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Intramuscular pressure, tissue oxygenation and EMG fatigue measured during isometric fatigue-inducing contraction of the multifidus muscle

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Abstract Simultaneous measurement of intramuscular pressure (IMP), tissue oxygen partial pressure (pO_2) and EMG fatigue parameters in the multifidus muscle during a fatigue-inducing sustained muscular contraction. The study investigated the following hypotheses: (1) Increases in IMP result in tissue hypoxia; (2) Tissue hypoxia is responsible for loss of function in the musculature. The nutrient supply to muscle during muscle contraction is still not fully understood. It is assumed that muscle contraction causes increased tissue pressure resulting in compromised perfusion and tissue hypoxia. This tissue hypoxia, in turn, leads to muscle fatigue and therefore to loss of function. To the authors' knowledge, no study has addressed IMP, pO_2 and EMG fatigue parameters in the same muscle to gain a deeper sight into muscle perfusion during contraction. As back muscles need to have a constant muscular tension to maintain trunk stability during stance and locomotion, muscle fatigue due to prolonged contraction-induced hypoxia could be an explanation for low back pain. Sixteen healthy subjects performed an iso-

metric muscular contraction exercise at 60% of maximum force until the point of localized muscular fatigue. During this exercise, the individual changes of IMP, pO_2 and the median frequency (MF) of the surface EMG signal of the multifidus muscle were recorded simultaneously. In 12 subjects with a documented increase in intramuscular pressure, only five showed a decrease in tissue oxygen partial pressure, while this parameter remained unchanged in six other subjects and even increased in one. A fall in tissue pO_2 was associated with a drop in MF in only five subjects, while there was no correlation between these parameters in the other 11 subjects. To summarize, an increase in IMP correlated with a decrease in pO_2 and a drop in MF in only five out of 16 subjects. High intramuscular pressure values are not always associated with a hypoxia in muscle tissue. Tissue hypoxia is not automatically associated with a median frequency shift in the EMG signal's power spectrum.

Keywords Tissue oxygenation · Muscular fatigue · Intramuscular pressure · Multifidus muscle · Isometric back extension

Introduction

It has been proposed that muscle fatigue is associated with low back pain [33]. The energy consumption resulting from the muscle contraction is considered to be

the cause of the muscle fatigue. It is fair to assume that the degree of individual nutritive perfusion of the muscles can be either a promotive or a protective factor in the development of back pain.

The physiological mechanisms that maintain a sufficient perfusion in the muscle—and thus the oxygen supply to the tissue—during exertion are largely hypothetical and described in models.

The physiological model of the chronic functional compartment syndrome (CCS) gives an explanation for exercise induced muscular pain while it also describes the physiological processes of muscle blood flow under load. According to the model, a muscle contraction leads to an increase in intramuscular pressure [20]. When the compartment pressure rises above a physiological threshold, the blood supply is reduced and the tissue's oxygen content decreases [11, 28, 44]. The decreasing oxygen content of the tissue leads to muscle fatigue and muscular dysfunction [8, 26]. Therefore the central hypotheses on which this model is based are as follows:

1. Increases in IMP result in tissue hypoxia;
2. Tissue hypoxia is responsible for muscular fatigue and loss of function.

The IMP is determined as a parameter for the muscle contraction which represents the trigger mechanism for compromising blood perfusion. The measurement of pO_2 allows the assessment of the tissue hypoxia which is expected as a result of the rise in pressure. The muscle fatigue resulting from the hypoxia is to be assessed by determining the median frequency shift.

To this day, the diagnosis of a CCS is based on the presence of the typical clinical symptoms in combination with high intramuscular pressure values. Nevertheless, considerable controversy exists regarding the optimal pressure criteria for the diagnosis of CCS. Both the threshold values and the time of evaluation (before, during or after exercise) are still under discussion. Perhaps one will never be able to distinguish between the group of patients with CCS and patients with other causes of pain or healthy individuals merely on the basis of IMP. It is possible that additional parameters have to be determined in order to guarantee a reliable distinction between the groups. Therefore the objective of the present study was to test the central parameters of the above hypotheses in healthy subjects and to gain more information regarding the nutrient supply to multifidus muscle during exertion. To our knowledge, no previous studies have addressed all three parameters (IMP, pO_2 , median frequency shift) simultaneously in a single paravertebral muscle compartment. In comparison to

clearly defined “compartment” situations at extremities, in which clinical IMP measure is daily routine, there are today controversial discussions about the existence of real compartments in paravertebral muscles. Therefore it seems not to be suitable to use the research data of compartment models at extremities. The results of healthy subjects in this study could be a reference database for future studies in patients with back trouble, especially exercise induced low back pain.

Subjects, material and methods

Subjects

Sixteen subjects (ten males, six females) with no pathology of the back participated in the study. Inclusion criteria for the study were as follows: age between 25 years and 50 years, and body-mass index (BMI) of 20–28 kg/m². Blood pressure had to be not higher than 135 mmHg systolic and 80 mmHg diastolic. In addition, self-reports of back pain, if present, had to be < 2 on a 100 mm visual analog scale (VAS) ranging from 0 (no pain) to 10 (excruciating pain). Table 1 shows subjects' characteristics with respect to sex, age, height, weight and BMI.

The study design was submitted to the ethics committee of the University of Ulm and approved (No. 45/97, January 13, 1998). All subjects gave their informed written consent prior to inclusion in the study.

Preparation for catheter implantation

Subjects were placed in prone position on an examination table. Following careful cleansing, removal of fatty secretions and disinfection of the skin, a catheter (see below) was placed at the level of the third lumbar vertebra. Depending on handedness, one side of the body was chosen for measurement based on the fact that static stress is more intense on the side of the body contralateral to the side of motor dominance. Catheters were fixed in place using strips of tape, which prevented their accidental dislocation.

Intramuscular pressure (IMP) measurement

Local anesthesia was induced by injection of local anesthetic agent (1 ml) down to the fascia at a point one

Table 1 Demographic data for the study population, broken down by sex

	Males (<i>n</i> = 10)			Females (<i>n</i> = 6)		
	Median	Min/max	25%/75%	Median	Min/max	25%/75%
Age (years)	30	25/47	26.5/32	25	25/29	25.3/28.8
Height (cm)	1.82	1.7/1.89	1.76/1.84	1.63	1.63/1.86	1.65/1.8
Weight (kg)	78.5	68/90	70.8/83.5	67	60/77	63.0/74.3
BMI (kg/m ²)	23.9	21.8/27.8	23.0/24.9	22.3	22.3/24.1	22.4/23.2

BMI body mass index

centimeter lateral to the midline. A piezoelectric pressure catheter (ARGUS, MIPM GmbH, Mannendorf, Germany) was then introduced through an indwelling venous catheter into the multifidus muscle in caudal direction at an angle of 45°. The catheter was introduced two more centimeters after having passed the resistance of the fascia. The analog signal of the catheter was digitized at 10 Hz and recorded for later evaluation.

The piezoresistive measuring method is based on the physical fact that semiconductors change their specific resistance under pressure. The silicon semiconductor is covered by a silicone sheath which is located in a metal cage. The cage has a small window where the silicone layer is exposed. This is where the pressure is absorbed indirectly via a blood lake that surrounds the catheter tip or directly by soft tissue. The probe provides excellent dynamic response to changes in IMP without artifacts from saline columns [13, 47].

Measurement of tissue oxygen partial pressure (pO₂)

The same technique was used for implantation of the pO₂-catheter (LICOX C1-Sonde, GMS, Mielkendorf, Germany) into the multifidus musculature. Implantation was performed at a spot 2 cm lateral to midline. The technique and processing of the data is described in detail by Boekstegers et al. [2]. In addition, a temperature measurement catheter (LICOX C8-Sonde, GMS, Mielkendorf, Germany) was also implanted into the contralateral multifidus muscle in order to correct temperature dependent pO₂-drift. Data were digitized at 0.2 Hz and recorded.

Electromyography (EMG)

Surface electrodes were attached bilaterally to the skin above the multifidus muscle below the points of insertion of the catheters. Potentials were recorded in bipolar fashion with a reference electrode over the vertebra prominens. Self-adhesive 1.2-cm silver/silver-chloride surface electrodes with gel cushion were used. Interelectrode distance was 2 cm. The EMG raw signal was recorded with a band width of 5–1,000 Hz and digitized at 2,000 Hz. Data were recorded for subsequent evaluation. The median frequency shift of the power density spectrum of the myoelectric signal was calculated as a parameter indicative of fatigue.

Data recording/experimental structure

Subjects were studied using fitness-training equipment (Model R 14; mkb-Systems, 88481 Balzheim, Germany). The unit permitted determination of maximum force and the exercise load on the subject could be set at 3-kg intervals as a percentage of maximum force. This

experimental design allowed force measurement with pre-extension of the musculature, as recommended by Tan et al. [40].

Following determination of maximum force, parallel recording of IMP, pO₂, and the EMG signal of the multifidus muscle was initiated during sustained isometric exercise at 60% of maximum force with the load pushing the subject into flexion. The subject was verbally motivated to resist the load as long as possible. As IMP increases with the extent of lumbar flexion measurement was only continued until the load resulted in a trunk flexion of > 10° due to muscle fatigue. Trunk movements were visually controlled on a protractor.

Data analysis/statistics

The study was conducted to gain first experience of the mentioned parameters during an isometric fatiguing contraction of the multifidus. Evaluation was therefore descriptive for all parameters. During the time of contraction the changes in IMP, pO₂ and MF were calculated at 25, 50, 75 and 100% of time. The parameters have been normalized to the values at 0% time. Therefore the baseline is one. Because the parameters recorded in this study show small variations even at rest, changes < 2% were defined as “no change”. Data are presented as rank distribution (box plots).

Results

Intramuscular pressure (IMP)

The IMP baseline values at rest in prone and sitting position are shown in Table 2. The IMP measured at initiation of sustained contraction exercise showed great interindividual variability (Table 2). Increases in IMP were observed in 12 out of 16 subjects during isometric exercise at 60% of maximum force. These increases ranged from 4% to 314% of baseline. Conversely, the remaining four subjects showed a decline in IMP that ranged from 4% to 30%. Before and after the tests the intramuscular position was checked by voluntary contractions that lead to simultaneous increases in IMP (Fig. 1).

Measurement of tissue oxygen partial pressure (pO₂)

The baseline pO₂ levels measured at rest in sitting position and the pO₂ during initiation of sustained contraction exercise also showed great inter-individual variability (Table 2). During exercise, decreases in tissue pO₂ of 1–90% were observed in 11 subjects, while 4 others showed increases of 1–9% over baseline. In the remaining subject, no change in pO₂ was observed (Fig. 2).

Table 2 Rank distribution values for the three measured parameters at rest and at initiation of sustained contraction

	IMP (mmHg)			pO ₂ (mmHg)		MF (Hz)	
	At rest prone	At rest sitting	At initiation of contraction	At rest sitting	At initiation of contraction	At rest sitting	At initiation of contraction
Minimum	0.0	1.4	15.4	4.2	3.2	32.5	31.8
Quartile 25%	7.0	20.1	116.8	28.8	33.4	41.3	43.8
Median	9.3	24.3	186.6	41.5	44.6	44.5	48.1
Quartile 75%	12.8	31.2	248.6	47.2	50.2	52.9	52.9
Maximum	22.5	97.3	375.4	57.3	63.0	70.5	76.1

EMG fatigue (MF shift)

Great interindividual variability at rest and at initiation of contraction was also observed in this parameter (Table 2). While 13 subjects showed a decrease in MF of 2–34% of baseline, 3 subjects exhibited an increase in MF of 2–12% (Fig. 3).

Intraindividual comparison of IMP, pO₂ and MF shift (Table 3)

In four subjects, increases in IMP were accompanied by a drop in tissue pO₂ and localized muscular fatigue. One subject showed an increase in IMP and a decrease in pO₂ but no muscular fatigue. Six further subjects showed both an increase in IMP and signs of muscular fatigue but constant tissue pO₂. Subject 7 exhibited an increase in pO₂ of 9% despite constant pressure readings and signs of muscular fatigue.

In the four subjects exhibiting a fall in IMP during exercise, one showed signs of muscular fatigue with a simultaneous drop in pO₂, while two others showed no

muscular fatigue despite a drop in pO₂. Finally, one subject showed neither a drop in pO₂ nor signs of muscular fatigue.

Fig. 2 Tissue pO₂ during sustained isometric contraction at 60% maximum force. pO₂ values have been normalized to the level at 0% time. Baseline therefore is one

Fig. 1 IMP during sustained isometric contraction at 60% maximum force. IMP values have been normalized to the level at 0% time. Baseline therefore is one

Fig. 3 Median frequency changes during sustained isometric contraction at 60% maximum force. Median frequency values have been normalized to the level at 0% time. Baseline therefore is one

Discussion

The objective of the present study was to measure intramuscular tissue pressure (IMP), tissue oxygen partial pressure (pO_2) and localized muscular fatigue in the multifidus musculature during isometric exertion.

It was shown that the hypothetical model of nutritive muscle perfusion in the exercising muscle could not be sufficiently explained on the basis of experimental findings in the majority of subjects (11 out of 16) studied. It was shown that high IMP leads to a decrease in tissue oxygenation in only half the subjects and that tissue oxygenation is not strongly associated with muscular fatigue.

Muscle contraction was shown to increase IMP [38]. IMP values as high as 500 mmHg are generated during normal vigorous skeletal muscle contraction [34]. This increase in IMP is expected to be the most relevant pathophysiologic factor compromising the blood perfusion of tissue [14]. During sustained exercise IMP is additionally affected by an increase in muscle volume of up to 20% via increased capillary perfusion, blood volume, and interstitial edema [31]. In this situation, oxygen diffusion has to cover a greater distance, which can be expected to cause hypoxia and muscle fatigue [39]. The level of IMP at which blood perfusion is compromised is still the subject of heavy discussion [14].

Studies using external compression and artificial interstitial edema due to venous obstruction in order to simulate increased IMP show a range of between 30 mmHg [44] and 80 mmHg [5] as a threshold at which tissue perfusion is compromised.

In the present study, 14 out of 16 subjects showed IMP values in excess of 80 mmHg at the same relative load levels for the entire period of exercise. Similarly high pressure values at rest and during contraction are also

described in other studies [25, 30, 37]. As fluctuations of IMP and pO_2 to some extent may exist in skeletal muscles, possible errors due to the measuring technique or the position of the catheter in the muscle can partly explain the wide range of pressure values, however, they do not constitute a sufficient explanation for the absolute values and not at all for the parameter course during exertion.

Based on the criteria discussed above, adequate supply of oxygen to the muscle under these conditions is not to be expected. However, only five subjects showed a decrease in pO_2 (5–89%), while in six others increased IMP appeared to have no effect on pO_2 (range: –2 to +2%). In one subject, an increase in pO_2 of 9% was observed at an IMP of 274 mmHg.

These results are only partly consistent with those in the literature. Jensen et al. [20] found a significant decrease in the oxygenation of the paravertebral muscles with corresponding intramuscular pressure levels of between 30 mmHg and 40 mmHg. However, these results are only given for six out of nine subjects in the study. It is not reported which criteria led to the exclusion of the three other individuals. The study describes the results as means + standard error of mean. This statistical description makes it impossible for the reader to estimate the range of the parameters. It is thus unclear whether this study also found major differences in the muscle oxygenation during exercise between individual subjects.

Styf and Lysell [37] measured intramuscular pressures under repetitive contractions in 12 highly selected patients. Selection criteria were low back pain induced only by exercise, relief of symptoms at rest and no neurological deficits. On the basis of the pressure values, the group was divided into patients with a chronic compartment syndrome (CS) of the paravertebral muscles and patients without a CS. The criteria for this division were defined by the authors as muscle relaxation pressure during exercise and IMP at rest after exercise exceeding 30 mmHg and no normalization of the pressure within 6 min after exercise. Only one individual met these pressure criteria and also showed a decrease in muscle blood flow. Although their pressure values were also high, no muscle blood flow measurements were performed for the individuals classified as non-CS. In this study, it is thus also unclear to what extent high intramuscular pressure values affect the muscle blood flow in individual cases.

The findings of our study suggest that the effect of high IMP as a mirror of muscle contraction on pO_2 may vary significantly and is apparently dependent on further parameters. In 1989, Heppenstall developed a theory for acute CS in an animal model, in which the critical perfusion pressure was not calculated in a simple linear relationship with the intramuscular pressure, as had been common practice until then, but as the difference between the mean arterial blood pressure and the intracompartmental pressure [16, 17]. But even this theory is frequently inadequate in the clinical

Table 3 Qualitative change in the individual parameters sorted by group

Subject no.	IMP	pO_2	MF Shift
6	>	<	<
10	>	<	<
13	>	<	<
16	>	<	<
14	>	<	=
1	>	=	<
2	>	=	<
4	>	=	<
8	>	=	<
9	>	=	<
15	>	=	<
7	=	>	<
5	<	<	<
12	<	<	=
3	<	<	>
11	<	=	>

IMP intramuscular pressure, pO_2 tissue oxygen partial pressure, MF median frequency

situation and appears to be incomplete for a sufficiently exact description of the actual perfusion conditions in the muscle. Heppenstall himself distinguishes between healthy individuals and patients with moderately traumatized muscles. Due to the different microcirculation in traumatized tissue, 40 mmHg is deducted from the mean arterial blood pressure for the patients whereas 30 mmHg is deducted for the healthy individuals [16]. It is likely that additional parameters are involved in the regulation of the microcirculation which make it impossible to determine an individual threshold value [48]. Such factors that might lead to individual abilities to maintain tissue pO_2 during exercise include increases in cardiac output and autoregulation of peripheral vasodilatation [12, 18, 21]. In addition, the different energy patterns of slow and fast-twitch fibers [42, 43], whose distribution varies between individuals, may affect the oxygen utilization of muscle.

In four subjects, exercise was associated with a decrease in IMP. The decrease in IMP may be the result of compensatory mechanisms. The strength of the erector spinae is generated in several muscles. A redistribution of the load during exercise may have resulted in a sparing of the multifidus with resulting decrease in IMP. An increase in intra-abdominal pressure may also result in muscular decompression while maintaining the load [15, 25]. Because high IMP levels were maintained during contraction (196.4–375.4 mmHg), it is clear that, despite a decrease in IMP, these proposed compensation mechanisms would still not assure sufficient perfusion and tissue pO_2 will fall (or remain constant, as was observed in one case).

Indeed, all these parameters may show individual differences in their effects. The additive and subtractive combination effects of these and other, unknown parameters yield the final, “actual” blood perfusion to the muscle.

The second hypothesis states that decreased pO_2 causes muscular fatigue followed by loss of function. In our study, both events predicted by these hypotheses occurred in only five out of 16 subjects while, in the other 11 subjects, either muscle fatigue or decrease in pO_2 occurred, but not both simultaneously.

Tissue pO_2 levels associated with a critical disturbance of nutrient supply have been measured in poorly perfused myocutaneous flaps. In such cases, pO_2 values of 11 mmHg have been measured, in comparison with pO_2 values of 35 mmHg in normally perfused flaps [46]. An anoxic situation is normally expected at pO_2 values of less than 5 mmHg [1, 10]. In the present study, a corresponding situation was observed in only two subjects who showed very low baseline pO_2 values of 3.2 mmHg and 5.0 mmHg, respectively. All other subjects showed pO_2 values above 18.4 mmHg after exercise. As in other studies, the pO_2 values measured in the present study showed wide interindividual variability [22, 23, 35]. Brückle et al. [3], who used a KIMOK histogram to study the paravertebral musculature of ten healthy sub-

jects, measured tissue pO_2 values of less than 10 mmHg with a frequency of ca. 6% and values above 60 mmHg with a frequency of about 4%. Using other measurement systems, other authors have found intraindividual pO_2 differences of up to 50% between adjacent areas of the same muscle and differences of 25–30% in sequential measurement [22, 35]. These data would suggest that variability in tissue pO_2 levels regularly occurs in different areas of the musculature and at different times.

In the setting of a dynamic study design, the use of a polarographic measurement method with a flexible catheter represented the only option for measurement of tissue pO_2 . The tissue displacement by the catheter represents a volume containing ca. 5,000 capillaries [41] and records a mean of intra- and especially extracellular space. Thus, tissue pO_2 readings provide information about a single, more or less large volume of the muscle compartment at time X, and may or may not be representative of the entire muscle. As well it has been found that IMP varies within a given muscle during sub-maximal contractions [34]. A possible explanation for the bad correlation between IMP and pO_2 might therefore be that the sites of measurement for the two techniques are different.

Tissue pO_2 also does not provide direct information about phosphorylative potential, redox potential and oxygen flow on the cellular level [6]. Transport of oxygen from the tissue into the cell depends on many parameters and has not been sufficiently studied [45]. Within myocytes, pO_2 values are in the range of 1–3 mmHg [45]. It is only when these values fall to levels below 0.5 mmHg that the function of the respiratory chain and energy metabolism is compromised [6]. This was explained on the basis of studies on isolated mitochondria, which showed that mitochondria exhibit unchanged oxygen consumption at pO_2 levels of between 0.05 mmHg and 0.1 mmHg [32, 45]. Hence, tissue pO_2 is not necessarily representative of the energy status of the cell. Poor energy conditions in the cell, however, are directly associated with loss of function in the cell.

Loss of function in the musculature has been described in another area of muscle physiology, namely, muscle fatigue. In 1973, Chaffin [4] defined localized muscular fatigue as loss of function in the musculature secondary to sustained muscle contraction. This phenomenon has been well studied in electromyographic investigations and is associated with a shift to lower frequencies in the power density spectrum of the ME signal [8, 9]. The median frequency (MF) shift has been described as a reliable parameter of localized muscular fatigue [24, 36].

This shift is caused by changes in the waveform of the motor-unit action potentials, due primarily to alterations in muscle fiber conduction velocity [9]. Conductivity in the muscle cell is affected primarily by the production of lactate and pyruvate during anaerobic energy metabolism, both of which are transferred into the interstitial space. At the same time, intra- and extracellular

accumulation of H^+ ions generated in the metabolic process also affect the conductivity of the muscle cell [19, 29]. Changes in MF, therefore, represent processes occurring at the level of the cell membrane. A change in tissue pO_2 represents aerobic energy metabolism. This parameter is not necessarily connected directly to the lactate or H^+ ion gradients at the muscle cell membrane.

In our experimental setting, based on exercise at 60% of maximum force, energy metabolism in the myocyte depends primarily on anaerobic, lactate-forming energy metabolism (glycolysis) once the supply of creatine phosphate has been exhausted [27]. Because aerobic energy metabolism requires 1–2 min to become effective [7], and because of the duration of exercise to fatigue (<120 s in 12 subjects), it is likely that aerobic energy metabolism does not play a significant role. In fact, aerobic energy metabolism may only become important in the process of rebuilding intracellular ATP stores and reduction in the oxygen deficit. Thus, it is conceivable that high lactate concentrations may occur in the presence of only a small drop in pO_2 . And this could explain

why a median frequency shift is associated with a drop in tissue pO_2 in only 5 out of 16 subjects. This makes it clear that an interpretation of muscle function only makes sense when many influence parameters are known.

Summary

The simultaneous recording of IMP, pO_2 , and MF of the ME signal allows a deeper insight of physiologic events in muscle during sustained contraction. Findings of the present study show that physiologic reactions to isometric sustained contraction are subject to wide individual variability. The criteria of the currently proposed physiologic model for chronic compartment syndrome were fulfilled in only 5 out of 16 subjects tested. In the remaining 11 subjects, findings can only be explained on the basis of additional parameters. The value of our present results for diagnosing exercise-induced muscle pain of the multifidus will require additional studies in the future.

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