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Median frequency of the myoelectric signal

Effects of muscle ischemia and cooling

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Summary. A study was performed to investigate the changes that occur in the median frequency of the myoelectric signal during local ischemia or reduction of intramuscular temperature produced by surface cooling. Data was obtained from experiments which involved the first dorsal interosseous muscle of 10 female and 16 male subjects. These subjects were asked to perform isometric constant-force abduction contractions of the index finger at 20% and 80% of maximal voluntary contraction level. The initial median frequency (IMF) of the myoelectric signal during the first 0.5 s of contraction was calculated. Results showed a significant reduction of the IMF in contractions performed under ischemic conditions; upon release, the IMF recovered quickly. At 80% maximal voluntary level of contraction, a greater decrease of the IMF was recorded. Similar results were demonstrated during reduction of intramuscular temperature with gradual recovery of the IMF after cooling. These results demonstrate that the median frequency of the myoelectric signal displays behavior similar to that reported for conduction velocity and this is consistent with the notion that accumulation of metabolic byproducts in muscle tissue causes a decrease in the conduction velocity of the muscle fibers.

Key words: Electromyography – Myoelectric signal – Frequency spectrum – Ischemia – Muscle cooling – Conduction velocity – First dorsal interosseous

Introduction

During a sustained muscle contraction the myoelectric (ME) signal detected on the surface of the skin undergoes a "slowing" of its waveform. The accompanying shift in the frequency spectrum, towards the lower frequencies, has been observed by numerous investigators (Gath and Stalberg 1975; Lindstrom et al. 1977; Stulen and De Luca 1979, and others). Lindstrom (1970), by mathematically modelling the power density spectrum of the ME signal, was able to show that the frequency shift could be explained by a corresponding decrease in the conduction volecity of the action potentials along the muscle fibers. He further noted that the mean frequency of the power density spectrum was directly related to the conduction velocity. More recently, Stulen and De Luca (1981) have shown that the median frequency also provides a reliable, consistent, and relatively unbiased estimate of a parameter of the frequency shift that is related to the muscle fiber conduction velocity. (The median frequency is defined as the frequency value which divides the ME signal power spectrum into two sections of equal energy content.) In general, the estimates of both the mean and median frequencies provide an acceptably good representation of the frequency shift. Both are superior to other parameters, such as the mode frequency and the ratio of high-to-low root-mean square (rms) value (Stulen and De Luca 1981; Hary et al. 1982). However, both have relative advantages and disadvantages, depending on the quality of the ME signal, the shape of the spectrum and other related factors. These two frequency parameters offer the additional advantage that the calculation of their estimate may be implemented in analog circuitry, allowing them to be obtained on-line and in real-time [Broman and Kadefors (1979), and Stulen and De Luca (1979)].

The most convincing associations between shifts in the frequency spectrum of the ME signal and conduction velocity have been through mathematical models. The only direct measurment has been reported as preliminary results by Stulen (1980), who simultaneously measured the conduction velocity and

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median frequency of the ME signal from the muscle fibers of the gastrocnemious muscle of rabbits while electrically stimulating the nerve to the muscle. He noted a simultaneous decrease in both parameters during sustained tetanic stimulation. Considering the lack of available information, it is difficult to postulate all the factors of the casual relationship between conduction velocity and the median frequency of the ME signal. In fact, the contribution of other biochemical events that affect the muscle fiber membrane cannot be eliminated. For example, Jones et al. (1979) have suggested that sodium ion depletion from the extracellular fluid may occur during muscular contraction.

In order to pursue the investigation of a relationship between conduction velocity and median frequency of the ME signal, it was decided to document the behaviour of the median frequency during local ischemia and reduction of intramuscular temperature. The decrease in conduction velocity during repeated electrical stimulation has been reported to be affected by blood flow (Mortimer et al. 1970) as well as by intramuscular temperature (Stalberg 1966), two mechanisms known to be altered in contracting muscles (Petrofsky and Phillips 1981; Kobayashi and Sugi 1980; Hermansen and Osnes 1972; Barnes 1980; Abe 1981).

Methods

A total of 26 normal adult subjects volunteered for this study; 10 were females with an average age of 26 ± 4.2 years; 16 were males with an average age of 27 ± 7.8 years. They all signed an informed consent form. None of the subjects had a past history of neurological disorders. The First Dorsal Interosseous (FDI) muscle was chosen for this study. The experimental arrangement is shown in Fig. 1. In order to record the force output from the FDI muscle a device was constructed to immobilize the subjects' right hands. Such a device also served to isolate the index from the other fingers by means of a rigid support while a pad placed against the proximal interphalangeal joint of the index finger connected it to a force transducer of negligible compliance (0.27 N/μm) (Transducer Model MB25, Interface Inc.). The voluntary contractions of the FDI muscle were monitored as the isometric constant force abduction of the index finger. The subjects were provided with visual feedback to maintain the required level of contraction of the muscle.

The surface ME signal was detected with a dry electrode probe consisting of two silver bars (10 mm long, 1 mm wide) spaced 10 mm apart and fixed on a small plexiglass block. Each of the two silver bars was connected to a Field Effect Transistor embedded in the plexiglass block. The electrode design was similar, but not identical, to that described by De Luca et al. (1979). The differential input resistance of the system was greater than 10^{10} Ω, the input bias current lower than 100 pA and the common mode rejection ratio (CMRR) greater than 75 dB as to guarantee negligible effects of the skin electrical properties and of common mode voltages. The skin was abraded with fine sandpaper and thoroughly cleansed with alcohol. The recording electrode was attached, with tape, to the skin on the belly of the muscle with the bars perpendicular to the direction of the muscle fibers. A saline-soaked strap located around the wrist provided a ground reference. The ME signal was monitored on the oscilloscope during each muscle contraction to ensure absence of noise, 60 Hz interference or motion artifacts. The ME signal was amplified via an optically isolated amplifier and fed to an analog device called the muscle fatigue monitor (Stulen and De Luca 1982). This device is able to calculate the median frequency on line. (The system had a bandwidth with 3-dB points at 30 Hz and 350 Hz.) The output voltage of the device, which was proportional to the median frequency, along with the amplified ME signal and the voltage output of the force gauge, were recorded on an FM tape recorder. Figure 1 presents the details of the experimental arrangement.

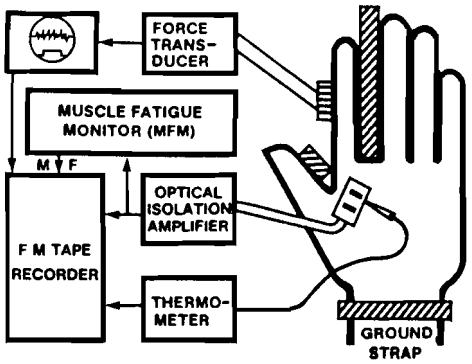

Fig. 1. Experimental arrangement for measuring the median frequency of the myoelectric signal and the intra-muscular temperatue during constant force isometric contraction of the first dorsal interosseous muscle. (See text for further details.) ■ = Mechanical restraints

At the beginning of each experiment, the level of each subject's maximal voluntary contraction (MVC) was obtained by averaging the value of three consecutive attempts to generate maximal contraction. Attempts lasted 4–5 s each and were spaced 2 min apart. The 20% and 80% MVC levels were then computed and appropriate targets were set on the oscilloscope screen. Subjects were asked to match a preset target on the oscilloscope screen as quickly as possible without overshooting it. The Initial Median Frequency (IMF) was measured as the highest value of the median frequency during the initial 0.5 s of each contraction.

Control experiments: Each subject was asked to perform three isometric abductions of the FDI muscle at 20% MVC and spaced 30 s apart. Each contraction lasted 6–8 s. After 2 min, three 80% MVC contractions were performed in a similar fashion, but spaced 1 min apart. These time intervals appeared sufficient to avoid bias of the results by the previous contractions as judged by lack of trend of the IMF values. The average of each of these groups of three contractions was used as the control value for subsequent measurements.

Ischemia experiments: Ten men and 10 women were involved in these experiments. A cuff was placed around the distal part of the forearm and was inflated to 180 mm Hg (24 kPa); after 3, 6 and 10 min the subjects were asked to abduct the index finger isometrically by contracting the FDI muscle to the 20% and 80% MVC level. In a second paradigm the contractions were performed at 1, 2, 3, 5, 7 and 10 min after cuff inflation. In both sets of experiments, the cuff was quickly deflated at the 10-min mark and recordings at both the 20% and 80% MVC were taken at intervals up to 30 min.

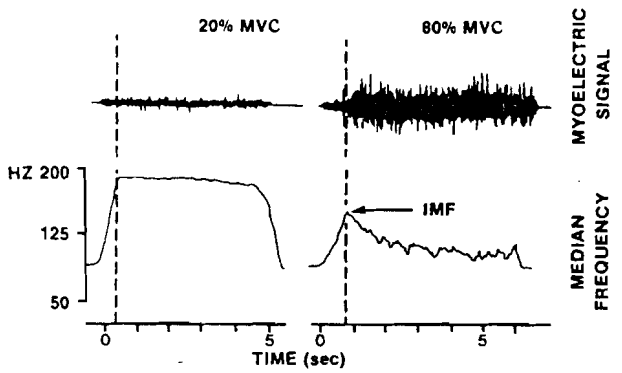

Fig. 2. Sample measurement of the myoelectric signal and of its median frequency detected from the first dorsal interosseous muscle during contractions performed at 20% and 80% of the maximal voluntary level. The diagram shows how the IMF is defined and shows the time course of median frequency at low (20% MVC) and high (80% MVC) levels of contraction

Cooling experiments: Ten men and 10 women were involved in these experiments. A fine (27 gauge) thermocouple needle was inserted into the muscle obliquely at an angle of 30° to a depth of 5 mm. The recording electrode unit was partially removed by lifting one side of the tape and a small plastic bag filled with crushed ice was placed over the muscle for 5 min. The bag was then removed and the electrode unit was replaced exactly in the original position. The subjects were then asked to abduct the index finger at the 20% and 80% MVC level. This was repeated after a further 5 min of application of the ice bag. The bag was finally removed and recordings were made at intervals up to 30 min. Temperature readings were taken continuously during each contraction with a precision of ± 0.25° C.

The percentage changes in the IMF during and after ischemia and cooling were assessed and plotted. Paired *t*-tests were used for measuring the significance of the difference in the observations.

Results

A typical recording of the ME signal and estimated median frequency for a 20% and 80% MVC is presented in Fig. 2. Note that the IMF corresponds to the largest value of the median frequency near the beginning of the contraction. The rise in the median frequency at the beginning of the contraction is due to the delayed response of the muscle fatigue monitor; IMF values were measured after these transients.

Ischemia experiments

The mean time course of the IMF during and after ischemia is shown in Fig. 3. The results were similar for males and females. In each individual, the IMF was normalized with respect to the mean control value for both the 20% and 80% MVC. In every individual case the percentage decrease in the 80% MVC was greater than that of the 20% MVC. After 10 min of ischemia, the difference between IMF decrease at the 80% MVC and at the 20% MVC was statistically significant for females ($P < 0.005$) and males ($P < 0.01$). When the cuff was released, the IMF recovered to the pre-ischemia control value in less than 4 min, with an apparent subsequent overshoot. This overshoot was noted in both males and females, but was more pronounced in males and lasted longer (approximately 40 min) in the 20% MVC (see Fig. 3A, B).

The value of the IMF decreased by a greater amount when more contractions (six rather than three) were performed during the application of the cuff for 10 min (cf Fig. 3A with Fig. 3B). The difference between the IMFs corresponding to the two levels of contractions is smaller (8% compared to 13% at 10 min of ischemia) and is not significant ($P < 0.1$) after 10 min of ischemia (Fig. 3B). When more contractions were performed during the application of the cuff, the recovery phase was slower and the overshoot less marked. It was clear that the IMF of the 80% MVC recovered faster than that of the 20% MVC.

Since contractions were performed in pairs (a 20% MVC contraction followed by an 80% MVC contraction a few seconds later) we assumed that the matabolic millieu of the muscle, obviously effected by the number and level of previous contractions, was approximately the same for each new pair of contractions.

Cooling experiments

Cooling of the FDI muscle caused an immediate and substantial reduction in the IMF of the ME signal in all subjects. The time course of the IMF during and subsequent to the cooling of the muscle is presented in Fig. 4. The IMF continued to decrease as long as the ice pack was located on the skin (up to 10 min) and increased immediately after its removal. The dramatic variation in the reduction of the IMF was the result of the degree of cooling which occurred within the muscle. Figure 5 presents the IMF trend for one male subject. In this typical case it can be seen that the IMF undergoes a greater decrease during the 80% MVC paradigm; this behavior was consistent among all subjects.

The reduction in the IMF obtained by cooling was generally greater than that obtained by ischemia. The recovery of the IMF after the removal of the ice bag was also slower than that following ischemia, not returning to control values in 30 min; this may simply be a demonstration of the slow warming rate of the muscle.

When the IMF is plotted as a function of the internal temperature of the muscle, the relationship

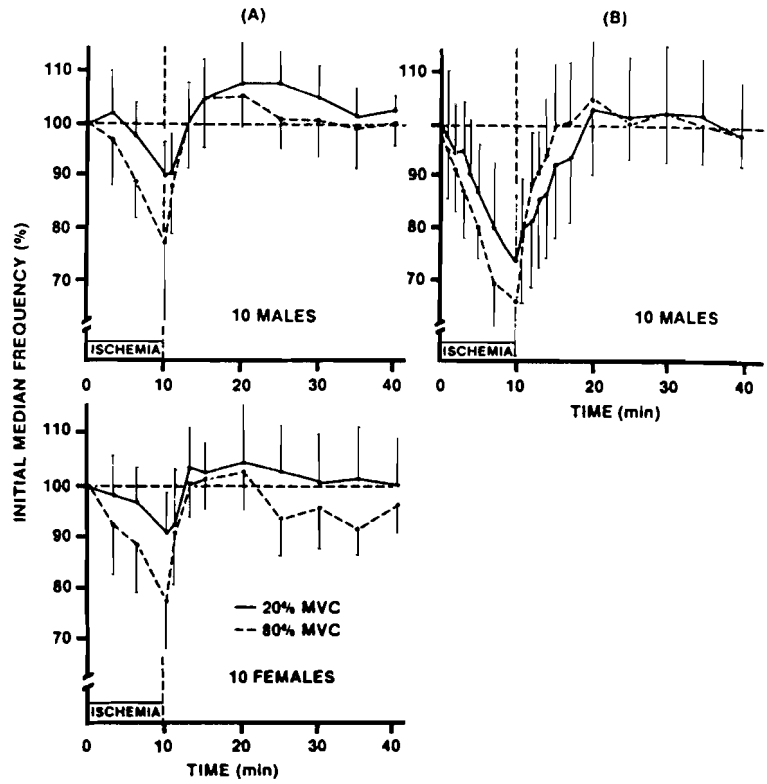

Fig. 3. Behaviour of the initial median frequency of the myoelectric signal during and subsequent to ischemia involving three contractions **A**, and six contractions **B** during 10 min of ischemia. The muscle contractions were performed separately at 20% and 80% of the maximal voluntary level. The diagrams show the average and one standard deviation from 10 females and 10 males. IMF values are normalized with respect to control values taken prior to ischemia for each contraction level

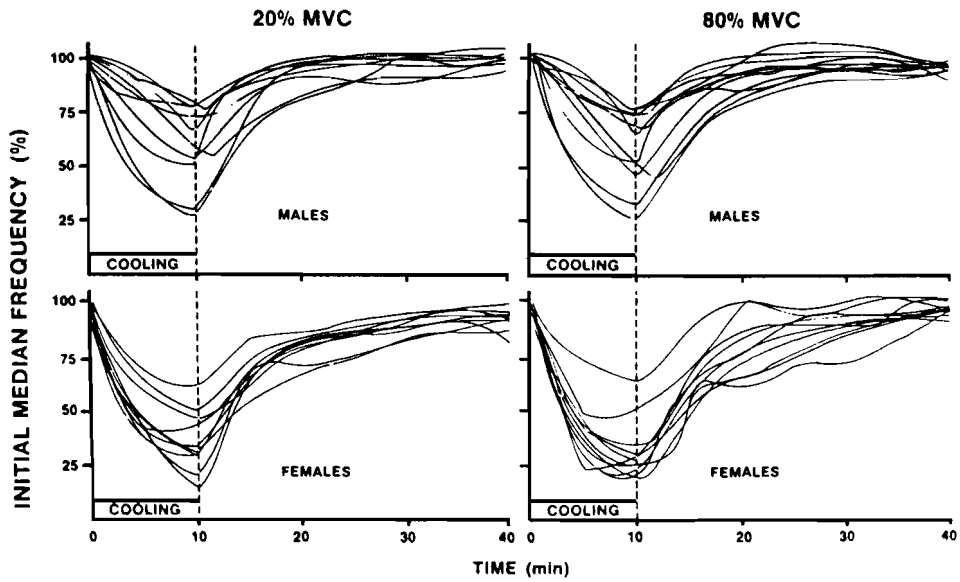

Fig. 4. The initial median frequency of the myoelectric signal as a function of time during and subsequent to cooling of the first dorsal interosseous muscle. Muscle contractions were elicited at 20% and 80% of the maximal voluntary level. IMF values are normalized with respect to control values taken prior to cooling. Each trace represents one subject. Results are from 10 males and 10 females

becomes clear. Figure 6 presents the combined results of all the subjects. Linear regression analysis reveals correlation coefficients of 0.86 and 0.83 for the 20% and 80% MVC. The slopes of the regression lines are 3.03 and 3.48%/°C for the 20% and 80% MVC respectively. Intersubject variations obscure this relationship in the pooled data (Fig. 6). However, when this relationship was calculated for individual subjects, the regression coefficients ranged from 0.991 to 0.998. These results demonstrate the presence of a highly linear and rigid relationship between the IMF and the muscle temperature.

Discussion

It has been suggested by Naeije and Zorn (1982) that ME spectral shift in the biceps brachii during localized muscle fatigue may occur without a con-

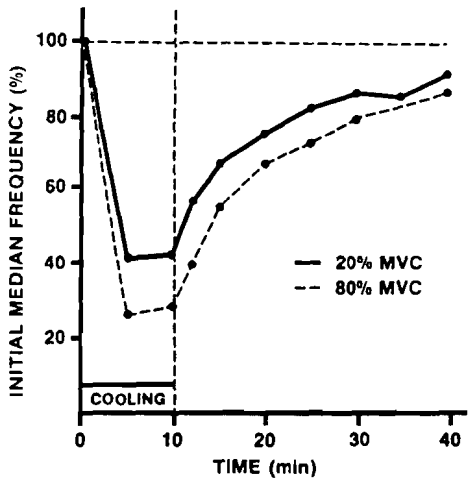

Fig. 5. The values of the initial median frequency of the myoelectic signal detected at two different force levels from one subject during and subsequent to cooling of the first dorsal interosseous muscle. Note that with the 80% maximal voluntary contractions (MVC) the initial median frequency undergoes a greater decrease than during the 20% MVC. This was observed in every subject. IMF values are normalized with respect to control values taken prior to cooling

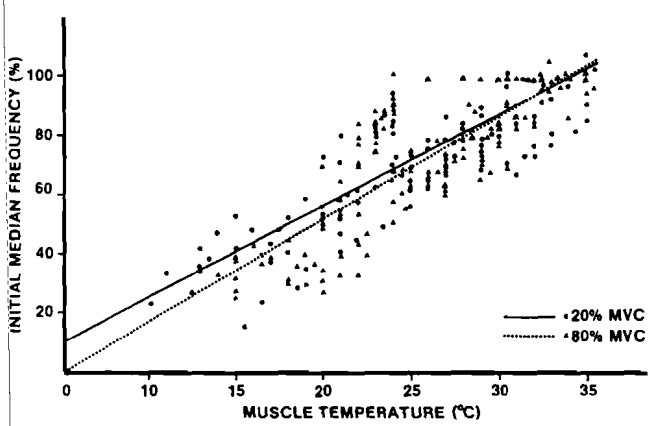

Fig. 6. A cumulative graph of the percentage changes in the initial median frequency of the myoelectric signal during 20% and 80% MVC contraction as function of muscle temperature in 10 males and 10 females. Regression lines for the 80% MVC and 20% MVC data illustrate that at 80% MVC, the initial median frequency undergoes a greater decrease than at 20% MVC during reduction in muscle temperature. The difference between the two slopes is statistically significant in individual cases

commitant change in muscle fiber action potential conduction velocity as measured with the cross-correlation technique. However, this technique is not consistently reliable, especially for low cross-correlation values and it may be affected by the different impedances of the paths between the signal source and the electrode locations on the skin. Our results indicate that in the FDI muscle, the behaviour of the ME signal median frequency is similar to that reported for conduction velocity by many authors in previous papers.

No consistent relationship was noted between the IMF and the level of contraction performed by the FDI muscle using our experimental set up. Figure 2 shows a representative result. These findings support our previous results (Sabbahi et al. 1979, 1981) and those of others (Kaiser and Petersen 1965; Ericson and Hagberg 1979; Petrofsky and Lind 1980b). This lack of consistency can be explained by noting that the power density spectrum is for the most part a transformation of the shapes of the motor unit action potentials which constitute the ME signal. (For specific details, refer to De Luca and Van Dyk [1975] and LeFever and De Luca [1976].) The motor unit action potential shapes are affected mainly by the diameter of the muscle fibers forming the motor unit, and by the location of the recording electrodes with respect to the active fibers (refer to De Luca 1979). The muscle fiber diameter is directly related to the conduction velocity which is in turn directly related to the median frequency of the power density spectrum (Stulen and De Luca 1981). The location of the recruited muscle fibers with respect to the recording electrodes modifies the value of the IMF of the ME signal due to the low-pass filtering effect of the muscle tissue. Hence, as the force level of the muscle contraction increases, the IMF of the ME signals may not display a consistent change, depending on the combined relative effect of the two factors.

The decrease of the IMF during ischemia presented in Fig. 3 is consistent with the concept that accumulation of the metabolic byproducts decreases the conduction velocity of muscle fibers (Mortimer et al. 1970; Stalberg 1966), and subsequently the median frequency of the ME signal (Stulen and De Luca 1981). Mortimer et al. (1970), working with cats, also noted that the decrease in conduction velocity was more pronounced in the gastrocnemius than in the soleus muscle, which contains fewer fast-twitch fibers and presumably produces fewer acidic byproducts during muscular contractions. The greater decrease noted during the 80% MVC supports this concept in that more acidic byproducts are formed during 80% MVC (when proportionally more fast-twitch fibers are activated) than during 20% MVC (Tesch and Karlsson 1977). Fig. 3B provides additional support by demonstrating that when the number of contractions performed during the ischemic period is doubled, the decrease in the IMF is even more pronounced. Karlsson et al. (1975) have shown that lactate production is not monotonically correlated with contraction level; however, they compared contractions of different durations (sustained to exhaustion). Our data show that accumulation of metabolites or other chemical changes during ischem-

ia and cooling reduce the median frequency of the ME signal in a fashion consistent with that reported by many authors for conduction velocity. Although factors such as hypoxia or temperature changes during ischemia could not *"a priori"* be excluded, it seems that hypoxia could not explain the different results obtained with paradigm A and B in Fig. 3.

The possibility that the reduction of the IMF might be caused by a reduction in the intramuscular temperature resulting from the diminution of local blood flow was tested by measuring the intramuscular temperature during ischemia. The intramuscular temperature decreased by less than 1° C during the 10-min period of ischemia. (The subsequent discussion on cooling will indicate that the minor decrease in temperature associated with ischemia cannot cause the noted decrease in the IMF.)

The recovery of the IMF post-ischemia was different for the two paradigms. In the one with the most frequent contractions (Fig. 3B), the relatively earlier recovery of the IMF of the 80% MVC is most likely caused by the faster wash-out of the metabolic byproducts due to the greater pumping action of the stronger contractions. But it should be remembered that the 80% MVC involves more anaerobic fast-twitch fibers, which in the FDI have larger diameters (Polgar et al. 1973). These larger diameter muscle fibers will have a greater conduction velocity, hence, the initial median frequency will be greater. This latter property is apparently not exploited or evident during the faster-occurring recovery phase of the paradigm in Fig. 3A. The observed behavior of the IMF in the recovery phase is consistent with that observed by Broman (1973) for conduction velocity and with that observed by Harris et al. (1981) for lactic acid removal after localized fatigue.

The overshoot in the value of the IMF seen in the paradigm of Fig. 3 may be explained by the muscle temperature increase caused by the renewed blood flow post-ischemia accompanied by vasodilation which allows for the removal of the metabolic byproducts at a faster rate than normal. (In one subject an intramuscular temperature increase of 0.5° C above the control level was measured during the overshoot phase.) The lack of overshoot in the paradigm of Fig. 3B indicates that the temperature increase and vasodilation dissipated during the longer (10 min) recovery phase and hence did not cause the IMF to overshoot. It is interesting to note that an overshoot in the value of the conduction velocity of the muscle fibers was seen in some of the experiments performed by Mortimer et al. (1970) in cats, and by Stalberg (1966) in humans. This coincidence of observations provides additional support to the existence of a direct relationship between the conduction velocity and median frequency (Stulen and De Luca 1981).

The decrease in the IMF with reduction of intramuscular temperature is also indicative of a relationship between the conduction velocity of the muscle fibers and the median frequency of the ME signal. A greater reduction in IMF was obtained by cooling, but like the ischemia measurements, no distinction could be made between the results of the males and females (Fig. 4). The substantial variability in the decrease of the IMF among such subjects was caused by the variable degree to which the muscle in each subject was cooled. In fact, when the decrease in the IMF was plotted as a function of intramuscular temperature, the relationship was linear ($r = 0.85$). Our observations are compatible with those of Petrofsky and Lind (1980b), who also found a direct relationship between the median frequency of the ME signal and the decreased intramuscular temperature.

In the present study, the linear relationship between the IMF of the ME signal and the intramuscular temperature within the range of 15–33° C was found to have a sensitivity (slope) of 3.03%/°C during 20% MVC, and 3.48%/°C during 80% MVC. Several examples may be found in the literature to indicate that a similar behavior exists between conduction velocity and temperature. Four decades ago, Wilska and Varjoranta (1940) found the conduction velocity of abdominal muscles of the frog to be linearly correlated to temperature. Hakansson (1956), also working with frog muscles, found a slope of 0.15 m/s/°C from 15–20° C; the sensitivity was 4.6%/°C. Jarcho et al. (1954), working with the anterior gracilis muscle of the rat, reported a value of 4.3 m/s and a sensitivity of 4–5%/°C in the range of 30–33° C. Buchthal and Engbaek (1963) also found a linear relationship in the frog sartorious muscle with a sensitivity of 2.8%/°C. Morimoto et al. (1980), while working with the human vastus medialis, found values of 3.3–3.7 m/s/°C in the range of 17–31° C; the sensitivity was 4.7%/°C. All these measurements of conduction velocity sensitivity are comparable to the sensitivity values of the IMF and temperature obtained in this study.

It remains to be pointed out that the IMF demonstrated a greater decrease while the muscle was being cooled during the 80% MVC than during the 20% MVC. Again, as in the case of ischemia, the formation and presence of additional metabolic byproducts in the muscle might explain this observation, although other factors may play important roles.

In conclusion, during ischemia and cooling, the median frequency of the myoelectric signal detected from the First Dorsal Interosseous muscle during

constant-force isometric contractions displays a behavior which is similar to that which has been reported for the conduction velocity of the muscle fibers. (In both cases, the median frequency decreased.) The results of the ischemia experiments are consistent with the notion that the accumulation of acidic byproducts in the muscle tissue causes a decrease in the conduction velocity of the muscle fibers.

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