls. The regression model which included body mass together with SAT-layers *5-front chest* and *6-lateral chest*, explained 70% of the variation in SBP in boys (adj. $R=0.83$, $p<0.0001$) (table 4).

In girls, estimates of body fat distribution contributed independently to the variation in DBP (table 4). The best fitting model, which included Wc together with SAT-layers *4-upper back*, *5-front chest*, and *12-lateral thigh* explained 35% of the variation in DBP (adj. $R=0.59$, $p<0.0001$).

Table 4. Stepwise, multiple regression analysis with systolic and diastolic blood pressure as dependent variables. Calculations were performed for boys and girls separately. β regression coefficient, SE standard error, adj. R^2 adjusted R^2 accounts for the percentage of the dependent variable(s) to contribute to the variability of the independent variable, adjusted for the number of the dependent variables included in the regression model.

	Dependent Variable: Systolic Blood Pressure				Dependent Variable: Diastolic Blood Pressure			
	Independent Variables	β	SE	adj. R^2	Independent Variables	β	SE	adj. R^2
Boys (n=29)	Body mass	0.647	0.127	0.487	Body mass	0.178	0.084	0.12
	<u>Intercept: 77.25; p<0.0001</u>				<u>Intercept: 48.2; p<0.0001</u>			
	Body mass	0.65	0.103	0.487				
	SAT-layer 6	-1.45	0.33	0.61				
Girls (n=56)	SAT-layer 5	0.923	0.335	0.696				
	<u>Intercept: 82.58; p<0.0001</u>							
	Body mass	0.307	0.09	0.167	Body mass	0.424	0.089	0.145
	<u>Intercept: 100.82; p<0.0001</u>				SAT-layer 11	1.42	0.41	0.248
					SAT-layer 2	-0.658	0.29	0.304
					<u>Intercept: 31.16; p<0.0001</u>			
					Waist circf.	0.495	0.121	0.138
				SAT-layer 12	1.06	0.466	0.247	
				SAT-layer 5	-0.73	0.236	0.30	
				SAT-layer 4	0.63	0.296	0.346	
				<u>Intercept: 13.99; p<0.0001</u>				

DISCUSSION

We studied the association between different estimates of body fat distribution and blood pressure in obese children and adolescents. We found an independent influence of different subcutaneous adipose tissue layers (SAT-layers) on systolic blood pressure (SBP) in boys. In girls, waist circumference (Wc) together with three SAT-layers contributed independently to the variation in diastolic blood pressure (DBP). However, body mass was a main determinant for the variation in SBP and DBP in boys and girls.

It has been shown that a centripetal fat pattern, characterized by the logarithm of the ratio of the subscapular to lateral calf skinfold thicknesses had a small but significant association with systolic blood pressure in men after adjustment for age and adiposity [1]. However, centripetal fat pattern was associated with an abnormal lipid profile in women, raising the possibility that gender dependent differences may modulate the association between total adiposity and age on blood pressures [1]. In an obese population (age range 15–71 years), correlations between mean arterial blood pressure and measures of body composition and body fat distribution retained significant after control for insulin levels [23]. In obese women, the waist-to-hip ratio (WHR) was significantly correlated to systolic and diastolic blood pressure [11] whereas others failed to find an independent relationship between fat patterning and blood pressure in adolescents [22].

We found that the well documented link between body mass and blood pressure is also present in obese boys and girls of different age (table 3). However, the relationship between estimates of adiposity and either SBP and DBP were almost of the same magnitude in girls but not in boys. In boys, percentage fat mass (%FM) was no associate of blood pressures. Whether the relative small number of obese boys studied ($n=29$) may account for this, or a existing relationship between body composition and blood pressure was masked by the fact that those boys were at different stages of biological maturation, is not clear. Nevertheless, waist- (Wc) and hip circumference (Hc) were correlated to SBP in both genders suggesting that adipose tissue from different anatomical origin

contribute to SBP. The resulting ratio of both circumferences (WHR) was no correlate of either SBP and DBP. It is questionable whether WHR is a valid estimate to describe a centripetal fat patterning in the state of childhood and adolescent obesity. It has been shown that waist circumference is an useful correlate of visceral adipose tissue accumulation not to be influenced by gender or by the degree of obesity [12]. This, in part could have contributed to the findings in girls. In girls, *Wc* was an independent associate of DBP in the body fat distribution regression model but not after adjustment for body mass (table 4). We used the newly developed optical device Lipometer [18] to measure the thickness of a subcutaneous monolayer at 15 different body sites to describe the distribution of subcutaneous adipose tissue [19, 20]. In boys, only SAT-layers *12-lateral thigh* and *15-calf* were significantly and inversely correlated to SBP. In girls, SAT-layers *1-neck*, *3-biceps*, and *6-lateral chest* were significantly and positively correlated to SBP (table 3). Whether this result suggest that an increase in SAT-layers from the upper body has a biological relevance for an increase in SBP in girls, remains to be elucidated. However, SAT-layers contributed independently to the variation in SBP in boys and to DBP in girls after adjustment for body mass (table 4). This suggest that the distribution of subcutaneous adipose tissue is associated with either SBP or DBP in a gender dependent way. It is unlikely that a different pattern of SAT-layers between boys and girls is responsible for this assumption. Only two SAT-layers from the lower extremities were different between boys and girls (table 2). This indicates that the distribution of SAT-layers from the upper body and the trunk does not strongly depend on gender and, probably, stages of maturation in these children. Combined, whether this could be a sign for a gender-different biological mechanism of subcutaneous adipose tissue distribution on the regulation of blood pressure, remains to be elucidated. Longitudinal studies are therefore warranted to investigate the relationship of SAT-layers with metabolic variables known to mediate some of the effects of body fat and body fat distribution on blood pressure, such as, e.g., insulin, glucose and leptin.

In conclusion, we found that different SAT-layers, measured by means of the optical device Lipometer, are associated with systolic blood pressure in obese boys and with diastolic blood pressure in obese girls. Nevertheless, body mass is a main and independent contributor to the variation in blood pressure in the state of childhood and juvenile obesity.

REFERENCES

1. **Baumgartner R. N., Roche A. F., Chumlea W. C., Siervogel R. M., Glueck C. J.** (1987) Fatness and fat patterns: association with plasma lipids and blood pressures in adults, 18 to 57 years of age. *Am. J. Epidemiol.* 126: 614–628.
2. **Baumgartner R. N.** (1996) Electrical impedance and total electrical body conductivity. In: *Human Body Composition*, A. F. Roche, S. B. Heymsfield, T. G. Lohmann (eds). Champaign, IL: Human Kinetics, 79–107.
3. **Berenson G. S., Srinivasan S. R., Bao W., Newman III W. P., Tracy R. E., Wattigney W. A.** (1998) Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. *New Engl. J. Med.* 338: 1650–1656.
4. **Björntorp P.** (1998) Etiology of the Metabolic Syndrome. In: *Handbook of Obesity*, G. A. Bray, C. Bouchard, W. P. T. James (eds). Marcel Dekker, pp 573–600.
5. **Bonora E., Targher G., Branzi P., Zenere M., Saggiani F., Zenti M. G., Travia D., Tonoli M., Muggeo M., Cigolini M.** (1996) Cardiovascular risk profile in 38-year and 18-year-old men. Contribution of body fat content and regional fat distribution. *Int. J. Obes.* 20: 28–36.
6. **Caprio S., Hyman L. D., Mc Carthy S., Lange R., Bronson M., Tamborlane W. V.** (1996) Fat distribution and cardiovascular risk factors in obese adolescent girls: importance of intraabdominal fat depot. *Am. J. Clin. Nutr.* 64: 12–17.
7. **Daniels S. R., Morrison J. A., Sprecher D. L., Khoury P., Kimball T. R.** (1999) Association of body fat distribution and cardiovascular risk factors in children and adolescents. *Circulation* 99: 541–545.

8. **Feldstein C. A., Renauld A., Akopian M., Olivieri A. O., Garrido D.** (1998) Relationship between hyperinsulinemia and ambulatory blood pressure monitoring of lean and overweight male hypertensive. *J. Cardiovasc. Risk* 5: 25–30.
9. **Flodmark C. E., Sveger T., Nilsson-Ehle P.** (1994) Waist measurement correlates to a potentially atherogenic lipoprotein profile in obese 12–14-year old children. *Acta Paediatr.* 83: 941–945.
10. **Gidding S. S., Bao W., Srinivasan S. R., Berenson G. S.** (1995) Effects of secular trends in obesity on coronary risk factors in children: The Bogalusa Heart Study. *J. Pediatr.* 127: 868–874.
11. **Kalkhoff R. K., Hartz A. H., Rupley D., Kissebah A. H., Kelber S.** (1983) Relationship of body fat distribution to blood pressure, carbohydrate tolerance, and plasma lipids in healthy obese women. *J. Lab. Clin. Med.* 102: 621–627.
12. **Lemieux S., Prud'homme D., Bouchard C., Tremblay A., Després J-P.** (1996) A single threshold value of waist girth identifies normal-weight and overweight subjects with excess visceral adipose tissue. *Am. J. Clin. Nutr.* 64: 685–693.
13. **Lurbe E., Alvarez V., Liao Y., Tacons J., Cooper R., Cremades B., Torro I., Redon J.** (1998) The impact of obesity and body fat distribution on ambulatory blood pressure in children and adolescents. *Am. J. Hypertens.* 11: 418–424.
14. **Miyazaki Y., Hirata A., Murakami H., Fukuoka M., Agata J., Higashiura K., Masuda A., Ura N., Shimamoto K.** (1998) Effects of aging on the insulin actions for the glucose metabolism and renal function in normotensives and essential hypertensives. *Am. J. Hypertens.* 11: 1056–1064.
15. **Mossberg H. O.** (1989) 40-year follow-up of overweight children. *Lancet* 2 (8661): 491–493.
16. **Moussa M. A., Skaik M. B., Selwanes S. B., Yaghy O. Y., Bin-Othman S. A.** (1994) Contribution of body fat and fat pattern to blood pressure level in school children. *Eur. J. Clin. Nutr.* 48: 587–590.
17. **Moussa M. A., Shaltout A. A., Nkansa-Dwamena D., Mourad M., Al-Sheikh N., Agha N., Galal D. O.** (1998) Association of fasting insulin with serum lipids and blood pressure in Kuwaiti children. *Metabolism* 47: 420–424.
18. **Möller R., Tafeit E., Smolle K. H., Kullnig P.** (1994) "Lipometer": determining the thickness of a subcutaneous fatty layer. *Biosens. Bioelectron* 9: xiii–xvi.

19. **Möller R., Tafait E., Smolle K. H., Pieber T. R., Ipsiroglu O., Duesse M., Huemer C., Sudi K., Reibnegger G.** (2000) Estimating percentage total body fat and determining subcutaneous adipose tissue distribution with the non-invasive optical device Lipometer. *Am. J. Hum. Biol.* 12: 221–230.
20. **Möller R., Tafait E., Pieber T., Sudi K., Vrecko K., Horejsi R., Reibnegger G.** (2000) The measurement of Subcutaneous Adipose Tissue Topography (SAT-Top) by means of the newly developed optical device Lipometer and the evaluation of standard factor coefficients for healthy subjects. *Am. J. Hum. Biol.* 12: 231–240.
21. **Schaefer F., Georgi M., Zieger A., Scharer K.** (1994) Usefulness of bioelectric impedance and skinfold measurements in predicting fat-free mass derived from total body potassium in children. *Pediatr. Res.* 35: 617–624.
22. **Stallones L., Mueller W. H., Christensen B. L.** (1982) Blood pressure, fatness, and fat patterning among USA adolescents from two ethnic groups. *Hypertension* 4: 483–486.
23. **Weinsier R. L., Norris D. J., Birch R., Bernstein R. S., Pi-Sunyer F. X., Yang M. U., Wang J., Pierson R. N. Jr., Van Itallie T. B.** (1986) Serum insulin and blood pressure in an obese population. *Int. J. Obes.* 10: 11–17.
24. **Wu C. H., Yao W. J., Lu F. H., Wu J. S., Chang C. J.** (1998) Relationship between glycosylated hemoglobin, blood pressure, serum lipid profiles and body fat distribution in healthy chinese. *Atherosclerosis* 137: 157–165.

Correspondence to:

Karl Michael Sudi

Institute of Sport Sciences, Karl-Franzens University

Mozartgasse 14, 8010 Graz

Austria

INSTRUCTION TO AUTHORS

Acta kinesiologiae Universitatis Tartuensis is a multidisciplinary (publishing papers on diverse subjects from sports and exercise sciences) annual publication.

Manuscripts are accepted for consideration with the understanding that their contents have not been published elsewhere. Manuscripts are read through by the editor and by two reviewers in blind review process, which takes 8–10 weeks.

Manuscripts are to be submitted in English. Submit three clear copies of the manuscript to the editor. Maximum volume of text is 10 pages, maximum total volume is 15 pages. Type the manuscript double-spaced on one side of A4 paper with margin of 3 cm on the left side.

The manuscript should be arranged as following

1. Title page

Title of the article in capital letters, names of authors, authors institution, name and address of the principal author, up to 5 key words. For blind review, a second title page is needed that contains only the title.

2. Abstract (up to 200 words, separate sheets).

3. Text

The text should contain the following sections: Introduction, Materials and Methods, Results, Discussion, References, Acknowledgements if any. Tables and Figures should be presented on separate sheets. Figures should be professional in appearance and have clean, crisp lines. Identify each Figure by marking lightly on the

back, indicating, Figure number, top side and abbreviated title of manuscript. Legends for the Figures should be submitted on a separate sheet. Tables should be double-spaced on separate sheets and include a brief title. The SI units should be used in presenting results.

Each citation in the text must be noted by number in parenthesis and must appear in the reference list as well. Each entry in the reference list must be double-spaced, arranged alphabetically and numbered serially by author with only one reference per number. Non-english papers should be cited in the original language.

Entries in the reference list should be as follows:

1. Sarna S., Kaprio J. (1994) Life expectancy of former elite athletes. *Sports Med.* 17 (3): 149–151
2. Gurbide E. (1975) *Tracer methods in hormone research.* Springer Verlag, New York
3. Morgan W. P., Borg G. (1976) Perception of effort in the prescription of physical activity. In: *The humanistic and mental health aspects of sports, exercise, and recreation*, T. Craig (ed). Chicago: Am. Med. Assoc. 256–259
4. Paoletti R. (1994) Future directions in drug treatment of atherosclerosis. 8th Int. Dresden Symp. on Lipoproteins and Atherosclerosis. Abstracts. Dresden, June 10–12, 1994, 22.

Page proofs will be sent to the author for marking of printer's errors. Ten reprints will be sent, free of charge, to the principal author.

Subscription rate: 20 USD (+postage) send to the editor.
1991–1997 issues are available.



ISSN 1406–9822