Muscle damage occurring in wheelchair sports people

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Seven college-age healthy men exercised on a wheelchair treadmill to evaluate muscle damage that may occur from wheelchair propulsion. An experimental model in which a participant performs up-hill running in a wheelchair was prepared. Plasma creatinekinase (CK), myoglobin (Mb) and lactatedehydrogenase (LDH) were measured as parameters. Blood samples were taken pre-, immediately after-, 24 h after- and 72 h after the exercise. All of these parameters significantly increased after the exercise, but their time-courses were apparently varied. It is concluded that wheelchair propulsion causes muscle damage in certain situations such as up-hill running.

Keywords: muscle damage; wheelchair propulsion; creatinekinase (CK); myoglobin (Mb); lactatedehydrogenase (LDH)

Introduction

Temporal muscle damage occurred in those participating in sports activities and physical exercise, and has been discussed by many researchers. Triathalon and marathon races appeared to be the most frequently discussed sports activities.[1-3] In laboratory conditions, severe exercise involving the elbow flexors has been commonly studied.[4-7] Delayed onset muscle soreness (DOMS)[8,9] and maximum voluntary contraction[4,6] were widely evaluated as non-invasive parameters. For invasive parameters, not many authors have used muscle biopsies and histology.[10] Many researchers have carried out blood sampling, measuring several parameters:[1,3,4,6,9,11] enzyme activities such as creatinekinase (CK) or lactatedehydrogenase (LDH), myoglobin (Mb) content appeared to be the most reliable parameters.

The wheelchair is the inevitable vehicle for seriously physically handicapped individuals who have lost their normal mobility. It is well known that wheelchair propulsion requires less energy consumption than does the use of crutches and an orthosis for gait, but rehabilitation professionals must always be concerned about the possibility of muscle fatigue from wheelchair propulsion by a handicapped person.[12]

The purpose of this study is (1) to establish an experimental wheelchair propulsion model in which muscle fatigue can be clearly observed, and (2) to evaluate the muscle fatigue occurring in the model by haematological studies.

Subjects

Seven healthy male college students were recruited for this study. All participated in some sports activities occasionally, however severe physical work was prohibited during the time of this study. Written informed consent was obtained at each experimental session.

Mean ($\pm$SD) age of participants was 23.9 ± 2.0 years (21 to 26 years). Anthropometric measurements of each subject were done to provide a physical description of the subjects. The average ($\pm$SD) of each measurements were: height 171.6 ± 4.6 cm, body weight 58.3 ± 2.3 kg, girth of chest 83.9 ± 2.8 cm, girth of right upper arm 25.0 ± 1.6 cm, arm span 167.9 ± 2.3 cm, and grasping power on right hand 40.7 ± 3.2 kg.

Method

Exercise protocol
A treadmill specially designed for wheelchair users was used in this study (Running Machine, product of Seno, Japan). Speed and inclination of the treadmill are adjustable in this equipment. A 4-wheel sportstype wheelchair was used for all of the participants to minimize the structural effect of wheelchair.

The model of the exercise is shown in Figure 1. The speed of the treadmill was fixed in 4.0 km/h. The inclination was increased by 0.5% every 5 min from 2.0% to 4.5%. After wheelchair propulsion for 30 min, the inclination was fixed at 3.0%. This exercise protocol was the modified one used by Okuma[13] who evaluated the physical fitness of paraplegic athletes. In this program, a participant performed wheelchair propulsion for 3 km in 45 min.

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Measurements
The heart rate was monitored during the exercise using a telegraphic electrocardiogram, to check for arrythmia and was recorded every 5 min at 1 min after newly increasing the inclination.

Blood samples were obtained before exercise, immediately after, 24 h after and 72 h after the exercise. The indwelling needles were kept patent by irrigation with heparinized saline solution and were used for sample collection in order to minimize pain from repeated needle insertions. Blood for biochemical analysis was centrifuged immediately after clotting, and the serum was frozen until assays were performed (except for LDH assay).

The blood cell count, electrolytes, total creatinekinase (CK) activity and isoenzyme composition, myoglobin (Mb) concentration, total lactatedehydro-genase (LDH) activity and isoenzyme composition were measured with the commercially available laboratory kit. The normal value of these parameters in this kit were: total CK activity $57 \sim 197$ IU/L, Mb concentration <60 ng/ml, total LDH activity $230 \sim 460$ IU/L.

Statistics
The results are presented as mean $\pm$ SD. The Paired Student's t-test was used to assess significance levels between pre-exercise and immediately, 24 h, 72 h after-exercise. Differences were considered significant when $P < 0.05$.

Results

The change of heart rate at each inclination is shown in Figure 2. Arrythmias were not detected in any other participants.

No significant changes were observed in the electrolytes. The white blood cell and hematocrit were slightly increased immediately after the exercise (Table 1).

The value of CK-MM, which reflected the damage of striated muscle, was estimated from the total CK activity and isoenzyme composition. The mean of CK-MM was normal prior to the exercise, and remarkably increased after the exercise reaching a peak value at 24 h after the exercise (Figure 3).

The mean of myoglobin (Mb) concentration was normal prior to exercise, and increased gradually (Figure 4). The value at 24 h after and 72 h after exercise showed significance.
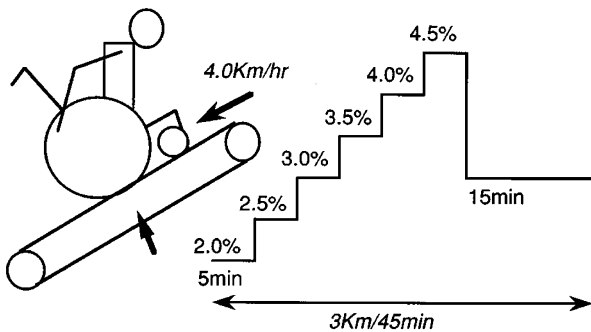

Figure 1 The model of exercise: speed of treadmill 4.0 km/h increase of inclination by 0.5% every 5 min from 0.2% to 4.5%
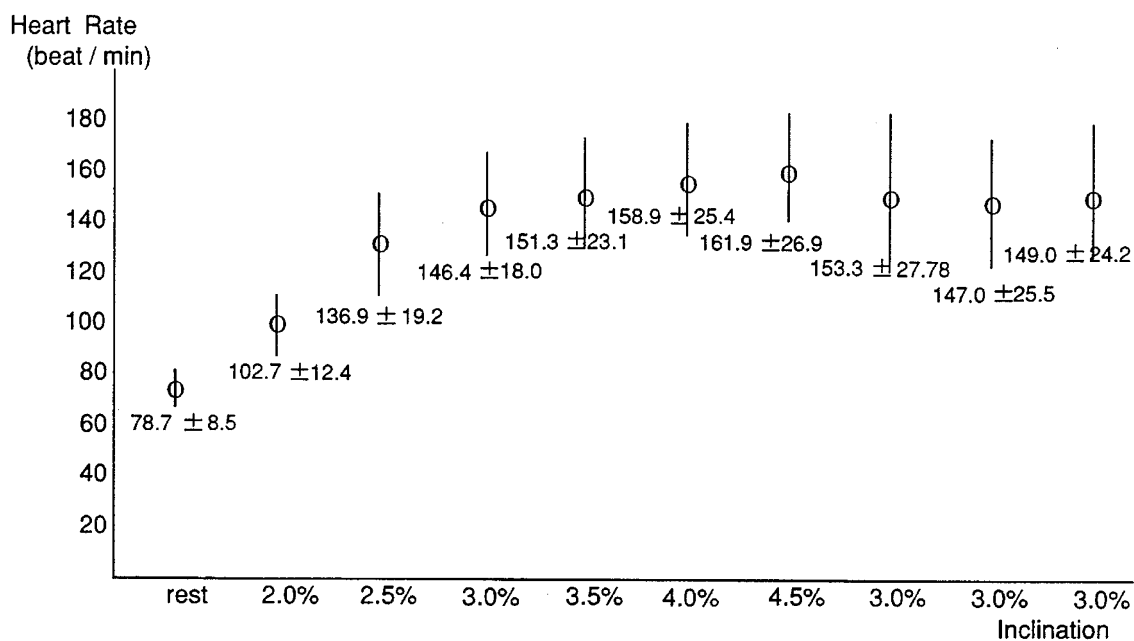

Figure 2 The change of heart rate

Table 1 Electrolytes and blood cells studies

	Pre-ex	*Immed-after*	*24 h-after*	*72 h-after*
Na	139.9 ± 2.1	139.4 ± 2.4	139.6 ± 1.0	139.6 ± 0.5
K	3.8 ± 0.1	4.0 ± 0.3	3.9 ± 0.2	4.0 ± 0.2
Cl	101.4 ± 2.6	100.3 ± 2.0	102.7 ± 1.4	103.6 ± 1.8
WBC	5185.7 ± 942.4	6328.6 ± 1481.8	5485.7 ± 1132.0	6071.4 ± 1924.2
Hb	15.0 ± 0.5	15.8 ± 0.5	14.7 ± 0.6	14.1 ± 0.6
Hct	45.6 ± 2.1	47.9 ± 2.3	43.3 ± 2.0	42.9 ± 2.4

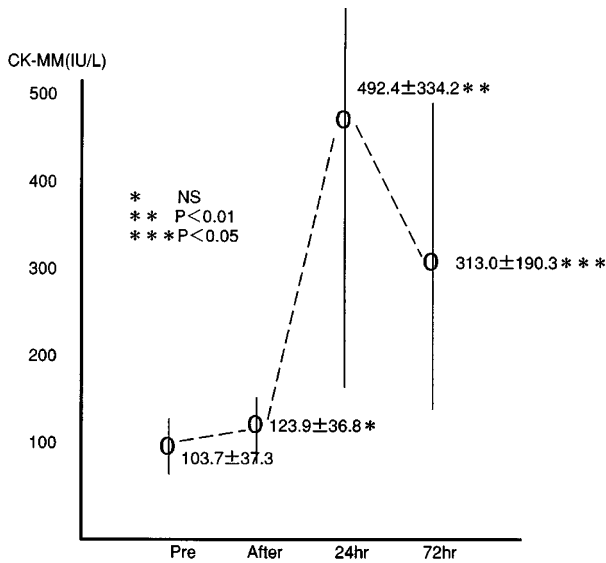Data are shown as mean $\pm$ S.D. $n = 7$

Figure 3 The change of CK-MM: estimated from the total CK activity and isoenzyme composition significantly increased 24 h-after and 72 h-after the peak value was observed 24 h-after
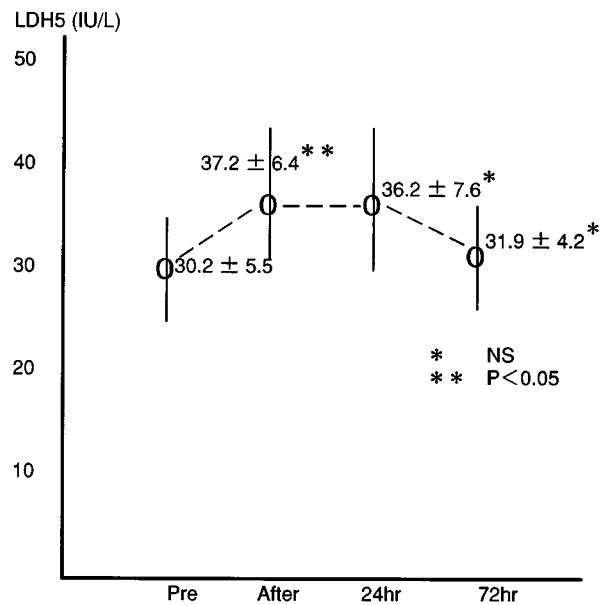

Figure 5 The change of LDH$_5$: estimated from the total LDH activity and isoenzyme composition significantly increased immediately-after only
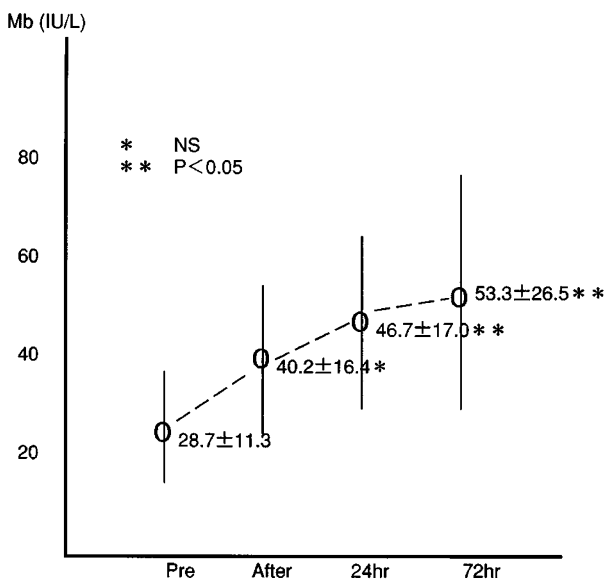

Figure 4 The change of Mb: significantly increased 24 h-after and 72-after the peak value was observed 72 h-after

The value of LDH$_5$ which reflected the damage of striated muscle was estimated from the total LDH activity and isoenzyme composition. The mean of LDH$_5$ was normal prior to exercise, and increased slightly but significantly after the exercise (Figure 5). It reached the peak value immediately after the exercise.

Discussion

Considering the mean age of subjects, the age-predicted maximum heart rate is nearly 195 beats/min and 'training-sensitive zone' (70% ∼90% of maximum heart rate) can be estimated as 136−175 beats/min.[14] Regarding cardiovascular fitness, the protocol used in this study may be recognized as being 'moderate exercise' (Figure 2).

The increased hematocrit immediately following the exercise indicated the possibility of hemoconcentration caused by dehydration (Table 1). The elevations of myoglobin (Mb) and enzymes (CK-MM, LDH$_5$) were significant (Figure 3−5). Although some of these

elevations may have been due to hemoconcentration or by decreased renal clearance, the existence of skeletal muscle damage was clearly suggested.

It is quite difficult to describe which muscles were mainly damaged in this exercise protocol. Muscles in the arms would be the first to be reflected, but the possibility of muscle damage in the trunk and legs remains because able-bodied subjects tend to stabilize their trunk using these muscles in the wheelchair. Isometric or concentric contraction of these muscles can be carried out easily even when in a wheelchair, and the increased inclination in this exercise protocol would require a more rigidly stabilized trunk than would be necesssary for propulsion on level ground.

The time-courses of parameters measured in this study were different from other studies. LDH_5 reached its peak value immediately after the exercise and CK-MM was reached 24 h later (Figure 3 and 5). But myoglobin increased gradually during the study (Figure 4). Many authors reported the time-course of these parameters stating: myoglobin reached the peak value immediately after the exercise and CK reached 24 h after.[1,8,9]

The molecule size of these parameters should be considered when we discuss their time-course. Because the increase of these parameters reflects/focal necrosis of muscle fibers,[15] the 'smaller' protein may appear in the blood earlier through a smaller lesion in the sarcolemma. The molecular weights of these parameters are: CK-MM 85,000 daltons,[16] LDH_5 135,000,[16] myoglobin 17,500 daltons.[17] It is reasonable to expect that myoglobin reaches the peak value first, CK-MM and LDH_5 follow.

The reason for the inconsistency between our results and this theory is somewhat unclear. The author would suggest one possibility. Most former studies have dealt with rather simplified exercise for healthy subjects, for example elbow bending exercise and down-hill running. However, in our study, no subject had experience of wheelchair propulsion. They were allowed to propel their wheelchairs without any regulation of propulsion form. It means that the variance of the muscles used in the protocol is quite large. Some subjects complained of neck muscle pain after the exercise, but others had pain in their shoulders or upperarms. The large variance may have been brought about by the time discrepancy and the extent discrepancy of the muscles damaged in the exercise. The time-course of enzymes described above appears to substantiate this.

This study suggests that wheelchair propulsion cause muscle damage in the able-bodied in certain conditions. The neurologically handicapped person such as someone with a severe spinal cord injury can be considered to damage residual functions easily. There is a report in which the high incidence of overuse injuries in wheelchair racing athletes was described.[18] Rehabilitation professionals should always be concerned about the possibility of muscle damage caused by wheelchair propulsion, not only in relation to sports activities, but also from other daily activities.

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