SUMMARY

The muscle fiber conduction velocity (MFCV) is the speed of the depolarization wave along the sarcolemma. This is a step in the cascade that causes the muscle to contract. This study describes two methods for the determination of the MFCV, an invasive and a surface EMG method. Each method has its specific advantages and limitations.

The invasive EMG method is performed in resting muscle, usually biceps brachii. The muscle is stimulated by a monopolar needle electrode, with a fixed frequency of 1 Hz, outside the end-plate region. A concentric needle electrode is inserted at some distance in the activated fiber bundle, and after optimal positioning a polyphasic potential is obtained. The MFCV is determined from the latencies of the potentials and the distance between the stimulation electrode and the recording electrode.

The surface EMG method is performed in voluntarily activated muscle. Two bipolar surface electrodes are placed in a linear array, parallel to the muscle fibers, nearly halfway between the endplate zone and the distal tendon. The MFCV is calculated from the latency shift between the two EMG signals. Other EMG parameters are also measured. The most studied parameters are: (1) the MFCV, based on the time shift between the two signals by means of the cross-correlation method; (2) the power spectrum of the signal; the position of this spectrum is reflected by the value of the median frequency (Fmed); (3) the energy content of the signal, by means of the integrated EMG (IEMG).

In chapter 2 an outline of both methods is given. The MFCV is studied in healthy volunteers using both techniques. A clear correlation is found between the MFCV measured by each method. Important advantages of the invasive method are direct muscle fiber activation irrespective of the state of innervation, and complete control of the stimulation frequency. The invasive method gives information about the variability of the MFCV, even in non-innervated muscle fibers. The surface method gives systematically higher MFCV estimates. This is due to a combination of physiological factors and possible measurement errors. This method is especially suited for research purposes. Its main advantages are its non-invasive character, easy repeatability and the capacity to measure at different force levels or during exercise and recovery.

In chapter 3 the MFCV and EMG parameters are studied during one minute of maximal isometric contraction and during recovery afterwards. The MFCV and Fmed both decreased during exercise. This has been attributed to lactate accumulation. The decrease of Fmed is more pronounced, which seems to be the result of additional central factors, such as a decrease of firing frequency during fatigue. During recovery, the MFCV and the Fmed increase, after a subnormal phase, to supernormal levels after 10-12 minutes. The supernormal phase lasted for at least 60 minutes. The increase of MFCV after maximal voluntary contraction was confirmed with the invasive MFCV technique. This excludes central factors as a possible cause. One explanation of the supernormal MFCV could be muscle fiber swelling, in combination with a membrane hyperpolarization due to an increase of sodium pump activity. The corresponding changes in fastest and slowest conducting fibers suggest a comparable effect on type I and II muscle fibers.
In chapter 4 the MFCV and EMG parameters are studied during intermittent isometric exercise at 50% maximal voluntary contraction in healthy males. Measurements were performed during several duty cycles, of 33%, 25% and 20%, and each contraction lasted 2 s. The main finding was a supernormal MFCV during exercise in the duty cycle of 25 and 20%. During the exercise phase of the duty cycle of 33% the MFCV decreased slightly, which suggests that the local anaerobic threshold had been passed. Afterwards, the MFCV increased to supernormal values. It is hypothesized that the changes of electrical properties form part of an adaptive mechanism of the muscle fiber membrane during exercise. In that respect, the increase of the MFCV could be a component of the well-known "warmup" effect.

In chapter 5 the MFCV is studied in biceps brachii in traumatic plexus lesions and amyotrophic lateral sclerosis. After complete denervation an exponential decrease of MFCV is found, T½ 1.1 month. This resulted after 4-5 months in severely reduced conduction velocities. With reinnervation, faster conduction velocities were found, together with fibers which were still conducting slowly. In ALS, a decrease of mean MFCV was found, in combination with an increased range of MFCVs. These findings were interpreted as an effect of a combination of slow-conducting atrophic fibers with fast conducting hypertrophic fibers, which compensate the loss of force. In some ALS patients (predominantly bulbar and distal cases) these disturbances were found without clear abnormalities in biceps brachii muscle by concentric needle EMG, and with unimpaired muscle force. This suggests that subclinical muscle involvement can be detected with this method. The surface EMG measurements in ALS revealed increased MFCV values, in combination with a decrease of Fmed. The increase in MFCV was probably due to hypertrophy of the remaining, voluntarily recruited, MUs. The decrease in Fmed seems to be caused mainly by the change in shape of the motor unit potential.

In chapter 6 the MFCV is studied in a large family known with hypokalemic periodic paralysis (HOPP) by means of both the surface and the invasive determination method. Both methods showed a significantly lower mean MFCV in all proven gene carriers, aside from the presence of paralytic attacks. It is concluded that MFCV determination is a reliable method to detect the membrane defect in HOPP carriers. The invasive method is easy to perform, and gives the highest sensitivity.

In chapter 7 surface EMG and MFCV are studied during attacks of hypokalemic periodic paralysis. After normalization of the serum potassium values, strength rapidly returned to interictal values, but the integrated EMG and, to a lesser degree, the MFCV recovered more slowly. These findings suggest that a complete electrophysiological recovery is not necessary for a restoration of muscle force, and substantiate that the pathogenetic defect in HOPP is localized in the muscle membrane.

In chapter 8 the MFCV is studied during an attack of thyrotoxic periodic paralysis (TPP). It appeared that the MFCV showed (low) normal values during the attack. This argues against a depolarization block as the cause of the paralysis, and suggests a different pathophysiologic mechanism in HOPP and TPP with respect to the paralytic attacks.

In chapter 9 the influence of five days of high-dose methylprednisolone therapy on MFCV and muscle force is investigated in a group of ms patients during a relapse. A significant decrease of MFCV was found by the invasive and the surface determination method. The decrease of MFCV
was not associated with a decrease in force, which argues against muscle atrophy as a major cause. It is hypothesized that partial depolarization accounts for the MFCV decrease, probably in combination with a slight fiber atrophy.

In chapter 10 the MFCV is studied in chronic myositis. Clear disturbances of MFCV were found in all myositis patients. The non-steroid-treated patients showed a slight decrease of mean MFCV, in combination with an increase in scatter of conduction velocities. It appeared that this is related to a combination of muscle fiber atrophy and hypertrophy. In the steroid-treated patients a clear decrease of the mean MFCV is found, with a further increase of scatter of conduction velocities. This seems to be related to muscle fiber atrophy, associated membrane alterations and partial depolarization. Determination of the invasive MFCV could be helpful in showing muscle involvement in possible myositis, and assist in the interpretation of inconsistent findings with concentric needle EMG. It is suggested that the MFCV changes and steroid myopathy are both manifestations of the catabolic effects of steroids on the muscle fiber. However, in (steroid) myopathy a clinical loss of force should be present.

In chapter 11 a literature review is given of the relation between surface EMG and current findings in magnetic resonance spectroscopy (MRS) with respect to the evaluation of muscle fatigue and recovery. Especially in intermediate and high-intensity exercise the changes in the surface EMG parameters, force, and biochemical parameters are closely interrelated. The same holds true with respect to the EMG and MRS (pH and PCr) changes during recovery. Although this does not imply a causal relation, it supports the use of surface EMG in the evaluation of muscle fatigue and recovery.

Additionally the changes in MFCV are interpreted in the time domain. The three main aspects are: (1) Changes of the MFCV immediately after depolarization, (2) short-lasting changes (from seconds to hours), mainly related to fatigue, recovery and exercise, and (for example) to attacks of periodic hypokalemic paralysis, which can be described as abnormal fatigue, (3) long-lasting changes (days, weeks, permanently), during denervation and reinnervation, fiber atrophy and hypertrophy, and because of genetically determined disorders as such HOPP and medication such as steroids.