Specificity of power improvements through slow and fast isokinetic training

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COYLE, E. F., D. C. FEIRING, T. C. ROTKIS, R. W. COTE III, F. B. ROBY, W. LEE, AND J. H. WILMORE. *Specificity of power improvements through slow and fast isokinetic training.* J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 51(6): 1437–1442, 1981.—College age males performed maximal two-legged isokinetic knee extensions three times per week for 6 wk at either 60°/s (slow) or 300°/s (fast) or both 60 and 300°/s (mixed). The velocity specific and action specific (two-leg vs. one leg) improvements in peak torque (PT) were compared to a placebo group receiving low-level muscle stimulation. The slow group improved PT significantly ($P < 0.05$) more than the placebo group only at its training velocity (60°/s) and more so when the specific two-legged training action was mimicked (+32% with two legs vs. +19% with one leg). The mixed group enhanced PT by 24 and 16% at their respective training velocities of 60 and 300°/s. These improvements were significantly larger than placebo and also significantly larger than the 9% improvement observed at the midvelocity of 180°/s. The training specificity demonstrated by the slow and mixed groups suggest that neural mechanisms contributed to their improvements in power. This is supported by their unchanging muscle morphology. Training solely at 300°/s (fast) however improved PT significantly more than placebo not only at the training velocity (+18%), but also at a slower velocity of 180°/s (+17%). The fast group demonstrated a significant enlargement (+11%) of type II muscle fibers. These data suggest type II fiber hypertrophy to be a plausible mechanism for the nonspecific improvement of the fast group; however, a neurological adaptation that enhances power at and below the training velocity cannot be excluded.

muscular strength; hypertrophy; neuromuscular; placebo

OUR PRESENT KNOWLEDGE of the physiological adaptations of the neuromuscular system to physical training is based predominantly on investigations that measured muscular force output at slow contraction velocities subsequent to slow training (6). It has been found that the muscular force developed during slow contractions is most effectively improved through training that demands near maximal tension development (2) and that this enhanced force generation is specific to the action and mode of training (3). The mechanisms (e.g., muscular and neurological adaptations, psychological factors) by which high-tension, slow-velocity training augments strength are unclear, and it is uncertain whether this slow-velocity training improves force development during fast contractions.

Few observations have been made regarding the velocity-specific training effects derived from fast contractions. Because the stimuli elicited by maximally performed slow and fast contractions differ (8, 9, 19), i.e., peak tension and duration, it can be hypothesized that the physiological and specific force-velocity adaptations might also differ.

The purpose of this study was to compare the training-induced changes in maximal quadriceps torque at specific isokinetic velocities in groups of individuals performing only slow, only fast, or a mixture of slow and fast maximal contractions. Because psychological factors can enhance performance (11), all improvements were compared to those exhibited by a placebo group to differentiate between those improvements that were of physiological and those of psychological origin. Our goals were to determine *a*) whether the performance improvements were related to muscle hypertrophy and *b*) whether they were specific to the action and velocity of knee extension training.

METHODS

Subjects. Twenty-two physically active college-aged males who had not been involved in any formal training during the previous 2 y, volunteered to participate in this study (Table 1). Following a detailed explanation of the purpose of, potential benefits from, and risks associated with participation in this study, these individuals provided written consent. The subjects were randomly assigned to five groups.

Training groups. Five individuals maintained their normal activity throughout the training period and thus served as a control group. Another five individuals were informed that they were selected to test the effectiveness of a new training method, faradic muscle stimulation. The subjects were informed that this treatment was known to be effective and this investigation would be aimed at quantifying their improvements. The faradic stimulator generates a high-frequency current that results in mild skin anesthesia. Large electrodes (4-in.) were placed over the distal vastus medialis and proximal vastus lateralis of both legs. Approximately 20 mA of current were passed through the quadriceps, resulting in mild skin anesthesia and a muscular contraction that produced less than 3% of the tension developed during a maximal voluntary contraction (MVC) or during maxi-

TABLE 1. *Physical characteristics of subjects pre- and posttraining*

Group	Age, yr	Ht, cm	Wt, kg		Midthigh Circumference, cm		Midthigh Skinfold, mm	
			Pre	Post	Pre	Post	Pre	Post
Control (5)	26.9 ± 3.1	180.2 ± 4.9	76.3 ± 4.7	78.4 ± 4.9	52.9 ± 2.9	53.9 ± 3.1	8.8 ± 2.9	9.9 ± 3.2
Placebo (5)	21.4 ± 3.0	180.2 ± 5.8	80.0 ± 4.2	80.3 ± 4.0	52.6 ± 1.9	53.1 ± 2.8	11.2 ± 1.6	10.9 ± 1.9
Slow (4)	25.1 ± 2.3	177.7 ± 5.6	74.0 ± 6.5	74.3 ± 6.5	51.4 ± 3.0	51.6 ± 3.1	12.1 ± 2.6	9.2 ± 1.6*
Fast (4)	25.0 ± 1.9	166.2 ± 8.1	64.2 ± 3.2	63.8 ± 3.8	49.2 ± 3.1	50.0 ± 1.9	11.3 ± 2.2	10.7 ± 2.9
Mixed (4)	22.3 ± 1.6	178.7 ± 4.3	79.2 ± 3.0	78.4 ± 3.0	53.1 ± 0.7	53.5 ± 0.10	10.6 ± 0.8	10.4 ± 0.9

Values are means ± SE; numbers in parentheses are numbers of subjects. * Significant difference ($P < 0.05$).

mal stimulation. This treatment was chosen as a placebo treatment because it produced a distinct sensation although it is unlikely that it provided any physiologically significant stimulus to the muscle because it has been shown that contractions greater that 40% MVC are required for this purpose (6). It is assumed that this afferent sensation did not result in chronic adaptations within the motor neurons that would augment power. The placebo subjects were treated identically to the other experimental subjects with regard to contact time, group meetings, length of treatment, and rest periods. They received five sets of stimulation, each lasting 10 s, while resting 8 min between bouts. This was performed three times per week on alternate days for a total of 6 wk.

The three remaining groups of subjects trained using a Cybex Orthotron (Cybex/Div. of Lumex, Bayshore, NY) modified to allow for two-legged knee extension. These groups performed the same amount of work during the training sessions and differed only in the velocity at which it was performed. Pilot work on 20 individuals determined that nearly twice (1.95 times) as much work (torque × angular distance, including the leg) is performed during a maximal knee extension at an isokinetic velocity of 60°/s as compared to 300°/s. One group of individuals performed five sets of six maximal two-legged knee extensions at 60°/s (slow, $n = 4$). Another group performed an equal amount of work (approx 10,000 J) at a velocity of 300°/s by executing five sets of 12 maximal two-legged knee extensions (fast, $n = 4$). A third group (mixed, $n = 4$) performed half their work at 60°/s (2 or 3 sets; 6 reps/set) and half at 300°/s (2 or 3 sets; 12 reps/set). The mixed group was included to determine whether there was a greater improvement in neuromuscular performance when both slow and fast contractions were incorporated into a training regimen. These groups met on Monday, Wednesday, and Friday for 6 wk. The subjects were instructed to perform each extension maximally. Flexion occurred passively, and the subjects came to a full stop before setting themselves and bursting into the next repetition. Each contraction was timed using a clock (±0.001 s) triggered by two microswitches, to make certain the velocities were accurate. Verbal feedback of the torque generated during each contraction was provided. There were 8 min of rest between sets. All sessions were supervised.

Testing equipment and protocol. A Cybex II isokinetic system was used to evaluate knee extension power pre- and posttraining. This system was modified by extending the cross bar and supporting it with an additional bearing mounted at the far side of the bench on which the subject was seated. It therefore became possible to measure the torque developed with two legs extending simultaneously, as well as with each leg independently. A comparison of improvements during the training mode of two legs simultaneously, as opposed to each leg independently, will provide insight into whether the adaptations are primarily muscular or neurological. For example, if the adaptations were solely muscular, then the percent improvement should be equal during one- and two-legged testing because the individual muscle itself is performing the exact same action. If the adaptations were neurological, involving enhanced motor unit recruitment or synchronization, it was theorized that the two-legged pattern specifically encountered during training would demonstrate a greater percent improvement than one-legged extension.

To ensure a stable system, the dynamometer head was mounted on a concrete wall, the bench was bolted to the floor, and the subject was secured to the bench with a belt across the hips to minimize extraneous movement. The pivot point of the knee was aligned with the rotational axis of the resistance arm. Both the undamped and damped (Cybex II recorder damp setting of 2) torque signals were displayed on a Gilson 5-channel polygraph recorder as was the goniometric tracing of joint angle. This permitted measurement of both peak torque (damped signal of 2 created by a 68K resistor in parallel with a 2.7 μF capacitor) and the undamped torque generated 30° prior to complete extension as described by Perrine and Edgerton (17). This damping generates a smooth readable torque tracing by averaging torque oscillations within a period of approximately 50 ms. Although the peak damped and undamped (30° prior to extension) torques differ in absolute magnitude, they were found to change in the same proportion as a result of the training. Therefore, only the peak torques (PT) generated at the various velocities are reported because it was found to be a more reliable measure ($r = 0.96$), determined from test-retest PT measures on alternate days of pretesting. The system was calibrated before each test by applying a known torque to the resistance arm, and it was found to be accurate within ±4 N·m, which represents ±2% over the ranges tested.

All testing was performed with the subject in the same position as during that of training. The only difference was that isokinetic resistance was provided by the Cybex II during testing, whereas isokinetic resistance was provided by Cybex Orthotron during training. Pre- and posttesting followed identical protocols and occurred at the same time of day. The subjects reported to the laboratory and sat quietly for several minutes prior to testing. The peak torque that could be produced at knee extension velocities of 60°/s (PT/60), 180°/s (PT/180), 300°/s (PT/300), and isometrically at a joint angle of 65° (PT/0) were determined in that order, on both legs simultaneously and on each leg individually. Test-retest measures were performed on separate days both pre- and posttraining, with the order of one- and two-legged testing being rotated.

The subject performed two extensions at each test velocity to become acquainted with the velocity. Subsequently he was verbally encouraged by the investigator to exert maximal effort through 90° of knee extension or 4 s isometrically. Two separate efforts were made routinely, and a third extension was performed if more than a 3% difference existed. Highest scores are reported.

Two weeks prior to the pretest, the subjects participated in two familiarization periods during which they practiced the pretest. They were allowed to experiment with techniques (i.e., shouting, Valsalva, etc.) that might enhance performance.

Anthropometry and histochemistry. Midthigh circumference and skinfold were determined as described by Behnke and Wilmore (1). Muscle biopsies (4) of the vastus lateralis were obtained approximately 24 h after the pre- and posttesting. Samples were oriented under a dissecting microscope, mounted in an imbedding matrix (OCT) and quickly frozen in isopentane cooled with liquid nitrogen. An individual's pre- and postsamples were strained during the same analysis. Sections of 10 μm were stained for myofibrillar adenosine triphosphatase after adjusting the preincubation pH to identify the percentage of type I, type IIa, and type IIb muscle fibers (5). The areas of 50 individual type I and type II fibers were determined from muscle sections stained for $NADH_2$ tetrazolium reductase (15). Individual fibers were randomly selected and projected onto a large screen on which their area was traced onto paper and subsequently compared by weight to a known area. Pilot work estimated the coefficient of variation (SD/mean × 100) of this method from test-retest to be ± 3%.

Statistical analysis. Changes in PT, muscle morphology, and anthropometry consequent to the training were subjected to a one-way analysis of variance or covariance (for prevalues) to determine whether a significant difference between group means existed, and Duncan's procedures for unequal sample sizes identified the means that differed. From this ANOVA the pooled variance within groups was established $[(\text{MSE within}/n)^{1/2}]$ Changes within a group from pre- to posttraining were tested through Student's t ratios, $[t = (\bar{x}\text{ post} - \bar{x}\text{ pre})/(\text{MSE within}/n)^{1/2}]$. All comparisons were made with $P < 0.05$.

RESULTS

The groups were not significantly different (mean ± SE) in age or muscle fiber composition (Tables 1 and 4). The fast group was slightly shorter and lighter, but their pretraining muscle fiber area-to-body weight ratio was very similiar to that of the other groups (Table 5). Adherence to the training program was remarkable in that none of the subjects missed a scheduled workout and only 5% of the 302 man sessions had to be rescheduled for a time other than when the individual groups met. The placebo group was convinced that the muscle stimulation was effective as evidenced by their unsolicited comments to individuals not associated with the investigation that they "felt stronger."

Pre-post PT changes within groups. As noted in Table 2, all groups decreased PT with increasing velocity. In the control group, PT did not vary by more than 2% at any velocity pre- to posttraining. The placebo group demonstrated a significant improvement of 8% in PT/0, a nonsignificant increase of 3–5% in PT/60 and PT/180, and no change in PT/300. The slow group showed a significant improvement of 20, 32, and 9% in PT at 0, 60, and 180°/s, respectively; yet, PT/300 was unchanged (+0.9%). The fast group had a +15 to +24% improvement at all velocities tested. The mixed group also improved significantly at all velocities; however, the 8% improvement in PT/180, although significant, was not as large as the increase observed at the specific velocities of training (60°/s, +23.6%; 300°/s, +16.1%). Mixed therefore improved PT significantly ($P < 0.05$) more at 60°/s and

TABLE 2. *Peak torque generation pre- and posttraining and percent change at various velocities during two-legged extension*

	PT/0			PT/60			PT/180			PT/300		
	Pre, N·m/kg	Post, N·m/kg	%Δ†	Pre, N·m/kg	Post, N·m/kg	%Δ	Pre, N·m/kg	Post, N·m/kg	%Δ	Pre, N·m/kg	Post, N·m/kg	%Δ
Control (5)	7.41	7.34	−1.5	5.52	5.39	−2.3	3.15	3.13	−0.7	1.96	1.93	−1.5
	±0.32	±0.24	±4.6	±0.20	±0.19	±1.7	±0.14	±0.35	±0.9	±0.11	±0.15	±1.7
Placebo (5)	7.59	8.15	+8.1*	6.05	6.25	+3.2	3.60	3.78	+5.0	2.24	2.25	+0.3
	±0.48	±0.34	±6.3	±0.31	±0.26	±1.5	±0.18	±0.13	±3.1	±0.08	±0.07	±2.8
Slow (4)	7.09	8.72	+20.3*	5.52	7.27	+31.8*	3.59	3.92	+9.2*	2.31	2.32	+0.9
	±0.14	±0.28	±6.1	±0.33	±0.38	±5.7	±0.25	±0.24	±2.6	±0.13	±0.14	±4.5
Fast (4)	8.22	9.83	+23.6*	5.60	6.45	+15.1*	3.35	3.91	+16.8*	2.03	2.40	+18.5*
	±0.77	±0.40	±7.9	±0.30	±0.26	±6.3	±0.13	±0.22	±3.2	±0.14	±0.14	±6.7
Mixed (4)	6.50	8.20	+18.9*	5.22	6.45	+23.6*	3.43	3.70	+7.9*	1.99	2.31	+16.1*
	±0.53	±0.77	±7.0	±0.34	±0.46	±6.0	±0.11	±0.13	±2.5	±0.07	±0.02	±4.0

Values are means ± SE; numbers in parentheses are numbers of subjects. PT/0 through PT/300, peak torque at knee extension velocities of 0°, 60°, 180°, and 300°/s; %Δ, percent change at the various velocities. * Denotes significant improvement pre- to posttraining ($P < 0.05$). † Denotes %Δ covaried for pre-PT/0.

300°/s than at 180°/sec. As noted in Table 2, improvement (%Δ) in PT/0 was the only measurement significantly related to its initial value. That is, weaker individuals tended to demonstrate the largest improvements in PT/0, and thus percent improvements were covaried for initial values. Individual differences in rate of strength gain could not be assessed and controlled for because frequent measurements would provide a training stimulus.

Two-legged improvements in PT compared to one-legged. The mean torque generated during two-legged extension was 96.9, 95.8, 97.5, and 97.2% of the sum of the individual right and left legs at velocities of 0, 60, 180, and 300°/s, respectively. This was derived during pretesting using the entire sample population. Although all training was performed with two legs simultaneously, the percent improvements in two-legged PT were within 5% of the improvement in one-legged PT and not significantly different. This was true at all velocities and in all groups, with one exception. The slow group's 32% increase in two-legged PT at its training velocity of 60°/s was significantly greater than the 19% improvement in PT obtained with one leg.

Comparison of PT changes among groups. Table 3 compares the various groups as to their mean percent change in PT at the specific velocities tested. Groups that do not differ (Duncan's procedure, $P < 0.05$) are indicated by their enclosure within the same lower bracket. Comparisons to the placebo group are of particular interest.

None of the training regimens improved PT/0 significantly more than did placebo (Table 3). The slow group improved significantly more than placebo only at its training velocity of 60°/s and significantly more so when tested with two legs simultaneously as compared to one leg individually. The fast group displayed significantly larger improvements than the placebo group at their training velocity of 300°/s and at 180°/s. Improvements greater than placebo in the mixed group were limited to its specific training velocities, i.e., 60 and 300°/s.

TABLE 3. *Comparison of mean percent change between groups*

PT/0	Fast +23.6	Slow +20.3	Mixed +18.9	Placebo +8.1	Control −1.5
PT/60	$\boxed{\text{Slow } +31.8}$	$\boxed{\text{Mixed } +23.6}$	Fast +15.1	Placebo +3.2	Control −2.3
PT/180	$\boxed{\text{Fast } +16.8}$	Slow +9.2	Mixed +7.9	Placebo +5.0	Control −0.7
PT/300	$\boxed{\text{Fast } +18.5}$	$\boxed{\text{Mixed } +16.1}$	Slow +0.9	Placebo +0.3	Control −1.5

The lower brackets enclose groups with mean percent changes that do not differ as determined by Duncan's procedure, $P < 0.05$. The boxed groups are those that improved significantly more than placebo. PT/0 through PT/300, mean peak torque at knee extension velocities of 0°, 60°, 80°, and 300°/s.

TABLE 4. *Muscle fiber composition, pre- and posttraining*

	% Type I		% Type IIa		% Type IIb	
	Pre	Post	Pre	Post	Pre	Post
Control (3)	41.3 ±10.1	43.7 ±10.1	42.5 ±8.0	41.1 ±7.0	16.3 ±3.9	15.6 ±3.7
Placebo (3)	37.5 ±1.6	35.4 ±1.7	39.1 ±1.4	38.5 ±1.3	22.4 ±0.8	26.1 ±1.7
Slow (3)	32.9 ±3.7	26.0 ±7.1	48.1 ±5.9	52.4 ±1.4	18.8 ±2.5	21.6 ±6.4
Fast (3)	36.8 ±3.5	42.8 ±1.5	48.9 ±1.5	41.5 ±0.8	14.3 ±2.2	15.8 ±2.3
Mixed (4)	40.6 ±4.9	43.7 ±5.2	41.5 ±3.2	41.9 ±3.8	18.0 ±4.5	14.3 ±4.4

Values are means ± SE; numbers in parentheses are numbers of subjects. * Denotes significant pre- and posttraining ($P < 0.05$).

Morphological adaptations. No significant changes in body weight or midthigh circumference were observed (Table 1). Midthigh skinfold was significantly reduced (−2.9 mm) in the slow group, whereas the other groups demonstrated small, nonsignificant changes. Six of the 44 muscle sections prepared for histochemical analysis displayed sufficient artifact (noncross-sectional fibers, too few fibers, <250) to necessitate their elimination from group comparisons, and therefore the total sample population for pre-post comparisons was reduced to 16 as indicated in Tables 4 and 5.

The mean (±SE) number of fibers obtained per biopsy was 802 (±180). The subjects were fairly homogeneous in their percent distribution of muscle fiber types (Table 4). There were no significant changes in muscle fiber composition (Table 5). The mean area of type I muscle fibers was not significantly altered in any group. The fast group displayed a significant increase (±11.2%) in the mean area of type II fibers. Type II fiber area did not change significantly in the other groups.

DISCUSSION

The physiological adaptations responsible for training-induced improvements in neuromuscular power are generally believed to occur within the trained muscle and/or the nervous system (6, 8, 9, 11, 12, 14). Potentially, the power-generating contractile mass of a muscle can be increased through the hypertrophy of single muscle fibers (9) or by hyperplasia, which has been observed in cats (10) and has been theorized to occur in humans. Neural factors control muscular tension by affecting the number of motor units recruited as well as their synchronization. It is technically difficult to determine the extent to which these mechanisms contribute to the training-induced improvements in neuromuscular power. In this study we described the velocity specific and action specific nature of these improvements to provide a data base on which mechanistic theories can be formulated and further explored.

It was found that the group that performed slow, high-tension contractions improved performance more than

TABLE 5. *Muscle fiber area, pre- and posttraining*

	Type I Area, μm^2			Type II Area, μm^2			Type I Area/Body Wt, $\mu m^2/kg$		Type II Area/Body Wt, $\mu m^2/kg$	
	Pre	Post	%Δ	Pre	Post	%Δ	Pre	Post	Pre	Post
Control (3)	7,388	6,821	−8.7	7,602	7,420	−2.6	96.8	87.0	99.6	94.6
	±393	±484	±4.0	±430	±602	±2.6				
Placebo (3)	7,012	6,777	−5.0	7,501	7,401	1.4	87.6	84.4	93.8	92.2
	±266	±546	±10.2	±151	±320	±2.3				
Slow (3)	6,508	6,432	0.4	7,186	7,614	+5.1	87.9	86.6	97.1	102.5
	±1,085	±501	±11.1	±637	±1,045	±5.8				
Fast (3)	5,887	6,187	+5.1	6,180	6,941	+11.2*	91.7	96.8	96.3	108.8*
	±842	±735	±7.9	±419	±1,023	±8.7				
Mixed (4)	7,431	7,041	−5.1	8,178	7,955	−2.5	93.8	89.8	103.2	101.5
	±637	±450	±3.0	±843	±799	±3.8				

Values are means ± SE; numbers in parentheses are numbers of subjects. * Denotes significant pre- and posttraining ($P < 0.05$).

the placebo group at their training velocity and in the absence of changes in cross-sectional girth and without muscle fiber hypertrophy. Also, the percent improvement with the training action of two legs extending simultaneously was 13% greater (32 vs. 19%) than that observed when the identical musculature and action were evaluated individually. This high degree of specificity and the inability to detect changes in muscle morphology suggest that neurological factors contributed significantly to the augmented torque. Previous investigations have observed training specificity, with improvements being most evident in the training mode (isotonic vs. isometric) and at the joint angle used for isometric training (3, 6, 18).

A possible neurological basis for this specificity of high-tension training may involve an ability to recruit more motor units during the activity experienced in training. Komi et al. (12) have observed, that maximal integrated electromyogram (IEMG) and isometric force increased, 38 and 20%, respectively, in the isometrically trained legs of adolescents, whereas IEMG remained unchanged in the contralateral leg. Another adaptation observed by Komi et al. (12) is a more economical usage of the motor units recruited so that a given number of motor units are more efficiently summated, resulting in a higher force output posttraining. Milner-Brown et al. (14) have found posttraining isometric contractions to have electromyogram (EMG) patterns that are temporally more synchronous. Although it is beyond the scope of the present investigation to identify the specific mechanisms underlying the improvement of the slow group, our data indicate that the adaptations do not result in an increased neuromuscular output during fast contractions.

The stimuli derived from performing maximal fast contractions are different from those produced by slow contractions. Peak tension is reduced 60% and the action is so rapid (300 ms to completion) that the recruitment pattern may also differ (19). Our data indicate that the improvements of the fast group in neuromuscular output were not as specific as those of the slow group. The fast group significantly improved PT at all speeds and demonstrated equal improvements during one- and two-legged extensions. An increase in contractile mass would theoretically result in this type of general increase in muscular power. Interestingly, the fast group demonstrated a significant enlargement (+11%) of type II fibers. These data suggest that type II fiber hypertrophy is a plausible mechanism for the improvement of the fast group; however, neurological adaptations that enhance neuromuscular output at and below the training velocity cannot be excluded.

A carry-over of high-velocity training to performance at slower velocities was previously observed by Lesmes et al. (13) and was also associated with a 7% increase in the area occupied by type II fibers as reported by Costill et al. (7) in the same subjects. These subjects trained their knee extensors isokinetically at 180°/s, yet, PT/300 was unchanged (13). If the improvements noted by Lesmes et al. (13) were solely a result of the increased type II area (7), PT/300 would also be expected to increase. The finding that it did not suggest again that a neurological factor may be operative. The mixed group demonstrated remarkable specificity, possibly neurally mediated because PT/180 was improved only 7.9% compared to the 23.6 and 16.1% enhancements in PT at the training velocities of 60 and 300°/s, respectively.

Psychological factors can improve muscular strength. Ikai and Steinhaus (11) have found that the hypnotic suggestion of improved strength acutely increases maximal isometric tension by 26%, presumably by removing neurological inhibitions. The present investigation attempted to control psychological factors, first, by teaching the subjects to "disinhibit" themselves to optimize muscular power during pretesting, and, second, by including a placebo treatment that would identify the portion of improved power that can be attributed to the psychological demand characteristics of a training investigation (16). The low level faradic stimulation provided a distinct afferent sensation, thus making it a convincing placebo modality. For it to be a physiologically valid placebo however, it must be assumed that it did not cause chronic morphological adaptations that could enhance neuromuscular power. This placebo group significantly improved PT/0 (+8%), wheres PT/60 and PT/180 were less influenced (+3–5%) and PT/300 was unaffected. This velocity-specific pattern of improvements was also observed during the pretesting period in that disinhibition techniques acutely improved PT/0 (3–16%), whereas PT/300 was unchanged. Perrine and Edgerton (17) have suggested that neural inhibition prevents the muscle from generating the tension that it is innately capable of during slow-velocity contractions. Apparently, psychological influences (i.e., learning to disinhibit, placebo effects) that acutely reduce these inhibitions should be considered when attempting to isolate the component of augmented neuromuscular power resulting from physiological improvements based in actual morphological adaptations. In doing so (Table 3), the increases of the slow group in PT were significantly greater than those of the placebo group only at their training velocity of 60°/s. Similarly the improvements of the mixed group (>placebo) were specific to its velocities of training (60 and 300°/s). The fast group improved PT more than placebo at its training velocity of 300°/s and at 180°/s, yet, the 15% increase in PT/60, although sizable, was not significantly greater than placebo. Three fast subjects improved PT/60 by 25, 24, and 14%, whereas the fourth demonstrated a 2% reduction, thus introducing variability and the probability of a type II error.

In discussing the practical applications of these results, it should be remembered that the duration of training was 6 wk. It seems likely that training of longer duration may be required for more substantial morphological changes to occur. Performance in a slow-velocity event, which demands maximal muscular tension, is best improved through training that specifically mimicks the performance action and velocity and that develops high tension. In other words, the event should be duplicated. This slow training however will not improve fast-velocity performance, at least during initial stages (6 wk). Muscular power (high-velocity tension) is enhanced through high-velocity training that may also improve output at slower velocities. This latter finding is of particular relevance to rehabilitation patients who quite often cannot tolerate high-tension contractions.

We express our sincere thanks to the conscientious subjects who unselfishly participated in this investigation. We are indebted to Dr. Ted Percy for providing the faradic stimulator.

Received 23 June 1980; accepted in final form 26 June 1981.

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