Changes in Muscle Oxygen Saturation Have Low Sensitivity in Diagnosing Chronic Anterior Compartment Syndrome of the Leg

Kajsa Rennerfelt, MD, PhD, Qiuxia Zhang, PhD, Jón Karlsson, MD, PhD, and Jorma Styf, MD, PhD

Investigation performed at the Department of Orthopaedics, University of Gothenburg, Sahlgrenska University Hospital, Gothenburg, Switzerland

Background: Near-infrared spectroscopy measures muscle oxygen saturation (StO_2) in the skeletal muscle and has been proposed as a noninvasive tool for diagnosing chronic anterior compartment syndrome (CACS). The purpose of this study was to investigate the diagnostic value of changes in StO_2 during and after exercise in patients with CACS.

Methods: The study comprised 159 consecutive patients with exercise-induced leg pain. Near-infrared spectroscopy was used to measure StO_2 continuously before, during, and after an exercise test. One minute post-exercise, intramuscular pressure was recorded in the same muscle. The cohort was divided into patients with CACS (n = 87) and patients without CACS (n = 72) according to the CACS diagnostic criteria. Reoxygenation at rest after exercise was calculated as the time period required for the level of muscular StO_2 to reach 50% (T_{50}), 90% (T_{90}), and 100% (T_{100}) of the baseline value.

Results: The lowest level of StO_2 during exercise was 1% (range, 1% to 36%) in the patients with CACS and 3% (range, 1% to 54%) in the patients without CACS. The sensitivity was 34% and the speci city was 43% when an StO_2 level of £8% at peak exercise was used to indicate CACS. The sensitivity and the speci city were only 1% when an StO_2 level of £50% at peak exercise was used to indicate CACS. The time period for reoxygenation was seven seconds (range, one to forty-three seconds) at T_{50}, twenty-eight seconds (range, seven to seventy-seven seconds) at T_{90}, and forty-two seconds (range, seven to 200 seconds) at T_{100} in the patients with CACS and ten seconds (range, one to forty-nine seconds) at T_{50}, thirty-two seconds (range, four to 138 seconds) at T_{90}, and forty-eight seconds (range, four to 180 seconds) at T_{100} in the patients without CACS. When thirty seconds or more at T_{90} was set as the cutoff value for a prolonged time for reoxygenation, indicating a diagnosis of CACS, the sensitivity was 38% and the speci city was 50%.

Conclusions: Changes in muscle oxygen saturation during and after an exercise test that elicits leg pain cannot be used to distinguish between patients with CACS and patients with other causes of exercise-induced leg pain.

Level of Evidence: Diagnostic Level II. See Instructions for Authors for a complete description of levels of evidence.

Peer Review: This article was reviewed by the Editor-in-Chief and one Deputy Editor, and it underwent blinded review by two or more outside experts. The Deputy Editor reviewed each revision of the article, and it underwent a nal review by the Editor-in-Chief prior to publication. Final corrections and clari cations occurred during one or more exchanges between the author(s) and copyeditors.

Patients with chronic anterior compartment syndrome (CACS) experience muscle swelling, impaired dorsi exion of the ankle joint, and anterior leg pain induced by exercise. Even though the pathophysiology of CACS is not fully understood, it is generally agreed that abnormally raised intramuscular pressure during exercise results in inadequate local tissue perfusion; ischemic pain; and, accordingly, impaired muscle function[1]. The abnormally elevated intramuscular pressure during

Disclosure: None of the authors received payments or services, either directly or indirectly (i.e., via his or her institution), from a third party in support of any aspect of this work. None of the authors, or their institution(s), have had any nancial relationship, in the thirty-six months prior to submission of this work, with any entity in the biomedical arena that could be perceived to in uence or have the potential to in uence what is written in this work. Also, no author has had any other relationships, or has engaged in any other activities, that could be perceived to in uence or have the potential to in uence what is written in this work. The complete Disclosures of Potential Con icts of Interest submitted by authors are always provided with the online version of the article.

Points of Observation	De nition
Baseline	Level (%) of StO_2 before exercise
During exercise	
Peak exercise	Lowest level (%) of StO_2 at maximum effort
End of exercise	Level (%) of StO_2 at end of exercise
At rest after exercise	
T_{50}	No. of seconds required for StO_2 level at end of exercise to reach 50% of its baseline
T_{90}	No. of seconds required for StO_2 level at end of exercise to reach 90% of its baseline
T_{100}	No. of seconds required for StO_2 level at end of exercise to reach 100% of its baseline
Maximum recovery	Maximum level (%) of StO_2 during recovery

exercise has been reported to impair muscle blood ow[1] and induce changes in muscle oxygen saturation (StO_2)[2].

Near-infrared spectroscopy has been proposed for the investigation of CACS[3]. Near-infrared light is emitted from a light source and transmitted into the tissue; 95% of the light is reabsorbed by the receiver, while 5% is scattered through the tissue. Hemoglobin changes its optical properties when binding to oxygen. The chromophore, the part of the hemoglobin molecule that binds to oxygen, absorbs and re ects light[4]. Oxygenated hemoglobin absorbs light at a wavelength of 830 nm, while deoxygenated hemoglobin absorbs light at 780 nm. Near-infrared spectroscopy provides continuous information about the absolute level of StO_2 locally in the exercising muscle[5]. It has been proposed that it be used as a noninvasive tool to measure StO_2 continuously during and after an exercise test that induces the patient's leg pain in establishing a diagnosis of CACS[2,3]. Previous studies indicate that patients with CACS have greater deoxygenation during exercise[2,3] and delayed reoxygenation after exercise compared with patients without CACS[3]. However, the magnitude of the decrease in StO_2 during exercise has also been reported to be an unreliable measurement for establishing the diagnosis of CACS[6]. Since contradictory nd ings are reported in the literature, the diagnostic value of StO_2 changes in CACS needs to be clari ed in a large group of patients with exercise-induced leg pain.

The purpose of the present study was to evaluate whether changes in StO_2 during and after an exercise test that elicits pain and muscle fatigue can be used to establish a diagnosis of CACS of the leg. The hypothesis was that StO_2 would decrease more during exercise and the time for reoxygenation after exercise would be longer in patients with CACS compared with patients without CACS.

Participants

The study comprised 159 consecutive patients, seventy-six males and eighty-three females with a median age of twenty-nine years (range, fourteen to sixty-seven years), who had exercise-induced leg pain and clinical signs of CACS. Most of the patients were involved in running activity several times a week or in high-level organized sports. The patients included in the study had been referred to the university clinic between May 2009 and January 2012 for an evaluation of suspected CACS. The study protocol was approved by the regional ethics committee (ID number 617-08).

Diagnostic Criteria

The diagnostic criteria for CACS were (1) exercise-induced leg pain over the anterior compartment with a reversal of symptoms at rest, (2) swelling and tenderness over the anterior compartment immediately after exercise, (3) impaired muscle function during activity, and (4) intramuscular pressure of ‡30 mm Hg one minute after exercise and ‡20 mm Hg ve minutes after exercise[7,8]. All four criteria were required for the diagnosis of CACS.

Inclusion Criteria

All patients admitted to our clinic during the time period de ned above were included in the study. After a clinical examination and exercise test followed by measurement of intramuscular pressure, the diagnosis was made and the cohort was divided into patients with CACS (n = 87) and patients without CACS (n = 72).

Exercise

All of the patients performed an exercise test after the clinical examination. The test was individualized to elicit the patient's leg pain and muscle dysfunction. The test started with running activity—on a treadmill, up and down stairs, or outside in the park—which was followed by concentric and eccentric dorsi- exion of the ankle joints in a standing position until the patient was unable to continue because of muscle exhaustion and/or leg pain.

Monitoring StO_2

To measure StO_2, a near-infrared spectroscopy device (InSpectra Tissue Spectrometer, model 325; Hutchinson Technology) was used. This device employs infrared light with wavelength signals between 650 and 900 nm. The center of separation between the light source and the detector is 25 mm, and approximately 95% of the detected optical signal is from a 0 to 23-mm depth[9]. Values were collected continuously every 3.5 seconds throughout the measurements. Each measurement was marked to indicate the time for the start of exercise, the end of exercise, and the end of recovery. Calibration was performed before all measurements by placing the probe in a calibration box.

StO_2 was measured continuously in the anterior tibial muscle before, during, and after the exercise test. A near-infrared spectroscopy probe was placed centrally over the anterior tibial muscle of the more symptomatic leg of each patient. The measurement was started with the patient resting supine and was continued while the patient was exercising in a standing position and then during a recovery period with the patient resting supine. The StO_2 was analyzed at baseline before exercise, when the value was lowest during exercise (peak exercise), and during the recovery phase. De nitions of the measurement points are given in Table I and shown in Figure 1.

Fig. 1

An example of muscle oxygen saturation (StO_2) before, during, and at rest after exercise. A = baseline, prior to the exercise test; B = the lowest level of StO_2 during exercise; C = end of exercise followed by the reoxygenation phase; and D = the maximum level of StO_2 at rest after exercise. T50, T90, and T100 describe the time in seconds required for the level of StO_2 at the end of exercise to reach 50%, 90%, and 100% of its baseline.

Monitoring Intramuscular Pressure

A microcapillary infusion system (Hemo 4; Siemens) and monitor (SC9000; Siemens) was used to measure intramuscular pressure, with the patient in a supine position, within sixty seconds after the exercise test[10]. The skin was penetrated by a needle 1.2 mm in diameter, with four side-holes at its tip. The needle was inserted at a 30 angle to the long axis of the leg in a distal direction into the belly of the anterior tibial muscle[10] parallel with the bers in the muscle. The measurements were performed in the more symptomatic leg of each patient. There was an infusion of 0.9% saline solution through the system and out at the tip of the needle, with an infusion rate of 0.2 mL/hr, to maintain the bulging of uid at the tip of the needle at the beginning of the measurement. There was no infusion during the test itself. The tip of the catheter and the transducer were placed at heart level to minimize the hydrostatic artifacts, and the position of the catheter tip was controlled by viewing under ultrasound. The patient lay supine in a relaxed position with the legs extended[11].

Blood Pressure and Local Perfusion Pressure

Blood pressure was measured before and after the exercise test using a pressure manometer (NAIS [Matsushita Electric Works]). Local perfusion pressure was calculated for all patients as the difference between the mean arterial blood pressure and the intramuscular pressure.

Ultrasound Imaging

The skin and subcutaneous tissue thickness above the anterior tibial muscle fascia (at the same anatomic site as the near-infrared spectroscopy-probe placement and the intramuscular pressure measurement) and the distance between the fascia and the tip of the intramuscular pressure needle were measured using ultrasound imaging with a linear probe (L10-5, Acuson CV70; Siemens)[9] with the subject in a supine position and with relaxed muscles.

Surface Electromyography (EMG)

To control the muscular activity with use of EMG, two bipolar surface electrodes (pre-gelled Blue Sensor; Medicotest) were placed 15 cm below the knee joint, and a reference electrode was placed on the lateral malleolus. The EMG signals were recorded during and after the exercise test and were ampli ed in two steps: rst by a custom-made pre-ampli er (gain 100, high-pass second-order Butterworth lter; fo = 10 Hz) and then by the main ampli er (variable gain 1 to 196, low-pass second-order Butterworth lter; fo = 2 kHz) before recording with a data acquisition system (Pentium III PC, 12-bit Data Acquisition [DAQ] board and LabVIEW Software; National Instruments)[10].

Visual Analog Scale

The intensity of leg pain was evaluated with a visual analog scale (VAS) ranging from 0 cm (no pain) to 10 cm (worst imaginable pain).

Data Analysis

To facilitate comparisons with previous studies, reoxygenation rates at rest after exercise were calculated as the time period required for the level of StO_2 to reach 50% (T_{50}), 90% (T_{90}), and 100% (T_{100}) of the baseline value. The results are given as the median and range. Signi cance was set at $p < 0.05$. The intergroup difference was determined using the Mann-Whitney U test. The correlation between T_{90} and perfusion pressure was determined using the Pearson correlation test. Sensitivity and speci city were calculated to analyze the usefulness of changes in StO_2 for the diagnosis of CACS.

Source of Funding

There was no external funding source for this study.

The median baseline StO_2 was 92% (range, 45% to 98%) in the patients with CACS and 84% (range, 40% to 98%) in the patients without CACS. The baseline values showed considerable inter-individual variation and differed signi cantly between the two patient groups ($p = 0.005$).

Exercise caused the baseline values to decrease to a median of 1% (range, 1% to 36%) in the patients with CACS and 3% (range, 1% to 54%) in the patients without CACS. The sensitivity of a peak-exercise StO_2 of £8% as an indicator of CACS was 34% and the speci city was 43%. The sensitivity and speci city of a peak-exercise StO_2 of £50% as an

Parameter	Median (Range)		P Value*
	Patients with CACS (N = 87)	Patients without CACS (N = 72)	
Baseline StO_2 (%)	92 (45-98)	84 (40-98)	0.005
StO_2 during exercise (%)			
Peak exercise	1 (1-36)	3 (1-54)	0.003
End of exercise	1 (1-68)	17 (1-69)	0.001
At rest after exercise			
T_{50} (sec)	7 (1-43)	10 (1-49)	0.001
T_{90} (sec)	28 (7-77)	32 (4-138)	0.013
T_{100} (sec)	42 (7-200)	48 (4-180)	NS
Maximum recovery of StO_2 (%)	95 (57-98)	93 (38-98)	0.001

*For difference between patients with and those without CACS. NS = not signi cant.

indicator of CACS were both 1%. When a percentage change between the peak-exercise StO_2 and the baseline StO_2 of ‡40% was evaluated as an indicator of CACS, the sensitivity was 94% and the speci city was 20%.

At rest after exercise, T_{50} (the time period required for the StO_2 to reach 50% of the baseline) was a median of seven seconds (range, one to forty-three seconds) in the patients with CACS and ten seconds (range, one to forty-nine seconds) in the patients without CACS. T_{90} was a median of twenty-eight seconds (range, seven to seventy-seven seconds) and thirty-two seconds (range, four to 138 seconds), respectively, and T_{100} was a median of forty-two seconds (range, seven to 200 seconds) and forty-eight seconds (range, four to 180 seconds), respectively (Table II).

Parameter	Median (Range)		P Value*
	Patients with CACS (N = 87)	Patients without CACS (N = 72)	
Post-exercise intramuscular pressure (mm Hg)			
All patients	45 (30-111)	16 (5-28)	0.001
Female	38 (30-72)	16 (5-28)	
Male	47 (30-111)	17 (6-25)	
Mean arterial blood pressure (mm Hg)			
Before exercise	88 (73-106)	83 (72-128)	0.005
At rest after exercise	92 (92-129)	89 (68-129)	0.014
Post-exercise perfusion pressure (mm Hg)	47 (2 11-73)	71 (50-117)	0.001
Exercise time (sec)	52 (12-315)	59 (27-174)	NS
VAS (cm)			
Before exercise	0 (0-8)	0 (0-6.3)	NS
Maximum during exercise	6.7 (0-10)	6.7 (0-10)	NS
Thickness of skin and subcutaneous tissue (mm)			
All patients	4.4 (2.2-10.4)	5.2 (1.8-13.7)	NS
Female	6.4 (2.8-9.3)	6.0 (2.2-13.7)	
Male	3.7 (2.2-10.4)	3.6 (1.8-9.6)	
Distance between fascia and tip of intramuscular pressure needle (mm)	5.8 (2.4-13.9)	6.0 (0.8-13.6)	NS

*For difference between patients with and those without CACS. NS = not signi cant.

When T_{90} was set at thirty seconds or more, its sensitivity to diagnose CACS in patients with exercise-induced leg pain was 38% and its speci city was 50%. The positive predictive value was 48% and the negative predictive value was 40%.

The median intramuscular pressure was 45 mm Hg (range, 30 to 111 mm Hg) in the eighty-seven patients with CACS and 16 mm Hg (range, 5 to 28 mm Hg) in the seventy-two patients without CACS one minute post-exercise. The median intramuscular pressure one minute post-exercise differed between males (47 mm Hg [range, 30 to 111 mm Hg]) and females (38 mm Hg [range, 30 to 72 mm Hg]) with CACS ($p = 0.054$). The median local perfusion pressure post-exercise was 47 mm Hg (range, 2 11 to 73 mm Hg) in the patients with CACS and 71 mm Hg (range, 50 to 117 mm Hg) in the patients without CACS (Table III). It exceeded 30 mm Hg at rest after exercise in 86% of the patients with CACS. It was not correlated with T_{50}, T_{90}, or T_{100} in either patient group.

The median maximum intensity of leg pain during exercise was 6.7 cm (range, 0 to 10 cm) on the VAS in both patient groups (Table III). There was no signi cant difference between the two groups in terms of exercise time.

The thickness of the skin and subcutaneous tissue was 4.4 mm (range, 2.2 to 10.4 mm) in the patients with CACS and 5.2 mm (range, 1.8 to 13.7 mm) in the patients without CACS (Table III).

The present study of patients with exercise-induced leg pain suggests that changes in StO_2 during and after an exercise test cannot be used to distinguish patients with CACS from those without CACS. Since the patients were free from symptoms at rest, we interpret the clinical signs during and after exercise that elicits leg pain as key for establishing the diagnosis. The need for consensus regarding, and a standardized approach to, intracompartmental pressure testing has been discussed[12]. We chose to individualize the exercise test because the intensity and duration of the test might differ as a result of an individual patient's tolerance of leg pain. This may affect the patient's level of StO_2 during and following exercise. As StO_2 decreases during exercise, patients with or without CACS may develop leg pain as a result of the accumulation of metabolites such as potassium and hydrogen ions and lactate that increase in the interstitial space. There was no signi cant difference in leg pain during exercise between the two groups. Pain may also be related to comorbidity, such as medial tibial syndrome, peroneal nerve entrapment, traction periostalgia, or in ammatory reaction. Some patients who feel pain developing slow the exercise intensity before they actually stop exercising. This is one possible mechanism explaining how exercise-induced leg pain may affect the outcome of the test. Our results show that the StO_2 level toward the end of exercise actually increases.

In the present study, the StO_2 level at peak exercise did not differ between the patients with CACS and those without CACS. This nding contradicts the ndings of previous studies[2,3]. The StO_2 difference of only 2% in our study cannot be used in a clinical setting to identify patients with or without CACS. It is not clinically relevant. To estimate the value with the highest sensitivity and speci city, several cutoff values were analyzed. The sensitivity was 34% when an StO_2 level of £8% at peak exercise was chosen to indicate CACS. A previous study[13] demonstrated a sensitivity of 78% when a cutoff StO_2 value of £50% at peak exercise was used to indicate CACS. When using the same cutoff value in the present study, we obtained a sensitivity of only 1%. Moreover, the previous study[13] revealed a sensitivity of 84% and a speci city of 67% when a ‡40% change between the peak-exercise StO_2 and the baseline StO_2 was used to indicate CACS. The sensitivity was 94% when we applied the same cutoff value in the present study, but the speci city was only 20%. However, the previous study[13] comprised forty-two patients with CACS and only three patients without CACS. The results of the present study are based on eighty-seven patents with CACS and seventy-two patients without CACS.

The level of StO_2 re ects a variation in work intensity and can be in uenced by differences in leg pain among patients. The change in StO_2 during work is not a reliable parameter with which to diagnose CACS. This is due to the small difference in StO_2 level between patients with and those without CACS and the poor sensitivity of peak-exercise StO_2 as an indicator of CACS. As noted, the reoxygenation rate after exercise was determined on the basis of three parameters, T_{50}, T_{90}, and T_{100}, to make our results comparable with those of others[2]. We did not observe a slower recovery of StO_2 in patients with CACS compared with patients without CACS. This is in contrast to the ndings in previous studies[3,14]. We chose T_{90} of thirty or more seconds as a cutoff value to indicate CACS, as this value produced the highest sensitivity in the reoxygenation phase and thirty seconds was the mean value for T_{90} among the patients with CACS. The poor sensitivity (38%) and speci city (50%) of T_{90} indicate its limitations in diagnosing CACS.

The relatively large cohort revealed an interesting result for the post-exercise intramuscular pressure values. The median intramuscular pressure value at one minute post-exercise in the CACS group was 9 mm Hg higher in males than in females. This result raises the question of whether the diagnostic criteria for CACS should be adjusted and measured separately for males and females. It is possible to speculate about whether there are biological differences between these two groups. Despite an elevation in intramuscular pressure, the local perfusion pressure was not signi cantly correlated with the time needed for reoxygenation in the present study. After the exercise test, the local perfusion pressure exceeded 30 mm Hg in the majority of the patients with CACS. A previous study by Heppenstall et al.[15] showed no metabolic abnormalities at local perfusion pressures exceeding 30 mm Hg. Our ndings of normal reoxygenation after exercise are supported by that nding.

The baseline StO_2 showed considerable inter-individual variation in the present study. Previous studies have demonstrated a signi cant negative correlation between skin and subcutaneous tissue thickness and StO_2 values measured with an InSpectra[9,16]. This means near-infrared spectroscopy would

differ in how it re ected StO_2 values depending on the thickness of the skin and subcutaneous tissue. We therefore measured the distance between the skin and fascia, which did not differ signi cantly between the diagnostic groups. However, in individual cases, the subcutaneous tissue thickness varied much more, between 2 and 14 mm. Patients without CACS had a lower median StO_2 at baseline, and this can be partially explained by the thickness of the skin and subcutaneous tissue. This should be regarded as a limitation of the near-infrared spectroscopy method.

The study comprised a large group of patients with chronic exercise-induced leg pain. The diagnosis of CACS is based on patient history, clinical examination, and an exercise test that elicits the patient's symptoms followed by intramuscular pressure measurements. The tip of the intramuscular pressure needle was controlled with use of ultrasound. Muscle activity was controlled with use of EMG.

A limitation of this study is that the exercise test was individualized, with each patient performing the test at the highest possible intensity to elicit the symptoms for which he/she had been referred. This could be a limitation when individuals and groups are compared. Also, the fact that measurement of StO_2 with the near-infrared spectroscopy technique is affected by subcutaneous tissue thickness could be regarded as a limitation for the use of near-infrared spectroscopy in the clinical setting.

In conclusion, changes in StO_2 during and after an exercise test that elicits the patient's leg pain cannot be used to distinguish patients with CACS from patients with other causes of exercise-induced leg pain.

Kajsa Rennerfelt, MD, PhD[1],
Qiuxia Zhang, PhD[1],
Jón Karlsson, MD, PhD[1],
Jorma Styf, MD, PhD[1]

[1]Department of Orthopaedics,
University of Gothenburg,
Sahlgrenska University Hospital,
Gothenburg, Switzerland

E-mail address for K. Rennerfelt: kajsa.rennerfelt@vgregion.se

1. Styf J, Körner L, Suurkula M. Intramuscular pressure and muscle blood ow during exercise in chronic compartment syndrome. J Bone Joint Surg Br. 1987 Mar;69(2):301-5.
2. van den Brand JG, Verleisdonk EJ, van der Werken C. Near infrared spectroscopy in the diagnosis of chronic exertional compartment syndrome. Am J Sports Med. 2004 Mar;32(2):452-6.
3. Mohler LR, Styf JR, Pedowitz RA, Hargens AR, Gershuni DH. Intramuscular deoxygenation during exercise in patients who have chronic anterior compartment syndrome of the leg. J Bone Joint Surg Am. 1997 Jun;79(6):844-9.
4. Scheeren TW, Schober P, Schwarte LA. Monitoring tissue oxygenation by near infrared spectroscopy (NIRS): background and current applications. J Clin Monit Comput. 2012 Aug;26(4):279-87. Epub 2012 Mar 31.
5. Chance B, Dait MT, Zhang C, Hamaoka T, Hagerman F. Recovery from exercise-induced desaturation in the quadriceps muscles of elite competitive rowers. Am J Physiol. 1992 Mar;262(3 Pt 1):C766-75.
6. Zhang Q, Rennerfelt K, Styf J. The magnitude of intramuscular deoxygenation during exercise is an unreliable measure to diagnose the cause of leg pain. Scand J Med Sci Sports. 2012 Oct;22(5):690-4. Epub 2011 Sep 27.
7. Pedowitz RA, Hargens AR, Mubarak SJ, Gershuni DH. Modi ed criteria for the objective diagnosis of chronic compartment syndrome of the leg. Am J Sports Med. 1990 Jan-Feb;18(1):35-40.
8. Styf JR, Körner LM. Diagnosis of chronic anterior compartment syndrome in the lower leg. Acta Orthop Scand. 1987 Apr;58(2):139-44.
9. Nygren A, Rennerfelt K, Zhang Q. Detection of changes in muscle oxygen saturation in the human leg: a comparison of two near-infrared spectroscopy devices. J Clin Monit Comput. 2014 Feb;28(1):57-62. Epub 2013 Jul 12.
10. Zhang Q, Jonasson C, Styf J. Simultaneous intramuscular pressure and surface electromyography measurement in diagnosing the chronic compartment syndrome. Scand J Med Sci Sports. 2011 Apr;21(2):190-5.
11. Gershuni DH, Yaru NC, Hargens AR, Lieber RL, O'Hara RC, Akeson WH. Ankle and knee position as a factor modifying intracompartmental pressure in the human leg. J Bone Joint Surg Am. 1984 Dec;66(9):1415-20.
12. Hislop M, Tierney P. Intracompartmental pressure testing: results of an international survey of current clinical practice, highlighting the need for standardised protocols. Br J Sports Med. 2011 Sep;45(12):956-8.
13. van den Brand JG, Nelson T, Verleisdonk EJ, van der Werken C. The diagnostic value of intracompartmental pressure measurement, magnetic resonance imaging, and near-infrared spectroscopy in chronic exertional compartment syndrome: a prospective study in 50 patients. Am J Sports Med. 2005 May;33(5):699-704. Epub 2005 Feb 16.
14. Breit GA, Gross JH, Watenpaugh DE, Chance B, Hargens AR. Near-infrared spectroscopy for monitoring of tissue oxygenation of exercising skeletal muscle in a chronic compartment syndrome model. J Bone Joint Surg Am. 1997 Jun;79 (6):838-43.
15. Heppenstall RB, Sapega AA, Scott R, Shenton D, Park YS, Maris J, Chance B. The compartment syndrome. An experimental and clinical study of muscular energy metabolism using phosphorus nuclear magnetic resonance spectroscopy. Clin Orthop Relat Res. 1988 Jan;226:138-55.
16. van Beekvelt MC, Borghuis MS, van Engelen BG, Wevers RA, Colier WN. Adipose tissue thickness affects in vivo quantitative near-IR spectroscopy in human skeletal muscle. Clin Sci (Lond). 2001 Jul;101(1):21-8.