[Downloaded free from http://www.ijddc.com on Monday, October 11, 2010, IP: 59.183.141.47]

ORIGINAL ARTICLE

Effect of electrical stimulation on blood glucose level and lipid profile of sedentary type 2 diabetic patients

Deepshikha Sharma, Shweta Shenoy, Jaspal Singh

Department of Sports Medicine and Physiotherapy, Guru Nanak Dev University, Amritsar, India

Background: Physical exercise has emerged as a major tool in the prevention and management of diabetes mellitus, but a large diabetic population is not physically fit for regular exercise programme due to associated complications of diabetes and old age. This unique study is an attempt to explore the use of electrical stimulation as a method of treatment of type 2 diabetic patients. **Aims:** This study was designed to analyze the effects of electrical stimulation on blood glucose level, lipid profile, and physiological parameters of sedentary type 2 diabetic patients. **Settings:** This study was experimental in nature with different subject design. **Materials and Methods:** The experimental group (N = 20) underwent electrical stimulation through surface electrodes placed over motor points of quadriceps femoris muscles for 40 min/day, 3 days/week continued for 2 weeks. Twenty diabetic subjects who received placebo treatment served as the control group. Physiological parameters were measured, and blood samples taken both before and after treatment on the first day and the last day of the intervention in both groups. **Statistical Analysis:** Related and unrelated t-tests were used for statistical analysis. **Results and Conclusion:** The percentage change in blood glucose level scores of the experimental group (12.71%) was much higher as compared to the control group (4.06%) after a single session of treatment. Following 2 weeks of electrical stimulation, the blood glucose level was significantly ($t = 5.026$, $P = 0.001$) reduced in experimental group, but there was an insignificant fall in the same for the control group. Our results suggest that electrical stimulation can be used to assist in the control of blood glucose level in type 2 diabetic patients.

KEY WORDS: Blood glucose level, carbohydrate oxidation, glucose uptake, lipid profile

DOI: 10.4103/0973-3930.70859

Correspondence to: **Dr. Deepshika Sharma**, 81-Krishna Square-1, Amritsar, India. E-mail: dshikha_vaanya@yahoo.com.

Manuscript received: 20.05.2009; Revision accepted: 09.08.2010

Introduction

Physical exercise has emerged as a major tool in the prevention and management of diabetes mellitus. The metabolic effects of a regular exercise programme are well documented and accepted as an important means of achieving blood glucose regulation in type 2 diabetes mellitus. Recent epidemiological studies have provided evidence that a low level of physical activity is associated with an increased incidence of type 2 diabetes.[1,2] Increased physical activity delays the onset of type 2 DM or even prevents the disease in about 50% of susceptible individuals (positive family history of T2DM, body mass index > 25, hypertension, or gestational diabetes),[3] whereas a lack of physical activity is associated with increased insulin resistance in muscles[4] and glucose intolerance despite increased endogenous insulin secretion.[5]

Physical training improves glucose tolerance in individuals with type 2 DM, in persons with type 1 DM it may diminish insulin requirements.[6] The beneficial effect of a regular exercise on glucose control appears to reflect the cumulative effect of the transient improvement in glucose tolerance following each individual bout of exercise.[3] A single bout of exercise results in the translocation of GLUT4 to the plasma membrane in skeletal muscle of an individual and through this mechanism exercise enhances skeletal muscle glucose uptake in individuals with type 2 diabetes.[7]

Tamura *et al*. found that 2 weeks of diet and exercise therapy decreased intramyocellular lipid content and increased muscle insulin-mediated glucose uptake.[8] Physical activity exerts pronounced effects on substrate utilization and insulin sensitivity, which in turn potentially lower blood glucose and lipid profile. Exercise training also improves many other physiological and metabolic abnormalities that are associated with T2DM such as lowering body fat, reducing blood pressure, and

[Downloaded free from http://www.ijddc.com on Monday, October 11, 2010, IP: 59.183.141.47]

normalizing dyslipoproteinemia,[9] but a large diabetic population is not physically fit for regular exercise programme due to associated complications of diabetes and old age.

Investigators have cast doubts on the efficacy and feasibility of physical training as a long-term treatment for a majority of type 2 diabetic patients, especially over the age of 55 because of other interfering diseases which may complicate or severely hinder all physical training apart from very low-intensity exercise programmes.[10,11]

Regular physical exercise helps to control blood glucose level in diabetic patients, but there are individuals who are restricted from voluntary physical activity or are in a bedridden state due to chronic illness, spinal cord injury, or complications of diabetes. More recently, Mikines *et al*. and Stuart *et al*. have concluded that physical inactivity caused due to bed rest for as little as 7 days is associated with a substantial reduction in insulin sensitivity primarily in an inactive skeletal muscle with little change in insulin action on the liver.[4,12] In addition, prolonged physical inactivity has been shown to decrease the oxygen-carrying capacity of skeletal muscle[13] and also results in decrease muscle GLUT-4 content associated with insulin resistance.

Clinically, electrical stimulation is used as a modality of assisting muscle contraction for those who have difficulties in performing voluntary exercise. A number of studies have focused on the effect of electrical stimulation in disabled or physical inactive individuals. Chilibeck *et al*. concluded that electrical stimulation-assisted training is effective for increasing glucose transporters and improving insulin sensitivity in patients with spinal cord injury.[14] According to Mohr *et al*., individuals with spinal cord injury have an increased prevalence of insulin resistance and type 2 diabetes mellitus. In able-bodied individuals, training with large muscle group increases insulin sensitivity and may prevent type 2 diabetes. However, individuals with spinal cord injury cannot voluntary recruit major muscle groups, but by functional electrical stimulation they can perform ergometer bicycle training. Mohr *et al*. concluded that electrically induced bicycle training performed three times per week increases insulin sensitivity and GLUT 4 content in skeletal muscle in subjects with spinal cord injury. According to Hamada *et al*, low frequency electrical stimulation may become a useful therapeutic approach to activate energy and glucose metabolism in humans. The study provided evidence for the enhancement of whole body glucose uptake during and after human skeletal muscle low frequency electrical stimulation.[15,16]

These studies are limited to subjects not clinically diagnosed with diabetes mellitus; similar studies in the diabetic population have not yet been conducted. This study is an attempt to explore the use of electrical stimulation as a new method for type 2 diabetic patients who are unable to participate in a regular exercise programme. To serve the purpose, relevant biochemical parameters, i.e. blood glucose, blood cholesterol, triglycerides, high density lipoprotein, and physiological parameters, i.e. heart rate, respiratory rate, and blood pressure, were measured for changes after intervention in the study.

To our knowledge, this is the first report demonstrating the effect of electrical stimulation on the blood glucose level and lipid profile of patients with type 2 diabetes mellitus.

Materials and Methods

Design

The study was experimental in nature with a different subject design.

Participants

Twenty patients with type 2 diabetes mellitus above the age of 55 years having a sedentary lifestyle and BMI > 27 were included in the study.

Experimental Protocol

The subjects attended a pre-participation screening phase 3 days prior to the start of experiment to rule out the associated complications of diabetes, recent injuries, cardiopulmonary problems, and disorders other than diabetes. Following this screening phase, a medical examination and clearance from a physician were taken for participation in the study. They were informed of the nature, purpose, and parameters of the study before taking voluntary consent to participate in the study, and they continued their medication, diet, and other activities of daily living as before. With the suggestion of a diabetologist, a carbohydrate snack including two pieces of bread and a cup of tea was added 3 to 4 h before the start of experiment to reduce the risk of hypoglycemia after an overnight fasting in diabetic patients. This study was approved by ethical committee in Guru Nanak Dev University, Amritsar.

[Downloaded free from http://www.ijddc.com on Monday, October 11, 2010, IP: 59.183.141.47]

The subjects were randomly divided into two equal groups; each group having 10 diabetic patients; one was the experimental group and other was the control group. The experiment group was made to undergo 40 min electrical stimulation of quadriceps with 20 min each; with intensity up to tolerable limits of the subject since this was the standard procedure.[17-20]

The faradic current was of sufficient intensity and produced a good amount of contraction. The current was surged so that contraction gradually increased and decreased in strength, in a manner similar to a voluntary contraction. On the other hand, the control group was made to undergo placebo treatment for the same time period, i.e. 40 min; this also involved electrical stimulation of quadriceps muscle but intensity only up to sensory level. A mild pricking sensation was experienced by the patient, but the faradic current was of insufficient intensity to produce muscle contraction.

Before the experiment, the subject was made to rest for 10 min in supine lying. The subject was made to wear the polar short-range telemetry strap on the chest and monitoring watch on his/her wrist. The physiological parameters such as heart rate, respiratory rate, and blood pressure were measured at the end of this period with the patient in supine lying, blood sample was taken from antecubital vein at the cubital fossa by a lab technician using a 21 gauge needle and 10 mL syringe for biochemical analysis. All these values were noted as the baseline values of first session, i.e., R1—for the experimental group and R1′—for the control group.

The stimulator used for experiment was biomed muscle stimulator, a commonly used stimulator in the rehabilitation department. A frequency of 50 Hz was used, and it has been the most common protocol for human studies.[21] Surface electrical stimulation at a frequency of 50 Hz or more on human quadriceps produces forces up to 60% of maximum voluntary isometric contraction.[22] Surface rubber electrodes were used to deliver the current, one of which was placed over the femoral triangle and the other positioned over the distal portion of quadriceps femoris, unilaterally. The face of the electrodes which was to be applied to the skin was coated with aquasonic gel to reduce the impedance. The electrodes were then strapped to the thigh by means of micropore tapes to maintain good contact.

After 20 min of electrical stimulation in the experimental group or placebo treatment in the control group, the electrodes from one quadriceps femoris were fixed to other side quadriceps femoris for the next 20 min. At the end of a total of 40 min of electrical stimulation, the subjects underwent measurement of all the physiological parameters and again blood samples were taken for biochemical analysis. The values obtained were recorded as changes at the end of first session; R2—for the experimental group and R2′—for the control group. With this, the first session of experiment was completed. Following the similar procedure, patients were made to undergo total six sessions of electrical stimulation/placebo treatment within 2 weeks, 3 days/week. On the last day of treatment, i.e., sixth session, subject again underwent measurement of all physiological parameters and blood samples were taken for biochemical analysis before and after the treatment. These values were recorded as R3, R4 for the experimental group and R3′, R4′ for the control group.

Statistical Analysis

Related *t*-test was used to determine the changes in pre-post readings of various parameters after application of electrical stimulation. The inter-group comparison, i.e., in between experimental group and control group, was done with unrelated *t*-test.

Results

The results obtained from this study showed a significant decrease in blood glucose level following the application of electrical stimulation, but the changes in other parameters of the study, i.e., lipid profile (blood cholesterol, triglycerides, and high density lipoproteins) and physiological parameters (heart rate, respiratory rate, and arterial blood pressure), showed a statistically insignificant change following the application of electrical stimulation [Figure 1].

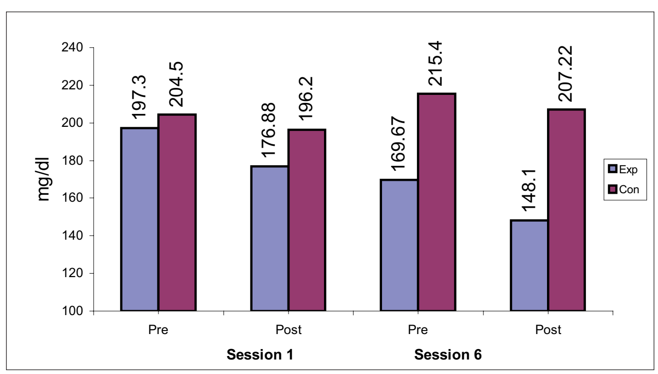

Figure 1: Effect of electrical stimulation on blood glucose level (mg/dL) of patients with type 2 diabetes mellitus.

[Downloaded free from http://www.ijddc.com on Monday, October 11, 2010, IP: 59.183.141.47]

After a single session of electrical stimulation, there was significant decrease in post-treatment scores of blood glucose level than the pre-treatment scores both in experimental group (t = 8.27, P = 0.000) and control group (t = 12.13, P = 0.000), but the percentage decrease in pre–post scores of experimental group (10.35%) was much higher as compared to control group (4.06%). The blood glucose level score in the sixth session of electrical stimulation showed a significant difference (decrease) in the experimental group (t = 5.026, P = 0.001), but an insignificant difference (t = 0.534, P = 0.607) in control group.

The intergroup comparison showed that there is no significant difference between the pre-treatment scores of experimental group and control group in the first session, but there is significant difference (t = 2.30, P < 0.05) between pretreatment scores of experimental group and control group in the sixth session.

Table 1 shows that post-treatment blood glucose level (176.88 43.74) mg/dL was significantly (t = 8.272; P = 0.000) different from pre-treatment blood glucose level (197.30 ± 47.027) mg/dL after a single session of electrical stimulation, in experimental group. There was also a significant (t = 12.136; P = 0.000) difference in blood glucose from (204.50 ± 53.86) mg/dL to (196.20 ± 52.47) mg/dL after placebo treatment in control group. However, the percentage decrease of blood glucose level in the experimental group (10.35%) was much higher as compared to control group (4.06%).

Table 1 also shows that blood glucose level after six sessions electrical stimulation (148.11 ± 18.53) was significantly decreased (t = 5.026, P = 0.001) in the experimental group as compared to the control group (207.22 ± 58.16) with reference to the pre-treatment scores.

The intergroup comparison showed that there is no significant difference between the pre-treatment scores of experimental group (197.30 ± 47.027) mg/dL and the pre-treatment scores of control group (204.50 ± 53.86) mg/dL in the first session, but there is a significant (t = 2.30, P < 0.05) difference between the pre-treatment scores of experimental group (169.67 ± 23.94) mg/dL and pre-treatment scores of control group (215.40 ± 58.13) mg/dL in the sixth session.

The first session post-treatment scores of experimental group (176.88 ± 43.74) mg/dL and control group (196.20 ± 52.47) mg/dL showed insignificant difference, but the post-treatment scores of sixth session showed significant (39.92%, P < 0.05) difference between experimental group (148.10 ± 18.53) mg/dL and control group (207.22 ± 58.16) mg/dL.

The line graph clearly represents the decreased blood glucose level in the experimental group which followed six session of electrical stimulation as compared to the control group which received placebo treatment for the same time period.

There was no significant difference between the pre- and post-treatment scores of blood cholesterol level in the experimental group as well as the control group. Though the six sessions of electrical stimulation did not produce a statistical significant change in blood cholesterol level, the mean value fall from 167.80 mg/dL (±40.58) to 162.10 mg/dL (±26.05) was noteworthy. Changes with prolonged use of electrical stimulation may yield statistically significant results. No such change (fall) was observed in control group.

There was no significant difference between the pre- and post-treatment scores of triglycerides level of experimental group as well as control group. Though the six sessions of electrical stimulation did not produce a statistical significant change in triglycerides, a mean

Table 1: Effect of electrical stimulation on blood glucose level (mg/dL) of patients with type 2 diabetes mellitus

Group	First session		Sixth session	
Experimental group	Pre-treatment (R1)	Post-treatment (R2)	Pre-treatment (R3)	Post-treatment (R4)
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
	197.30 ± 47.027	176.88 ± 43.74	169.67 ± 23.94	148.10 ± 18.53
Control group	204.50 ± 26.86	196.20 ± 52.47	215.40 ± 58.13	207.22 ± 58.16
	R1 R1′	R2 R2′	R3 R3′	R4 R4′
t values	0.318^{NS}	0.898 ^{NS}	2.30*	3.063*
	R1R2	R1′R2′	R1R4	R1′R4′
	8.272***	12.136***	5.026**	ns0.534

*Significant at P < 0.05, **significant at P < 0.01, and ***significant at P < 0.001. NS, non significant.

[Downloaded free from http://www.ijddc.com on Monday, October 11, 2010, IP: 59.183.141.47]

value fall from 126.00 mg/dL (±57.31) to 115.70 mg/dL (±49.18) was observed. No such change (fall) was observed in control group.

There was no significant difference between the pre- and post-treatment scores of high density lipoproteins of experimental group as well as control group. The mean value fall was also negligible.

Discussion

Diabetes is a major public health problem and is emerging as a pandemic. Several efficacious and economically acceptable treatment strategies are currently available to reduce the burden of diabetes complications. Regular physical exercise is widely perceived to be beneficial for type 2 diabetic patients as it influences several aspects including blood glucose concentration, insulin action, and cardiovascular risk factors. However, a large diabetic population is restricted from voluntary physical activity due to associated complications of the syndrome.

With this study, we tried to develop a new treatment strategy for these sedentary type 2 diabetic patients. The study analyzed the metabolic effects of electrical stimulation and the results of our study favored the hypothesis that electrical stimulation of group muscles has a significant effect on the blood glucose level of sedentary type 2 diabetic patients. The comparison between the blood glucose pre-treatment scores of experimental group and the pre-treatment scores of control group on the last session of study showed a significant difference between the two groups. Although with a single session treatment results were statistically significant in both the experimental group and the control group, after the 2-week treatment period results remained significant only in the experimental group. The control group showed an insignificant fall in blood glucose level.

Exercise can increase the rate of glucose uptake via two distinct mechanisms, i.e., an insulin independent one (contraction-stimulated glucose uptake) and an insulin-dependent one (post-exercise increase in insulin sensitivity). Wallberg-Henrikson *et al.* have shown in isolated rat skeletal muscle that the activity of insulin-independent glucose uptake to maximally enhanced immediately after exercise and then gradually wears off, but in 34% of the initial activity *in situ* present at 180 min. In contrast, increased insulin sensitivity is undetectable in the early phase of the post-exercise period and becomes prominent at 180 min after exercise.[9] In line with these findings, Price *et al*. have shown in human muscles that post-exercise glycogen repletion occurs in an insulin-independent manner for ~ 1 h after exercise and thereafter insulin-dependent glycogen repletion becomes significant.[23]

In our study insulin-independent mechanism, i.e. contraction stimulated glucose uptake is proposed to be responsible for the decrease in blood glucose level in a single session. Our application of electrical stimulation directly activates the glucose uptake in quadriceps muscle by inducing *trans* location of GLUT -4 glucose transporters to the cell surface *via* this insulin-independent mechanism. However, the decrease in blood glucose level after 2 weeks of electrical stimulation found in this study is likely to be due to the accumulation of insulin-dependent effect of increase in insulin sensitivity. This insulin-dependent effect may come into play during the post-stimulation period, i.e. in between the various sessions of electrical stimulation and this gives more positive results in the final session of the study. The stimulation of AMP-activated protein kinase (AMPK) in response to contraction induced by electrical stimulation may be associated with increased glucose uptake in quadriceps muscle. AMPK is stimulated by various glycogen depleting stimuli, such as contraction, hypoxia, and hyperosmolarity and have positive effects on glucose uptake and fatty acid oxidation in skeletal muscles.

Hultman and Spriet studied the muscle metabolism in response to the electrical stimulation and demonstrated that the use of electrical stimulation for 45 min at 20 H_2 frequency causes a 44% decrease in glycogen concentration in quadriceps muscle,[24] and Kim *et al*. have also found a significant decrease in glycogen content using ES for 60 min at 50 Hz.[25] Henning *et al*. revealed that with external ES, motor units for type II fibers are more active due to larger axonal diameter, thus ES results in preferential activation of type II fibers that have a larger capacity for glycogen utilization in humans.[26,27] In addition, rates of glycogenolysis during ES are twofold higher in type II fibers than type I fibers.[28] These findings and earlier observations seem to suggest that there occurs a large activation of glycolytic type II fibers by ES resulting in a significant decrease in blood glucose level and thereby possibly improving insulin sensitivity in the long run.

Lehmann and Spinas evaluated the effects of a 3-month physical training programme on the lipid profile of type

[Downloaded free from http://www.ijddc.com on Monday, October 11, 2010, IP: 59.183.141.47]

2 diabetic patients and found a significant improvement of plasma lipids with a 20% decrease in triglycerides, unchanged total cholesterol, and 23% increase in high density lipoproteins, respectively.[3] Baldwin *et al*. and Carlson *et al*. reported that during prolonged exercise, energy for muscle contraction is derived from both plasma free fatty acids and intramuscular triglycerides.[29,30] Although the effects of a regular exercise programme on the lipid profile of type 2 diabetic patients is well documented, the influence of electrical stimulation on the lipid profile of diabetic patients is not well documented in the literature.

In this study, the effect of ES on all the parameters of lipid profile was insignificant and this may be attributed to the shorter duration and lesser intensity of the contractile activity of the muscles. With the electrical stimulation of quadriceps muscles, the intensity of muscle contraction was not critical enough to produce significant changes in the lipid profile of patients with type 2 diabetes mellitus.

In our study, the changes in heart rate, respiratory rate, and arterial blood pressure were not statistically significant. These findings are in agreement with Davis *et al*.[31]

All subjects who participated in this study were sedentary type 2 diabetic patients without associated complications of syndrome; therefore, it is not clear whether similar metabolic effects would be shown in the bedridden diabetic patients with chronic illness. In addition, the muscle mass involved in contraction is expected to affect the magnitude of decrease in blood glucose level. A greater decrease in blood glucose level may be achieved if more muscles are employed by using more stimulation electrodes.

From this study, it was concluded that electrical stimulation can be used as a helpful modality to control the blood glucose level in type 2 diabetic patients, but the intensity of muscle contraction with the application of electrical stimulation was not critical enough to produce significant changes in lipid profile and other physiological parameters.

Acknowledgments

The authors express sincere thanks to the Amritsar Charitable Hospital for allowing to conduct the study in the hospital. They also thank Dr. Navneet Maini who had given the clearance to the patients for the participation in the study.

References

1. Eriksson KP, Lindgrade F. Prevention of type 2 2 (Non-insulin-dependent) diabetes mellitus by diet and physical exercise. The 6 year malmo feasibility study. Dialectologia 1991;34:891-8.
2. Helmrich SP, Ragland DR, Leung RW, Paffenbarger RS. Jr. Physical activity and reduced occurrence on non-insulin dependent diabetes mellitus. N Engl J Med 1991;325:147-52.
3. Lehmann R, Spinas GA. Role of physical activity in the therapy and presentation of Type 2 II diabetes mellitus. Ther Umsch 1996;53:925-33.
4. Stuart CA, Shangraw RE, Prince MJ, Peters EJ, Wolfe RR. Bed rest induced insulin resistance occurs primarily in muscles. Metabolism 1988;37:802-6.
5. Lipman RL, Raskin P, Love T. Glucose intolerance during decreased physical activity in man. Diabetes 1972;21:101-7.
6. Franz MJ. Exercise and the management of diabetes mellitus. J Am Diet Assoc 1987;87:872-80.
7. Kennedy JW, Hirshman MF, Gervino EV, Ocel JV, Forse RA, Hoenig SI, *et al*. Acute exercise induces GLUT 4 translocation in skeletal muscle of normal human subjects and subjects wit type 22 diabetes. Diabetes 1999;48:1192-7.
8. Tamura Y, Tanaka Y, Sato F, Choi JB, Watada H, Niwa M, *et al*. Effect of diet and exercise on muscle and liver intracellular lipid contents and insulin sensitivity in type 22 diabetic patients. J Clin Endocrinol Metab 2005;90:3191-6.
9. Wallberg - Henriksson H, Rincon J, Zierath JR. Exercise in the management of non-insulin-dependent diabetes mellitus. Sports Medicine 1998;25:25-35.
10. Zierath JR, Wallberg - Henrikson H. Exercise training in obese diabetic patients. Special considerations. Sports Med 1992;14:171-89.
11. Skarfors ET, Wagener TA, Lithell H, Selinus I. Physical training as treatment for type 2 2 (non-muslin-dependent) diabetes in elderly men. A feasibility study over 2 year. Dialectologia 1987;30:930-3.
12. Mikines KJ, Richter EA, Dela F, Galbo H. Seven days of bed rest decrease insulin action on glucose uptake in leg and whole body. J Appl Physiol 1991;70:1245-54.
13. Saltin B, Blmqvist G, Mitchell JH, Johnson RL Jr, Wildenthal K, Chapman CB. Responic to exercise after bed rest and after training. Circulation 1968;38:1-78.
14. Chilibeck PD, Bell G, Jeon J, Weiss CB, Murdoch G, Maclean I, *et al*. Functional electrical stimulation exercise increases GLUT-1 and GLUT-4 in paralyzed skeletal muscle. Metabolism 1999;48:1409-13.
15. Hamada T, Sasaki H, Hayashi T, Moritani T, Nakao K. Enhancement of whole body glucose uptake during and after human skeletal muscle low frequency electrical stimulation. J Appl Physiol 2003;94:2107-12.
16. Hamada T, Hayashi T, Kimura T, Nakao T, Moitani. Electrical stimulation of human lower extremities enhance energy consumption, carbohydrate oxidation, and whole body glucose uptake. J Appl Physiol 2004;96:911-6.
17. Currier DP, Lehman J, Light foot P. Electrical stimulation in exercise of quadriceps femois muscle. Physical Theraphy 1979;59:1508-12.
18. Eriksson E, Haggmark T, Kiessling KH. Effect of electrical stimulation on the size and strength of skeletal muscle. Int J Sports Med 1981;2:18-22.
19. Eriksson E, Haggmark T. Comparison of isometric muscle training and electrical stimulation supplementing isometric muscle training in recovery after major knee ligament surgery; A preliminary report. Am J Sports Med 1979;7:169-71.
20. Godfrey CM, Jaywardena H, Welsh P. Comparison of electro stimulation and isometric exercise in strengthening the quqdriceps

[Downloaded free from http://www.ijddc.com on Monday, October 11, 2010, IP: 59.183.141.47]

muscle. Physiother Can 1979;31:265.
21. Trankolzy A. Functional electrical stimulation of extremities: Its basis, technology and role in rehabilitation. Automedica 1978;2:59.
22. Edwards R, Young A, Hosking GP, Jones DA. Human skeletal muscle function: Description of tests and normal values. Clin Sci Mol Med 1977;52:283-90.
23. Price TB, Rothman DL, Taylor R, Avison MJ, Shulman GI, Shulman RG. Human muscle glycogen resynthesis after exercise: Insulin – dependent and independent phases. J App Physiol 1994;76:104-11.
24. Hultman E, Spriet LL. Skeltal muscle metabolism contraction force and glycogen utilization during prolonged electrical stimulation in humans. J Physiology 1986;34:493-501.
25. Kim CK, Strange S, Bangsbo J, Saltin B. Skeletal muscle perfusion in electrically induced dynamic exercise in man. Acta Phys Scand 1995;153:279-87.
26. Henning R, Lomo T. Firing patterns of motor units in normal rats. Nature 1985;314:164-6.
27. Sinacore DR, Delitto A, King DS, Rose SJ. Type 2 II fiber activation with electrical stimulation: A preliminary report. Phys Ther 1990;70:416-22.
28. Greemhaff PL, Soderlund K, Ren JM, Hultman E. Energy metabolism in single human muscle fibers during intermittent contraction with occluded circulation. J Physiol 1993;460:443-53.
29. Baldwin KM, Reitman JS, Terjung RL, Winder WW, Holloszy JO. Substrate depletion in different type 2s of muscle and in liver during prolonged running. Am J Physiol 1973;225:1045-50.
30. Carlson LA, Ekelund LG, Froberg SO. Concentration of triplycerides, phospholipids and glycogen in skeltal muscle and of fue fatty acids β-hydroxy butyric acid in blood in man in response to exercise. Eur J Clin Invest 1971;1:248-54.
31. Davis GM, Servedio FJ, Glaser RM, Gupta SC, Suryaprasad AG. Cardiovascular responses to arm cranking and FNS induced leg exercise in paraplegics. J Appi Physiol 1990;69:671-7.

Source of Support: Nil, **Conflict of Interest:** None declared

New features on the journal's website

Optimized content for mobile and hand-held devices
HTML pages have been optimized of mobile and other hand-held devices (such as iPad, Kindle, iPod) for faster browsing speed.
Click on **[Mobile Full text]** from Table of Contents page.
This is simple HTML version for faster download on mobiles (if viewed on desktop, it will be automatically redirected to full HTML version)

E-Pub for hand-held devices
EPUB is an open e-book standard recommended by The International Digital Publishing Forum which is designed for reflowable content i.e. the text display can be optimized for a particular display device.
Click on **[EPub]** from Table of Contents page.
There are various e-Pub readers such as for Windows: Digital Editions, OS X: Calibre/Bookworm, iPhone/iPod Touch/iPad: Stanza, and Linux: Calibre/Bookworm.

E-Book for desktop
One can also see the entire issue as printed here in a 'flip book' version on desktops.
Links are available from Current Issue as well as Archives pages.
Click on View as eBook