Mechanical properties of morphological different muscles interpreted as a consequence of neural activation patterns - implications to specific training

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INTRODUCTION

As we know from several studies, the fiber structure and the mechanical properties of a muscle depends on the innervation frequency, innervation time (IT) and the innervation–rest ratio (Monster et al. 1978; Hennig and Lømo 1985; Gundersen and Eken 1992). Recently Hämälainen & Pette (1996) reported that it is possible not only to transform fast contracting MHC II fibers to slow contracting MHC I fibres (for review see Pette & Staron 1997 and Gundersen 1998) but also in the opposite direction. They demonstrated the transformation of MHC I/ MHC IIA to MHC IID/X / MHC IIB fibres of the rat soleus in response to a innervation frequency of 150 Hz, an IT of 166ms and an inter-attraction interval of 15 minutes. On the other hand high portion of type II fibres in the quadriceps of fast running animals like the African cheetah or human sprinters suggests a relationship between the innervation modalities, the morphological muscle structure and the functional output (Williams et al.1997; Mero et al. 1981). The aim of this study was to compare the innervation strategies of four different sports groups to discuss them under the focus of neuromuscular plasticity.

METHODS

EMG measurements were taken from the M. quadriceps femoris of volleyball players (first national league), sprinters (100m best time M = 10.8s), marathon runners (42,2km best time M = 2.40 h) and not specifically trained students of our physical education department in two different isometric explosive test procedures. In the Test ET1 (ET1) the subjects were asked to extend the lower leg as explosive and as maximal as possible. For the Test ET2 (ET2) the subjects were asked to contract with the association “explosive” only without any torque demands. Trials in ET1 were accepted above 75 % of the maximum torque (Tmax) measured in the MVCT before.

The EMGs of the VM, the VL and the RF were integrated from the beginning of the neural activity over a period of time which was identical with the time to peak of torque (TTPT). Additionally all EMG and force parameters were calculated for portions of 25% TTPT each (for more methodical details see Hering et al. 2001, Mechanical properties of morphological different muscles… in this volume).

RESULTS

1. While the association between explosive and maximal was expressed in a force production mostly between 80% and 100% (mean = 91%) of the maximal voluntary torque (MVT), the association to contract only fast was transformed with a maximal torque (Tmaxx) between 23% and 82% (mean = 58%) of MVT. In both tests the relation between IT and Tmaxx was quite variable (see Figure 3 ).
2. The mean IT of Test ET2 with 135ms was about half as long as that of Test ET1 with 260 ms.
3. The mean power frequency (MPF) and the mean electromechanical delay (EMDm) of the 3 muscles as well as the normalized torque-EMG ratio (RTn/EMGn) between the two contraction tasks were quite different between ET1 and ET2 (see Figure 1 and 2).

Figure 1: a. Mean-Power-Frequency (MPF) of ET1 and ET2 for all subjects (N=48) and b. ET2 for the sport groups N=4:12
4. The mean power frequency (MPF) and the mean electromechanical delay (EMDm) of the 3 muscles as well as the normalized torque-EMG ratio (RTn/EMGn) between the two contraction tasks were significantly different (EMDm ET1/ET2 = 31.7/28.5***).

5. The significant differences between the MPF for sprinters and volleyballers for the first 50% TTP in Test ET2 as well as the absent significant correlation between the MPF and the torque during the first 25% of the time to peak torque for VG suggests different physiological processes between both sports groups.

6. The volleyballers reached with lower MPF-values significant higher rates of force development (Trmaxn_ET2 SG = 1.13; VG 1.53 [%Tmax/ms]).

6. There were highly significant correlations between the 100m sprint time and relaxation parameters within the sprint group and across all subjects.

**Discussion**

Under the assumption, that short innervation times and large innervation frequencies are able to prevent MHC IID/X fibers to be transformed into MHC IIA fibers or as it was demonstrated by Hämälainen (1996), that short and phasic activation patterns initiate transformation processes from slow to fast MHC-isoforms respectively, the contraction form ET1 with the association “explosive and maximal” has to be discussed critically as a training exercise for sprinters. With a mean innervation time (IT) of 135ms the contraction form ET2 is shorter than the IT of 166ms, used by Hämälainen (1996). With a mean of 260ms the IT of ET1 it is nearly double of that of ET1. Moreover, the IT is in a range, where Gundersen & Eken (1992) found with a stimulation frequency of 150 Hz the isotonic contraction velocity of the extensor digitorum longus of the rat diminished.

Remembering that there is a significant correlation between the ground contact time during the push off (CTP) and the portion of FT-fibers for high-jumpers (Tihanyi, 1983) and that volleyballers, high- and long-jumpers have only about 48% fast twitch fibers with a CTP of 210-300ms, a mean innervation time of 260ms seems to be long for top sprinters with a CTP of 80-100ms (for more details see Hering, 2000).

Although many arguments favor ET2 as an adequate contraction strategy for sprint training, two conflicting results are not explained up to now. If, according to the literature, sprinters had much more fast twitch fibers than volleyballers, why is the torque rate of volleyballers higher in comparison to that of sprinters? And why is the maximal torque rate for ET2 (Trmaxn_ET2), associated to an explosive fast contraction, smaller than in ET1, which is associated to an explosive maximal contraction?

To begin with the last question, the Torque-EMG-Ratio (QTn/EMGmn) for the first 50% of the TTP_Torque for ET2 is significantly smaller than for ET1 (see Figure 2), meaning that the same torque in ET2 is produced with a larger amount of EMG. How can this be explained?

As we know from several studies the faster and thicker fibers are placed mostly at the muscle surface and they produce more EMG than the smaller slow contracting units. Assuming that fast twitch units were more or selectively recruited in ET2, the question arises again: “Why is this not reflected in the maximum torque rate?” The only explanation is:” In a concentric contraction, if the fast twitch units are recruited

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**Figure 2:** Torque-EMG-Quotienten for 25% steps of the TTPT. Comparison between ET1 and ET2; N = 48

**Figure 3:** Relation between innervation time and normalized torque rate for ET1: top and ET2: bottom; the solid line is set at IT=166 ms; N = 48
earlier or before the slower once, they are not able to transfer their contraction force immediately to the bone, because they end mostly mid-fascicularly between other fibers as described by Roy & Edgerton (1992).” The fast units have to stretch out first the elastic tissue, before the force can be developed directly. Therefore, the EMDm for ET2 is significantly shorter than in ET1. The significant correlations between the 100m sprint time and the relaxation parameters support this explanation, because the elastic tissue before the relaxation is stretched and the time course of the force decline is determined by the muscle fibers itself. If the elastic tissue would be stretched by an external force at the beginning of the contraction, the FT-fibers would be able to develop an intrinsic contraction potential. This assumption is strongly supported by the work of Hill 1970 (see Figure 4).

Answering the last question, we have to remember that with a high \( \text{Ca}^{2+} \) influx into the cell slow twitch fibers can reach twitch times comparable to that of FT-fibers (Kugelberger, 1983). The \( \text{Ca}^{2+} \) influx is determined generally by the innervation frequency and the volume of the sarcoplasmatic reticulum (SR). A larger SR releases a larger amount of \( \text{Ca}^{2+} \) per stimulus. Consequently, a lower innervation frequency is needed with a well developed \( \text{Ca}^{2+} \)-system to achieve comparable force rates. This would be a good explanation for the volleyballers significantly lower MPF in comparison to the MPF of sprinters. Because the transient \( \text{Ca}^{2+} \)-concentration in connection with the phosphorylization potential is discussed as a factor, initiating fiber transformation, the relative low portion of FT-fibers described for volleyballers is not surprising (for more details see Hering, 2000).

In conclusion, the large differences in the execution within and between the two contraction tasks ET1 and ET2, interpreted as a result of different recruitment and innervation strategies, suggest to be careful with uncontrolled training exercises because of unmeant physiological adaptations. For sprinters short and phasic innervation pattern in connection with stretch contractions are recommended. Training exercises for sprinters has to be distinguished from that of volleyballers or high- and long-jumpers because of generally different adaptation processes. Biofeedback may be a helpful procedure for the athletes.

**Literature**


