Effects of real and imagined training on voluntary muscle activation during maximal isometric contractions

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ABSTRACT

In this study we directly tested the hypothesis that isometric strength training increases voluntary drive to muscles. In addition, it was attempted to replicate the findings of an earlier study that showed imagined training increases voluntary strength as much as actual training, as this finding provides key support for the hypothesis that training increases voluntary drive (Yue & Cole 1992). Fifty-four subjects were randomly allocated to groups that performed 8 weeks of isometric training of the elbow flexor muscles, imagined isometric training, or a control task involving the lower limbs. Voluntary isometric strength and activation of the elbow flexor muscles were measured before and after training. Voluntary activation was measured with a sensitive form of twitch interpolation. Training, imagined training and control groups increased voluntary isometric elbow flexor strength by means of 17.8% (±3.1 SEM), 6.8% (±2.6) and 6.5% (±3.0), respectively. The training group increased in strength significantly more than imagined training and control groups (P < 0.01 for both comparisons), but the small difference between imagined training and control groups was not significant (P = 0.31). Prior to training, voluntary activation of all subjects was high (96.2 ± 0.5%). These data challenge the hypothesis that training of the elbow flexor muscles increases isometric strength by inducing adaptations of the central nervous system, because they show that training does not increase voluntary activation and imagined training does not increase strength.

Keywords imagined training, isometric strength, skeletal muscle, training, voluntary activation.

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The mechanism by which high-resistance exercise increases voluntary muscle strength has been the subject of intense investigation. It is thought by some that increases in strength produced by short periods of training (less than about 2 months duration) are mediated primarily by adaptations within the central nervous system, rather than by adaptations within muscle fibres (this is sometimes called the 'neural training hypothesis', for reviews see Sale 1988 and Enoka & Fuglevand 1993). It has been hypothesized that training enables subjects to recruit more motor units and drive them at high frequencies.

A range of experimental observations have been used to support this hypothesis. For example, training has been shown to increase maximal voluntary electro myographic activity, EMG, and this has been thought to indicate that training increases voluntary neural drive to trained muscles (Moritani & DeVries 1979, Häkkinen & Komi 1983, Narici et al. 1989). However, increases in EMG might also be expected if muscle fibres were to hypertrophy, or if there was a decrease in the impedance of tissues between muscle fibres and electrodes, so demonstration of an increase in EMG does not provide unambiguous evidence of an increased neural drive to muscles. Another observation used to support the neural training hypothesis is that strength gains produced by short periods of training usually greatly exceed apparent increases in muscle cross-sectional area (e.g. Ikai & Fukunaga 1970, Narici et al. 1989). It is therefore argued that increases in strength cannot be mediated by adaptations of muscle. This argument relies on the assumption that a single anatomical cross-section of a whole muscle group will change its area in exact proportion with the muscles’ intrinsic capacity to produce torque, but this assumption is probably not valid. Many investigators have also reported that unilateral strength...
training is accompanied by contralateral increases in strength (Kannus et al. 1992, see Enoka 1988 for references). Some investigators have suggested that this is because unilateral training enhances a 'motor program' in a way which is accessible to both the trained and untrained limb. Unfortunately, none of the studies of contralateral effects of isometric training which have used appropriate controls (i.e. a group which does not train) have demonstrated greater increases in strength of untrained limbs of trained subjects than in untrained controls. Thus it is possible that the apparent contralateral effect of isometric training is simply due to familiarity with testing procedures. Lastly, isometric training at one joint angle usually produces angle-specific increases in strength (Thépaut-Mathieu et al. 1988), and it has been argued that the specificity of the training response is not easily explained by adaptations of muscle and must indicate that subjects learn angle-specific strategies to increase voluntary drive. It is possible, though, that training-induced changes in muscle and tendon could shift the muscles' torque-angle curve and manifest as an angle-specific training response (Jones & Round 1990), so angle-specific adaptations need not be evidence of a neural training effect. Clearly, there is some uncertainty about the interpretation of all of these experimental observations.

A recent study provides relatively unambiguous support for the neural training hypothesis (Yue & Cole 1992). In this study, 30 subjects were randomly allocated to groups which trained the little finger adductor muscles for 4 weeks, either with maximal voluntary isometric contractions or imagined maximal isometric contractions. Increases in voluntary strength produced by imagined training were comparable in magnitude to strength increases produced by actual training. These results were interpreted as indicating that increases in voluntary isometric strength result from improvements in the 'construction or planning of the motor program involved in maximal contractions'. The findings of this study have not yet been replicated, but they strongly suggest that strength increases are mediated by adaptations of the central nervous system.

The present study sought to test the neural training hypothesis in two ways. First, we directly tested the hypothesis that training increases voluntary neural drive to muscles, by measuring maximal voluntary activation with twitch interpolation before and after training. In addition, we attempted to replicate the findings of Yue and Cole on the effects of imagined training. Like Yue & Cole (1992) we compared the effects of actual and imagined training, but we trained the elbow flexor muscles instead of the adductors of the fifth finger. The elbow flexors are more typical of muscles that are commonly trained, and they are more suited to testing with the twitch interpolation method.

MATERIALS AND METHODS
Fifty-four subjects (31 women and 23 men), all student volunteers, participated in the study. Subjects were not undertaking and had not recently undertaken high resistance upper-limb training. Voluntary isometric strength and activation of all subjects was measured before and after a supervised 8-week training program which consisted of either maximal isometric contractions, imagined maximal isometric contractions or a control condition. Studies were conducted within the Declaration of Helsinki and the procedures were approved by the local ethics committee.

Measurement of voluntary isometric strength
The methods used to measure voluntary strength were identical to those described by Allen et al. (1995). Subjects were seated with the right elbow held at 90° and strapped firmly with inextensible webbing into a dynamometer. They were allowed to perform several ‘warm-up’ contractions prior to testing. For test contractions, they produced maximal elbow flexion efforts of 2–3 s duration. The need for truly maximal efforts was emphasized and, to maximize motivation, a prize was offered for the subject who attained the highest level of activation (see below). Visual feedback of un-scaled elbow flexor torque was provided on an oscilloscope during each contraction, and throughout contractions subjects were loudly exhorted to produce maximal efforts. The signal from a calibrated load cell attached to the dynamometer was sampled at 1000 Hz, and the peak torque produced during the contraction was determined. Subjects were asked if they felt they had made a maximal effort and, if not, the result was discarded and the contraction repeated. This applied to only a small proportion of contractions (<5%). The median peak torque from six contractions was taken as a measure of voluntary isometric strength. A rest of at least 45 s separated contractions.

Measurement of isometric activation and isometric twitch properties
The interpolated twitch method was used to measure voluntary activation during the same maximal isometric contractions (Merton 1954, Allen et al. 1995, Gandevia et al. 1995). During the plateau of each contraction a single supramaximal stimulus (0.1 ms, constant current, 50–300 mA) was delivered to the biceps through two surface electrodes over the mid-belly and distal tendon of the biceps muscle. The force signal was also passed through a sample-and-hold amplifier (additional gain of 10, Hales & Gandevia 1988) so that small changes in the level of force from those at the time of the stimulus
could be resolved with a peak detection algorithm. The amplitude of the interpolated twitch, divided by the amplitude of a potentiated control twitch evoked 5 s later with an identical stimulus delivered to the relaxed muscle, was used as a measure of voluntary activation. Voluntary activation was calculated as $100 \times (1 - a/b)$, where $a$ is the amplitude of the interpolated twitch and $b$ the amplitude of the control twitch. The median level of activation obtained from the same six contractions used to measure voluntary strength was taken as a measure of each subject’s maximal voluntary activation. Three measures were taken from each subject’s control twitches: mean twitch amplitude, contraction time (from onset of force to its peak) and half-relaxation time.

To assess strength of other muscle groups in the upper limb, voluntary grip strength of the right hand was measured with a standard dynamometer and procedure (Jaymar dynamometer, Asimow Engineering, CA, USA; Mathiowetz et al. 1985). The highest force produced during three maximal efforts was taken as a measure of voluntary grip strength.

**Training**

After testing, subjects were randomly allocated to one of three equally sized groups. Subjects in the training group (12 women and six men) underwent an isometric strength training program in which they trained the right elbow flexor muscles three times each week for 8 weeks. They were positioned throughout training sessions in the same way as for testing. All training sessions were supervised and consisted of six 10 s maximal isometric contractions with a 60 s interval between contractions. Similar training schedules have been used to increase voluntary isometric strength (McDonagh & Davies 1984). Unscaled visual feedback of isometric force was provided, and subjects were exhorted to produce maximal efforts throughout each contraction. This was done in a standardized way using a tape-recorded message played to the subject (‘Get ready to produce a maximal contraction. Now, pull as hard as you can, keep pulling as hard as you can, keep pulling... Now stop’).

Subjects in the imagined training group (nine women and nine men) trained in an identical manner to the training group except that they only imagined producing isometric contractions. Again, they were positioned as for testing sessions and strapped into the dynamometer. Another taped message exhorted the subject to imagine producing maximal contractions using similar instructions to those given to subjects in the training group (‘Get ready to imagine producing a maximal contraction. Now, imagine you are pulling as hard as you can, keep imagining you are pulling as hard as you can, keep imagining you are pulling... Now stop’). Subjects were asked to completely relax their elbow flexor muscles during imagined contractions.

The control group (10 women and eight men) did not train. However, like subjects in the training and imagined training groups, each control subject attended the laboratory three times each week and was positioned with the arm strapped into the dynamometer for the same duration as the training groups. They were asked not to contract their elbow flexor muscles and, to ensure that attentional demands put on control subjects were the same as those in the training and imagined training groups, they were asked to concentrate on keeping the weight of their feet evenly distributed. Again, a recorded message gave standardized instructions (‘Get ready to keep the weight of your feet evenly distributed. Now, keep the weight of your feet evenly distributed, keep the weight of your feet evenly distributed... Now stop’). All subjects in all groups were asked to refrain from undertaking other upper limb exercise for the duration of the study.

Of the 54 subjects randomized into groups, 50 completed all their training sessions in 8 weeks. One subject discontinued training at the end of the second week without explanation, and three other subjects withdrew for reasons unrelated to the study. Within 1 week of the last training session voluntary isometric strength and activation of all other subjects was re-measured using identical procedures to those used initially. A prize was again offered for the highest level of voluntary activation attained in these tests. After all testing, subjects completed an anonymous written questionnaire which asked ‘how well, on average, [they] complied with instructions during the actual tasks [they] were required to perform in training sessions’, by choosing from the following statements: ‘I did not comply with instructions’ or ‘I complied with instructions very little/somewhat/mostly/completely’. The main purpose of the questionnaire was to ensure that subjects in the imagined training and control groups performed their mental tasks (imagining maximal contractions, keeping weight of feet evenly distributed) as instructed, as the nature of these tasks meant they could not be formally monitored by the person supervising the training.

**Statistics**

Two-way (group × time) repeated measures analysis of variance was used to determine the effects of training on voluntary isometric strength, voluntary activation, voluntary grip strength, control twitch amplitude, contraction time and half-relaxation time. Where significant interactions were found, $t$-tests were used to compare changes in scores (percentage differences
between initial and final measurements). One subject in the control group attained a voluntary activation score of only 63% at the pre-test and increased her voluntary activation by 29% at the post-test. These values differed so greatly from published norms (>5 SD, Allen et al. 1995) that they were omitted from all analyses. Regression analysis was used to describe the relationship between changes in voluntary strength and activation, and the procedure described by Carmines & Zeller (1979) was used to correct for attenuation of the correlation by random measurement error. Results are given as means ± SEM. For all analyses a two-tailed probability of <5% was considered significant.

RESULTS

Compliance with training protocols

Most subjects indicated in the questionnaire that they had complied with the training instructions. Ninety-six per cent of subjects indicated that they had complied with instructions ‘completely’ or ‘mostly’. Only two subjects (4% of total subject number, both from the imagined training group) indicated they had complied with instructions ‘somewhat’.

Voluntary muscle strength

Voluntary isometric muscle strength of the elbow flexor muscles increased in the training, imagined training and control groups by means of 17.8% (±3.1%), 6.8% (±2.6) and 6.5% (±3.0), respectively (Figs 1 & 2a). The increase in voluntary strength was significant for all groups (main effect of time, \( P < 0.001; P < 0.001, P = 0.02 \) and \( P = 0.047 \) for training, imagined training and control groups, respectively). On average, the training group increased voluntary strength significantly more than both imagined training and control groups (group × time interaction, \( P = 0.02 \), \( P = 0.01 \) for comparisons with both imagined training and control groups). Mean voluntary strength increases in the imagined training group did not differ significantly from those in the control group (\( P = 0.31 \)). These data indicate that training produced a greater increase in voluntary strength than either imagined training or the control task. However, given the sample size, they do not constitute strong evidence of a lack of difference in the effects of imagined training and the control task.

Mean voluntary grip strength increased by 2.1% (±1.8), 8.9% (±2.9) and 5.9% (±2.2) for training, imagined training and control groups, respectively.

Figure 1 Effects of training on voluntary strength. Circles represent each subject’s strength before (B) and after (A) the 8 week experimental period. Lines connect individual subject’s pre- and post-training data.

Figure 2 Effects of training on voluntary strength and activation. Mean percentage change (±SEM) for control, imagined training and training groups. (a), isometric elbow flexor strength, (b), voluntary activation, (c), grip strength. There were significant between-group differences only for changes in elbow flexor strength. Significant differences between groups are shown with horizontal bars. Asterisks indicate significant within-group changes.
The increases in voluntary grip strength of the three groups did not differ significantly (group \times time interaction, $P \geq 0.09$).

**Voluntary activation**

Prior to training the mean voluntary activation of all subjects was 96.2% (±0.5). This increased slightly to 96.9% (±0.3) after training, but the increase was not significant, and did not differ significantly between groups (mean changes of 1.7, 0.7 and 0.1% for training, imagined training and control groups, respectively; main effect of time, $P = 0.11$, group \times time interaction, $P = 0.81$, Fig. 2b). These data indicate that training did not significantly increase voluntary activation of the tested muscles.

Examination of the relationship between changes in voluntary activation and changes in voluntary strength provides another way of testing whether changes in activation cause increases in strength (Fig. 3a). For each group, the linear regression of change in strength on change in activation had a positive slope, but this did not differ significantly from zero (slopes of 0.86, 0.54 and 1.73, $P = 0.27$, 0.55 and 0.08 for training, imagined training and control groups, respectively). The correlation between the two variables was low, with $r^2$ values of 0.08, 0.02 and 0.20 for training, imagined training and control groups (weighted mean of 0.10). The weakness of these correlations is not due to the extra scatter produced by measurement error because when a correction was made for measurement error (Carmines & Zeller 1979, using published and unpublished data of Allen et al. 1995), $r^2$ increased to only 0.14 (weighted mean for all three groups) and to only 0.11 for the training group. Thus changes in voluntary activation of the test muscles explain at most only a small amount of the variance of changes in voluntary strength. The conclusion that changes in voluntary activation account for only a small proportion of the increase in voluntary isometric strength is robust, as the same conclusions were drawn when the regressions were repeated with the one outlying data point included, or with higher-order polynomial regression, or using implausibly high estimates of measurement error.

The preceding analysis is based on the method of calculating activation described in methods. However, the conclusions did not change when alternative measurements of voluntary activation were used: there was only a weak relationship between changes in voluntary strength and either the absolute size of the interpolated twitch or the size of the interpolated twitch divided by voluntary isometric strength (mean weighted $r^2$ of 0.13 and 0.16, respectively, Fig. 3a and c).

**Control twitches**

On average, the size of control twitches increased by 16.6% between initial and final tests ($P < 0.001$), but this increase did not differ significantly across groups (mean increases of 16.2% (±6.1), 23.1% (±7.2) and 10.9% (±5.8) for training, imagined training and control groups, respectively group \times time interaction, $P = 0.34$, Figure 4a). There was also a slight decrease in twitch half-relaxation times (mean of 3.1 ± 1.3 ms, main effect of time, $P = 0.02$), but not contraction...
time (mean increase of 1.5 ± 1.2 ms, main effect of
time, \( P = 0.36 \), Figure 4b and c). The group \( \times \) time
interaction was not significant for either half-relaxation
time or contraction time (\( P = 0.3 \) and 0.9,
respectively) which indicates that there were no between-
group differences in changes in these variables.

**DISCUSSION**

The results of the present study indicate that increases
in voluntary isometric elbow flexor strength produced
by 8 weeks of training are primarily due to mechanisms
other than an increase in voluntary drive to the test
muscles. Three observations support this conclusion:
(i) voluntary activation was high prior to training,
(ii) training did not significantly alter voluntary activa-
tion and changes in voluntary activation did not differ
between training and control groups, and (iii) changes
in voluntary activation were not significantly correlated
with increases in strength.

To our knowledge no other studies have directly
tested the neural training hypothesis by measuring
voluntary activation with twitch interpolation before
and after training. However, Jones & Rutherford (1987)
used twitch interpolation to measure voluntary activa-
tion of the knee extensor muscles prior to training, and
reported that five out of six subjects could fully activate
their muscles. These findings are a little surprising, as
other studies have reported that subjects usually cannot
fully activate their muscles during maximal voluntary
efforts. Nonetheless, our findings are consistent with
the findings of that study, because they suggest that
training does not increase maximal voluntary activation
of muscles.

To establish that a change in voluntary activation
was not the primary mechanism mediating increases in
voluntary strength it was necessary to be able to detect
small changes in interpolated twitch amplitude. This is
because at high levels of voluntary activation of the
elbows flexors, interpolated twitch amplitude decreases
less than in proportion with increases in voluntary
force (e.g. Dowling et al. 1994, Allen et al. 1998), so
relatively small changes in voluntary activation could
produce significant changes in voluntary strength.
Allen et al. (1995) investigated the resolution of an
identical measurement system to that used here and
concluded that it was possible to routinely resolve in-
terpolated twitches of less than 1% of the amplitude of
twitches evoked in resting muscles. When multiple
measures are taken on each subject, as in this study,
even smaller changes in median activation can be re-
solved. Thus our methods provide sufficient resolution
to detect physiologically meaningful changes in activa-
tion. We showed that random measurement error
did not obscure changes in voluntary activation, as
changes in voluntary activation were poorly correlated
with changes in voluntary strength even after correct-
ing for attenuation of the correlation by measurement
error.

A methodological issue arises because the amplitude
of isometric twitches evoked from resting muscle
increased in all three groups. Differences between groups
were not significant, which suggests that twitch ampi-
itude was not an effect of training but rather was as-
associated with some part of the testing or training
protocol performed by all three groups. The most likely
explanation is that the amount of elbow flexor muscu-
lature activated by the stimulus was greater when
voluntary activation was tested after training. While
every effort was made to ensure that stimulus intensity
was supramaximal, these data suggest that stimulus
intensity may have been slightly submaximal initially.
The effect of increasing stimulus intensity is likely to be
small because it produces increases in the size of both
the interpolated twitch and the control twitch (i.e. both the numerator and denominator in the calculation of voluntary activation). There is a potential for bias only if any extra motor units stimulated in the final test were activated to a different level (Rutherford et al. 1986), and even then the likely direction of the bias is to make the muscle appear better activated at the final test. In addition, increases in twitch amplitude did not differ significantly across groups so even if such a bias existed, between-group comparisons of changes in activation should still be valid. Lastly, when voluntary activation was measured simply by taking the absolute size of the interpolated twitch (i.e. without dividing by the control twitch) there were no differences between groups and the correlation with changes in voluntary strength was still weak. On these grounds it seems reasonable to conclude that the increase in twitch force did not bias the findings.

The primary mechanisms responsible for the observed increases in muscle strength remain unresolved. Subjects in the control group experienced a mean increase in voluntary strength of 6.5% without performing any training. Presumably most or all of this increase in strength can be attributed to familiarity with the test procedure, and so was common to control and training groups. This interpretation is supported by the observation of a comparable increase in grip strength. The additional strength increase in the training group may be due to neural mechanisms not reflected in our measures of voluntary activation. For example, it is possible that subjects became better able to drive the elbow flexor muscles whose activation was not measured here (i.e. other elbow flexors such as brachioradialis). Support for this suggestion comes from the observation that these muscles are less well activated in maximal contractions than the biceps (Allen et al. 1998). However, it is unlikely that this mechanism could explain all of the observed increase in strength, as it would require improbably large increases in force from these muscles. Strength increases probably did not occur as a result of subjects becoming better able to relax the antagonist triceps muscle, as intramuscular recordings have shown that the triceps is electrically silent during maximal voluntary isometric contractions of the elbow flexors (Allen et al. 1995). Possibly training gave subjects an opportunity to learn strategies, other than increasing voluntary activation, which increase force production (such as optimizing the direction of pull on the dynamometer). In addition, training may have increased the intrinsic force-generating capacity of the elbow flexor muscles. This suggestion is contrary to the conclusions of studies which claim to have shown that short-duration training does not substantially increase a muscle’s intrinsic force-generating capacity (see Sale 1988 for review), but existing methods for estimating intrinsic force-generating capacity of human muscles probably lack the resolution necessary to rule out small increases. Thus, the possibility of significant peripheral muscle adaptations cannot be discounted.

It was previously reported that imagined training produced increases in voluntary strength comparable to those produced by actual training (Yue & Cole 1992). This finding provides one of the strongest arguments for the hypothesis that neural adaptations mediate training-induced increases in strength. However, the effects of imagined training were not replicated in the present study. Yue and Cole found that imagined training produced increases in voluntary strength which were significantly greater than in a control group, and which did not differ significantly from a group which performed actual training. In contrast, we found that imagined training produced increases in voluntary strength which were significantly smaller than those attained by a training group, and which did not differ significantly from those attained by a control group. The two studies differ in several respects: the present study used different muscles (elbow flexors vs. adductors of the little finger), a larger sample size (50 vs. 30 subjects), fewer and shorter training contractions (6 x 10 s vs. 15 x 10 or 15 s) and a longer training period (8 vs. 4 weeks). It is difficult to see how these methodological differences could explain the differing findings, although it may relate to the muscle being trained. We have previously shown that untrained subjects are capable of complete activation of both the elbow flexors and the adductor digiti minimi in at least some contractions (Gandevia & McKenzie 1988), although tests of voluntary activation of the intrinsic adductor pollicis muscle and elbow flexors in the same subjects suggests that median voluntary activation is lower for the intrinsic hand muscles (Herbert & Gandevia 1996), so there may have been more potential for increasing voluntary activation in the intrinsic finger adductors than in the elbow flexors. Thus, a hypothesis that is consistent with the findings of both studies is that imagined training may be more effective for those muscles with rather lower initial levels of voluntary activation.

In conclusion, this study has shown that strength training does not increase maximal voluntary activation of the biceps and brachialis muscles, and that imagined training does not produce significant increases in strength of the elbow flexor muscles. These data challenge the hypothesis that short periods of training increase isometric strength by inducing adaptations of the central nervous system.

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