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POST-CONTRACTION ERRORS IN HUMAN FORCE PRODUCTION ARE REDUCED BY MUSCLE STRETCH

BY ROBERT S. HUTTON*, KOJI KAIYA†, SHUJI SUZUKI† AND SHIROH WATANABE†

*From the * Department of Psychology, University of Washington, Seattle, Washington 98195, U.S.A. and the † Department of Physiology, Kyorin University School of Medicine, 6–20–2, Shinkawa, Mitaka-shi Tokyo 181, Japan*

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SUMMARY

1. Based on findings from a previous study of plantar-flexor muscles, the effect of a conditioning 25, 50 or 100% maximum voluntary contraction (m.v.c.) of elbow flexor muscles on the accuracy of reproducing a learned criterion muscle force (2% m.v.c.) was investigated.

2. Each conditioning contraction induced a significant error in reproducing the criterion muscle force under conditions of no visual feed-back of force. As with plantar-flexor muscles, the error was consistently in the direction of a positive bias. The magnitude of the error co-varied with the magnitude of the previous contraction and, in all cases, decayed toward criterion force values over a 35 s period.

3. A brief muscle stretch, induced before subjects attempted the criterion force, reduced the size of the error but did not completely eliminate the bias.

4. The findings provide indirect evidence of post-contraction potentiation of stretch reflex pathways. Residual post-contraction errors in force production after muscle stretch may be attributed to other central or peripheral neural factors such as, for example, potentiating effects of prior activation on submaximal tension production in skeletal muscle.

INTRODUCTION

Moderate to high intensity skeletal muscle contractions produce an aftermath of excitatory responses in spinal segmental proprioceptive circuits (Granit, Homma & Matthews, 1959; Eldred, Hutton & Smith, 1976; Hulliger, 1984; Hutton, 1984) resulting in potentiation of motoneuronal activity and enhancement of submaximal muscle tension to subsequent motoneuronal activation (Burke, Rudomin & Zajac, 1976; Suzuki & Hutton, 1976; Vandervoort, Quinlan & McComas, 1983). It is known that a brief period of muscle tetany enhances multi-unit afferent discharge from the stimulated muscle for several seconds (Hnik & Payne, 1965; Hutton, Smith & Eldred, 1973). This phenomenon has been called 'post-contraction sensory discharge' or p.c.s.d. and it has been attributed to after-discharge in group Ia and II sensory

Authors' names are in alphabetical order.

receptors (Hutton *et al.* 1973; Smith, Hutton & Eldred, 1974; Eldred *et al.* 1976; Durkovic, 1976; Suzuki & Hutton, 1976; Hulliger, 1984). P.c.s.d. was recently demonstrated in human forearm flexor muscles following concentric contractions using microneurographic procedures (Hagbarth, Hagglund, Nordin & Wallin, 1985). In all studies cited stretch of the muscle several millimetres beyond the muscle length at time of stimulation will eliminate the phenomenon. Most investigators agree (Brown, Goodwin & Matthews, 1970; Eldred *et al.* 1976; Emonet-Dénand, Hunt & Laporte, 1985) that contraction-induced augmented spindle afferent responses are due to a mechanical alteration in intrafusal muscle fibres, leading to increased stiffness in the polar regions.

If one assumes static voluntary contractions as a rule involve α–γ co-activation (Vallbo, 1971; Burke, Hagbarth & Skuse, 1978), then the occurrence of p.c.s.d. in the aftermath of a voluntary static contraction should lead to greater tonic drive of homonymous motoneuronal pools (Hutton, Smith & Eldred, 1975; Suzuki & Hutton, 1976; Enoka, Hutton & Eldred, 1980). It is conceivable that muscle tension produced by the p.c.s.d. may summate with voluntary motor commands and introduce an excitatory bias of reflex origin. The outcome would be that subjects, deprived of visual feed-back of force production, would show a positive bias in reproducing a learned criterion force in the aftermath of a maximum contraction. This is precisely what occurs (Hutton, Enoka & Suzuki, 1984). Following a 5 s maximum voluntary contraction (m.v.c.) of plantar flexors, subjects show a consistent positive bias in reproducing a learned 5% m.v.c. criterion force over a period of 45 s. Likewise, the magnitude of this error decays toward criterion force values over a time course similar to the decay seen in the p.c.s.d. response. Hutton *et al.* (1984), however, could not distinguish between effects induced in proprioceptive or other pathways. Since p.c.s.d. can be abolished by muscle stretch, the study by Hutton *et al.* (1984) was replicated in elbow flexor muscles and measurements of reproduced criterion forces were again examined after a static contraction but, also, immediately after muscle stretch.

METHODS

Subjects

After informed consent was obtained, experiments were conducted on seven female and seven male adults in a soundproof room. The right elbow flexor muscles were tested. Seven subjects completed all treatment conditions explained below while thirteen subjects completed all conditioning contraction conditions.

Experimental conditions

Conditions of the experiment are shown in Fig. 1. Subjects were seated before one storage (Fig. 1*E*) and one non-storage (Fig. 1*D*) oscilloscope placed at eye level. Their right elbow rested on a table located next to their right side. With the forearm in supination, subjects gripped a handle which was attached to a spring-loaded scale and potentiometer (Fig. 2*B*) by a wire rope. The angle of pull was adjusted by vertically aligning the manipulandum (Fig. 1*A*). Subjects were positioned so that the initial elbow angle and angle of attachment of the wire rope and handle approximated 90 deg. Force was recorded by the potentiometer. An electrogoniometer (Fig. 1*C*) was attached to the lateral side of the elbow for recording elbow angle.

Testing procedures

Initially, subjects were shown how to perform an isometric contraction of the elbow flexors. Since the subjects were pulling against a spring-loaded potentiometer, each isometric contraction in-

volved a small initial concentric contraction. Following a short practice period, two to three 100% m.v.c.s were attempted using visual feed-back of force from the left storage oscilloscope (Fig. 1*E*). The largest of these m.v.c. responses was designated maximum and a corresponding vertical elevation of the oscilloscope sweep beam was marked. The width of the marker was equivalent in time to a 5 s contraction. A contraction force of 2% m.v.c. was calculated and later used as the criterion force. Subjects were trained to accurately reproduce this force level by practising with visual feed-back of their force output displayed on the non-storage oscilloscope (Fig. 1*D*). To accomplish this, subjects superimposed two oscilloscope beams that were initially offset by a vertical distance equivalent to a 2% m.v.c.

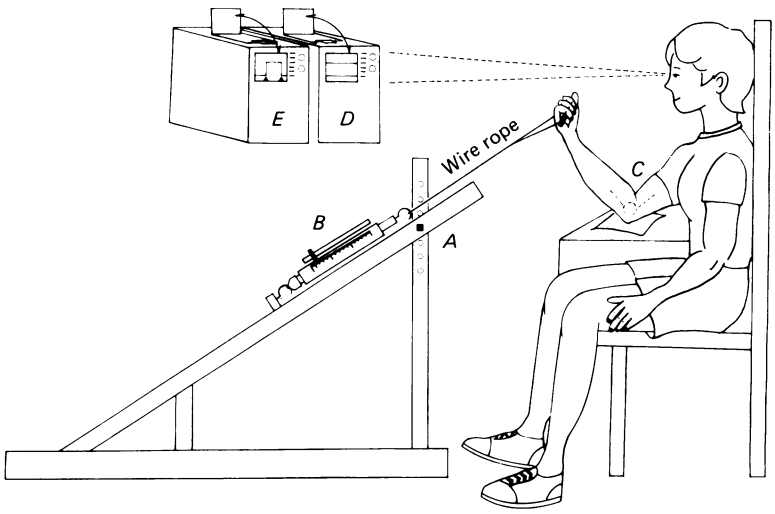

Fig. 1. Schematic drawing of the experimental arrangement. See text for explanation.

Force data were recorded on a chart recorder. Subjects practised for 5–10 min the sensation of 'tension' and 'force' (both verbal cues were used) while exerting a 2% m.v.c. The oscilloscope faces were then covered (Fig. 1*D* and *E*) and the subject was 'blind' tested for accuracy in producing a 2% m.v.c. If the error were greater than 30% of the criterion force, they continued practising with visual feed-back. Ten subjects accomplished accurate reproductions under 'blind' conditions within 10 min or less.

Subjects reproduced a 2% m.v.c. under 'blind' conditions over three trials with an intertrial interval of 1 min. Prior to each 'blind' test, subjects sustained a 2% m.v.c. with visual feed-back for 10–30 s. These periods were immediately followed by a 5 s relaxation interval. The average force level of the post-relaxation 'blind' condition, measured at 5 s intervals over a 35 s period, was used as the control data. Subjects were then asked to reproduce a 2% m.v.c. under 'blind' conditions following a 25, 50 or 100% m.v.c. As in the control condition, each conditioning contraction effort was preceded by a 10–30 s period of sustained 2% m.v.c. effort under conditions of visual feed-back. The average error of three trials after each conditioning contraction was calculated in the same manner used to obtain the control data. Each conditioning contraction was randomly assigned across subjects. A 5–10 min rest interval was allowed between each trial-set of the assigned conditioning contraction. During these rest intervals, subjects practised reproducing the criterion force 2–3 times, with visual feed-back. Immediately following each 'blind' estimate of force, the oscilloscope was uncovered and subjects were allowed to see in which direction they had erred. All subjects, therefore, were allowed the necessary information needed to compensate for the direction of error seen at the end of each trial.

Some subjects were retested following a 5–10 min rest period. Test conditions involving a 25% (n = 9 subjects) or 100% m.v.c. (n = 8 subjects) were then repeated as in the above format. Just prior to each post-contraction reproduction of a 2% m.v.c. force, however, subjects were asked to voluntarily extend their elbow from 90 to 180 deg, thereby stretching the flexor muscle groups over a joint excursion of 90 deg. In two subjects, an additional session was added wherein stretch was

induced passively by the experimenter. The type of stretch (active or passive) appeared to play no role in the outcome of the results.

Data analysis

All individual force values were considered together, averaged and either converted to percentage of the absolute 2% m.v.c. force values or averaged in newton (N) units. Two, two-way analyses of variance were applied to the data with treatment factors being the magnitude of the conditioning contraction force, trials (time) and the magnitude of the conditioning contraction force, stretch *vs.* non-stretch test conditions, respectively. Data showing significant F ratios ($P < 0{\cdot}05$) were treated with a *post hoc* Tukey test to determine differences among mean values.

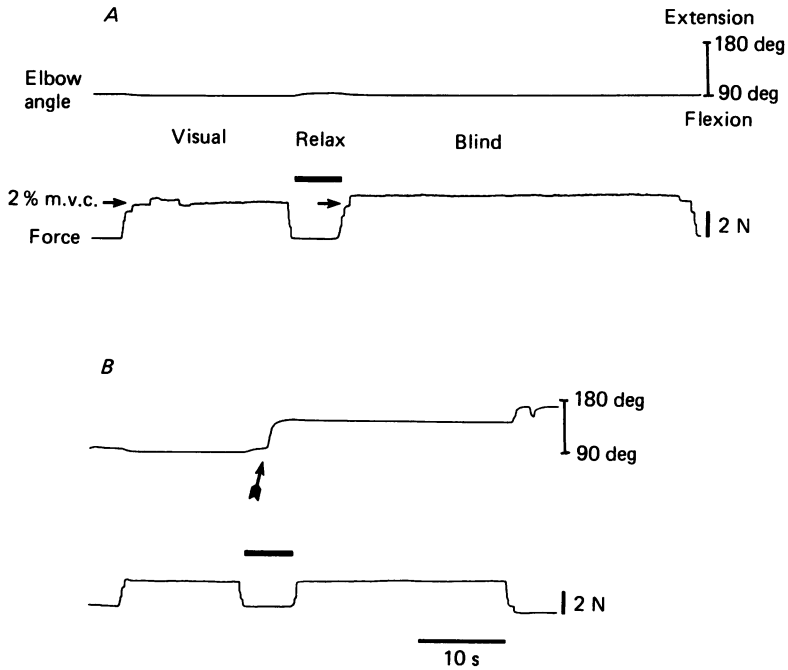

Fig. 2. *A*, a control estimate of a 2% m.v.c. criterion force is shown before (with visual feed-back) and after (blind) a 5 s period of relaxation (relax). A slight positive bias in force (second arrow) during the blind estimate was noted. *B*, changing the elbow angle from 90 to 140 deg prior to the blind force condition did not influence the accuracy of the force estimate.

RESULTS

An example of a test trial following 5 s of relaxation is shown for one subject in Fig. 2. As noted (Fig. 2*A*), there was a natural tendency towards a positive bias in force even after a period of relaxation (mean absolute 2% m.v.c. value = 2·6 N; mean control 'blind' value = 3·1 N for thirteen subjects). In a few subjects, the elbow angle was changed from 90 to 140 deg during the control relaxation period to determine if joint position was a factor influencing the accuracy of reproducing the criterion force. As shown in Fig. 2*B*, a 2% m.v.c. could be closely re-established even at a new joint angle during the 'blind' control condition.

An example of post-contraction force values from a single subject is shown in Fig. 3.

A progressive increase in the positive bias of the criterion force (Fig. 3, C-1 control) can be seen as the conditioning muscle force was increased from 25 to 100% m.v.c. (Fig. 3, T-1, T-2, T-3, respectively). Since these conditioning efforts were assigned in random order across subjects, errors were not associated with the ordering of contraction intensities.

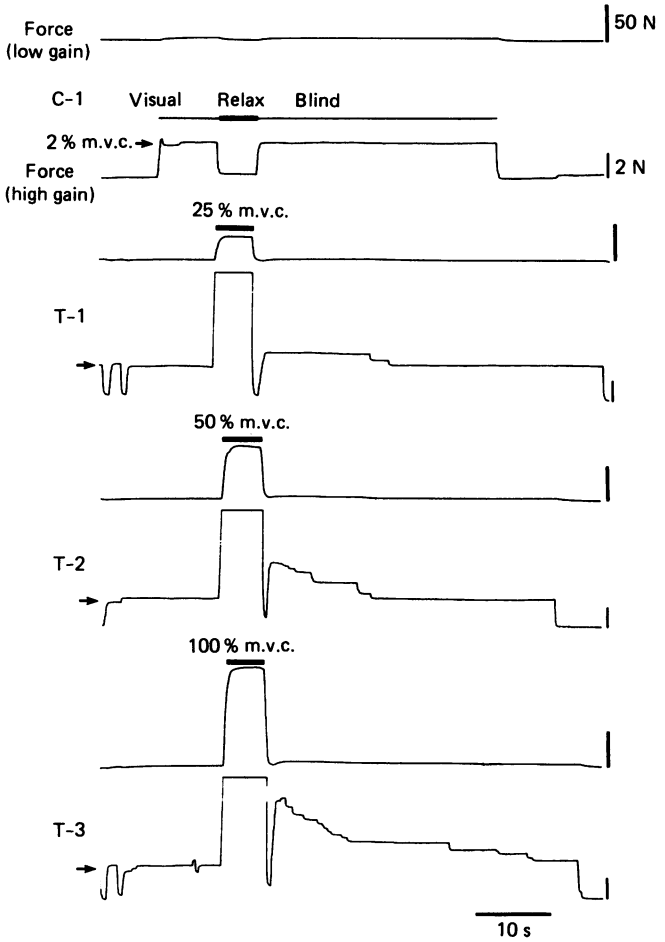

Fig. 3. Examples of post-contraction 'blind' estimates of a 2% m.v.c. force in a single subject. Condition C-1 is as in Fig. 2*A* but traces show force at low (top trace) and high gain (lower trace). Attempts to produce the criterion force following a 25% m.v.c. (T-1), 50% m.v.c. (T-2), and 100% m.v.c. (T-3) are shown. A progressive increase in the magnitude of error is noted.

Post-contraction errors, expressed in percentage mean force of the mean pre-contraction 2% m.v.c. force, are shown for thirteen subjects completing all the conditioning contraction conditions in Fig. 4. The general shape of these curves and characteristic decay towards the criterion force values for all three contraction intensities are essentially identical to those previously reported by Hutton *et al.* (1984) for plantar-flexion muscles. The post-contraction mean force values were significantly

higher than the control values ($P < 0.001$). Immediately following the conditioning contractions of 100, 50 and 25% m.v.c., the errors in force were 204, 152 and 103% higher than the control values, respectively. All 100% m.v.c. post-contraction mean force values per trial were significantly higher than the 25% m.v.c. post-contraction values at the $P < 0.01$ level but differed non-significantly from the 50% m.v.c. post-

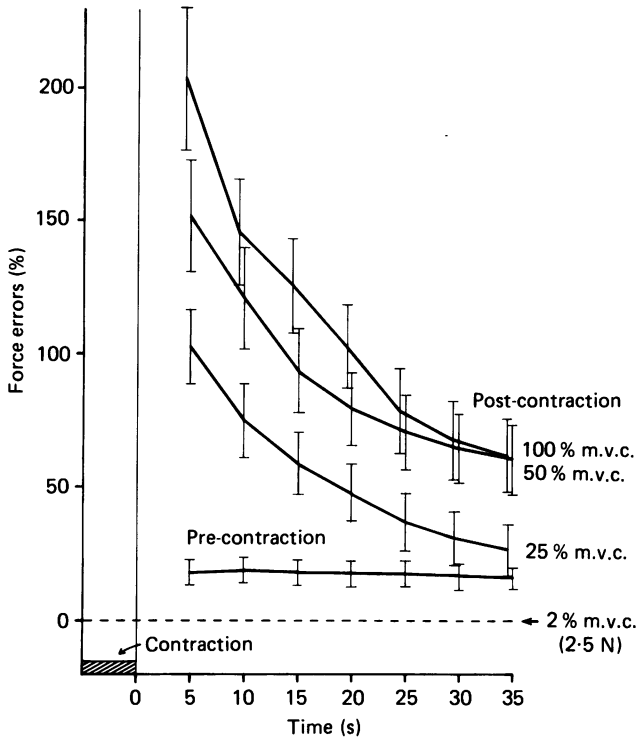

Fig. 4. Summary of the post-contraction average percentage errors in force relative to the average post-relaxation (pre-contraction control) 2% m.v.c. force is shown for a 25, 50 and 100% m.v.c. ($n = 13$). Vertical bars indicate ±S.E. of the mean values. See text for further comments.

contraction mean forces except for the force level observed 5 s after contraction ($P < 0.05$). All 50% m.v.c. post-contraction values were significantly greater than the 25% m.v.c. post-contraction values at the $P < 0.05$ level. Likewise, all 25% m.v.c. post-contraction forces were significantly greater than control values at the $P < 0.05$ level. The conditioning contraction force, therefore, was significantly associated with the magnitude of the error during 'blind' testing. There was a highly significant trials (time) effect ($P < 0.01$) and magnitude of conditioning contraction force–trials interaction ($P < 0.01$), i.e. the magnitude of post-contraction errors in force for all conditioning contractions, diminished towards criterion force values at differing rates over the 35 s period of observation (Fig. 4). For example, 35 s after the conditioning contraction, the errors in force for the 100, 50 and 25% m.v.c. conditions were 62, 61 and 27% higher than control values, respectively; however, at 15 s

following contraction, these force values were 126, 94 and 59% higher than control values, respectively.

For a single subject, post-contraction errors in force during 'blind' testing are shown in Fig. 5 without (Fig. 5*A–C*) or following biceps muscle stretch (Fig. 5*D–F*)

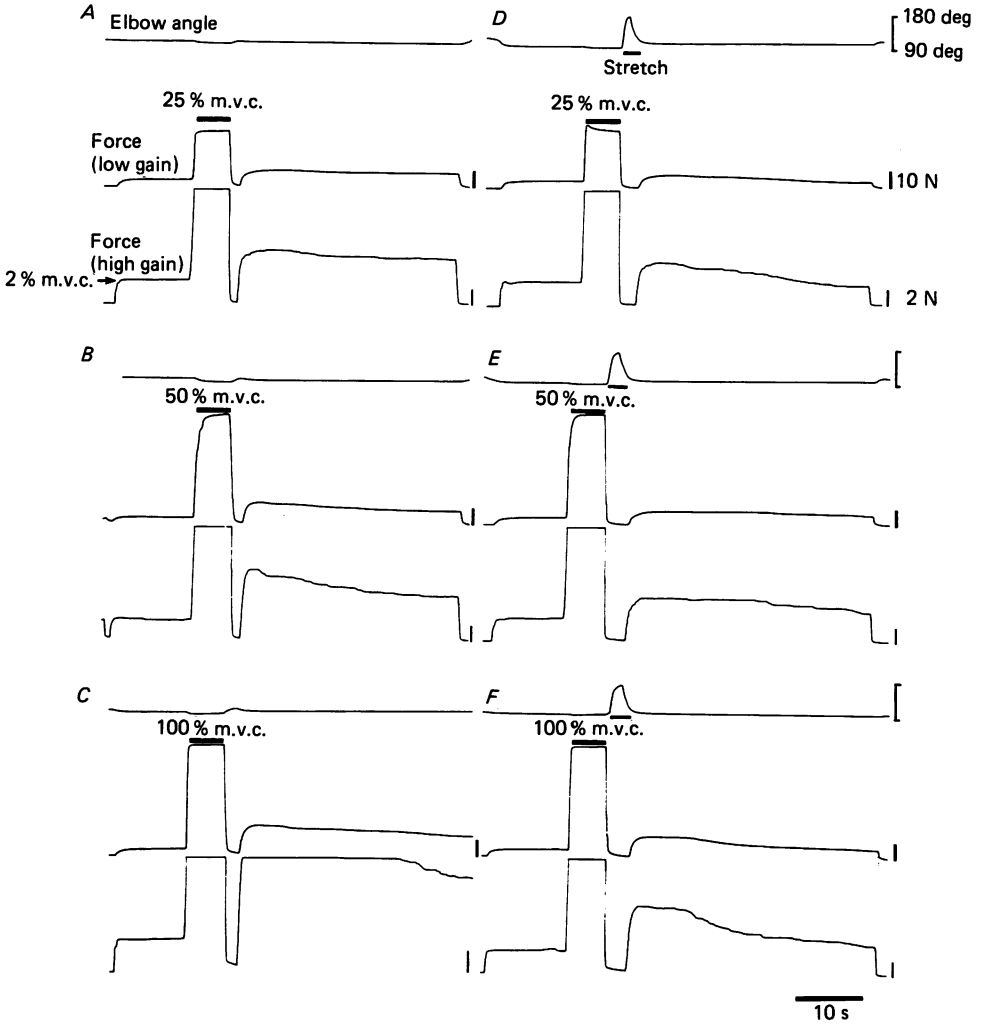

Fig. 5. Examples of post-contraction errors in force are shown before (*A–C*) and after (*D–F*) muscle stretch (90 deg of active elbow extension) for the 25, 50 and 100% m.v.c. conditions. It is seen that muscle stretch reduces the post-contraction error but does not eliminate it.

induced by a momentary 90 deg elbow joint excursion into extension. Immediate post-contraction muscle stretch attenuated the size of the error in force at all contraction force levels. Of particular interest is the observation that the degree of attenuation was more marked the greater the magnitude of error.

The effect of muscle stretch on the size of the post-contraction constant error in force is summarized in Fig. 6. For the 25% m.v.c. condition, all trials involving non-stretch differed significantly from trials preceded by stretch at the $P < 0.05$ level. However, under the 100% m.v.c condition, the mean values between the post-contraction non-stretch and stretch trials were found to be significantly different at the $P < 0.01$ level.

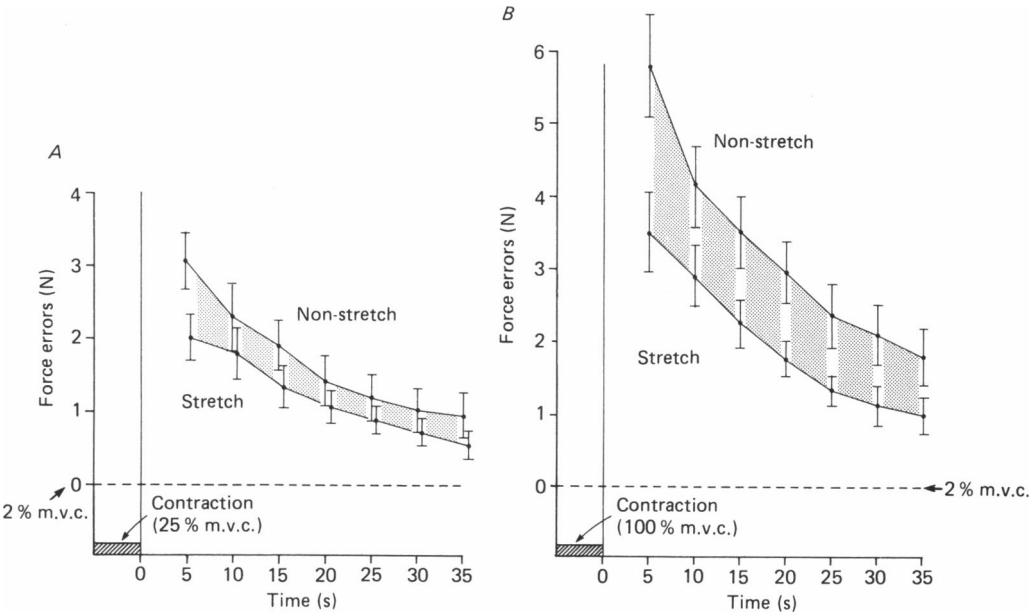

Fig. 6. Summary of post-contraction average errors in force (N), is shown for the 25% (*A*, $n = 9$), and 100% m.v.c. (*B*, $n = 8$) conditions before (non-stretch) and after (stretch) a 90 deg active elbow displacement into extension. Vertical bars show ±S.E. of the mean values. Shaded areas denote the decrease in size of the post-contraction error following muscle stretch. For all trials, differences in mean force values were statistically significant ($P < 0.05$, 25% m.v.c. condition, and $P < 0.01$, 100% m.v.c. condition).

DISCUSSION

Elbow flexor muscle contractions at 25, 50 and 100% m.v.c. were found to produce significant errors (positive bias) in a subsequent 'blind' reproduction of a learned 2% m.v.c. criterion force. The magnitude of the errors paralleled the force of contraction. Hutton *et al.* (1984) have previously proposed that such errors in post-contraction force estimates could be attributed to post-contraction potentiation of proprioceptive excitatory pathways; however, other factors occurring within the central nervous system and the motor unit could not be excluded. Since potentiation of excitatory drive in proprioceptive pathways, attributed to p.c.s.d., has been shown to be eliminated by muscle stretch beyond the length at which the respective fibres were activated (Brown, Goodwin & Matthews, 1969; Hutton *et al.* 1973; Smith *et al.* 1974; Baumann, Emonet-Dénand & Hulliger, 1982), an interpolated muscle stretch occurring before a post-contraction estimate of the criterion force should be effective in reducing or eliminating the error observed. If a reduction in error occurs but the

positive bias nevertheless persists, then the residual error may be attributed to other mechanisms occurring within other pathways or the motor unit. Our findings support the latter proposition and, therefore, this conclusion warrants further discussion.

After-discharge in muscle receptors

Prolonged after-discharge in muscle spindles induced by stimulation of γ-motor-fibres was first reported by Kuffler, Hunt & Quilliam in 1951. Subsequently, Granit and co-workers (1959) reported similar findings following brief taps given to the tendon of leg extensor muscle or shock-induced muscle twitches. A shortening of the 'post-stretch pause' in Ia afferent receptors also occurs during a sequence of muscle twitches (Decandia, Schieppati & Crenna, 1978). The mechanism underlying this after-discharge appears to be associated with either contraction- or stretch-induced increased stiffness in intrafusal muscle fibres causing tonic stretch in the nuclear bag region, the site of Ia afferent receptors (Brown *et al.* 1969, 1970; Hutton *et al.* 1973; Emonet-Dénand, Laporte & Tristant, 1980; Morgan, Prochaska & Proske, 1984). Since stretch alone may induce contractions of the bag_1 intrafusal fibre (Poppele & Quick, 1981), it remains to be resolved whether intrafusal fibre activation is a requisite factor in inducing after-discharge in spindle Ia afferent fibres. Of particular importance to the present investigation is the common observation that p.c.s.d. can be abolished by muscle stretch. Therefore, any increase in the excitatory drive to the motoneuronal pools caused by p.c.s.d. (Suzuki & Hutton, 1976; Hutton & Suzuki, 1979) should be abolished by muscle stretch of sufficient magnitude. On the assumption that post-contraction after-discharge of muscle spindles does occur in humans, providing that the contractions are of sufficient intensity and are not performed eccentrically (Enoka *et al.* 1980; Hagbarth *et al.* 1985), Hutton and co-workers (1984) hypothesized that contraction-induced errors in force estimates above a learned criterion force could be explained as a summation of excitation between spinal reflex pathways and a previously learned set of motor commands. This condition would thereby produce errors with a positive bias in perceived effort. Arguments for this position were based principally on the findings that (1) the post-contraction errors in force were in the appropriate direction, (2) the size of the positive bias in reproducing the criterion force co-varied with the magnitude of the conditioning contraction force (see Hutton *et al.* 1973), and (3) the error in force showed a temporal decay toward control values over the time course of several seconds, as does the occurrence of the p.c.s.d. (Hutton *et al.* 1973; Smith *et al.* 1974). However, in the Hutton *et al.* (1984) study, an interpolated muscle stretch between muscle contraction and the 'blind' test of reproducing the criterion force was not investigated. The present investigation replicated all previous observations of Hutton *et al.* (1984) using elbow flexor muscles instead of plantar flexors. In addition, post-contraction muscle stretch significantly reduced the size of the post-contraction error.

Other peripheral or central factors

Because a residual positive bias in force persisted after muscle stretch, this result may be partly attributed to potentiating factors occurring within the motor units, such as post-contraction twitch potentiation (Vandervoort *et al.* 1983), since the mechanism for this potentiated response is not similarly affected by muscle stretch

(e.g. contrast Fig. 6 in Vandervort *et al.* 1983 with Fig. 3 in Smith *et al.* 1974). Given the low criterion muscle force (2% m.v.c.), it might be argued that only smaller motor units were recruited during the 'blind' test as determined by the size principle and the distribution of Ia afferent fibres to homonymous motoneurones (Suzuki & Hutton, 1976). As these units tend to show less twitch potentiation after voluntary contraction (Vandervoort *et al.* 1983), the significance of this peripheral mechanism might be questioned. It should be recalled, however, that post-contraction attempts to reproduce the criterion force were initially 100–200% above the criterion value. Likewise, similar post-contraction errors in force production have been shown for a 5% m.v.c. plantar-flexion criterion force (Hutton *et al.* 1984) and for elbow flexion criterion forces as high as 50% m.v.c. (E. R. O'Connell, unpublished observations using a slightly different testing protocol). Part of the initial large bias in post-contraction force might be attributed to a temporary desensitization of Golgi Ib receptors to muscle stretch and/or tension (Smith *et al.* 1974; Hutton & Nelson, 1986).

Recently, Hagbarth *et al.* (1985) reported changes in finger flexor muscle stiffness following conditioning contractions. Isometric contractions decreased and finger flexion shortening contractions increased muscle stiffness, respectively. Potentiated reflex responses and enhanced multi-unit afferent discharge (presumably due largely to muscle spindle afferents) were also associated with enhanced muscle stiffness. Since the conditioning contractions used in the present investigation initially involved a small shortening contraction (see Methods), our findings of a consistent positive bias in post-contraction muscle force would be consistent with the findings of Hagbarth *et al.* (1985). As it was shown also that *passive* finger flexion movements enhanced muscle stiffness, a change in the inherent properties of muscle (e.g. persistent actin–myosin bonds in extra- and intrafusal muscle fibres) cannot be ruled out as an additional, albeit small, contributing peripheral factor to post-contraction errors in force; however, since it was shown that muscle extension movements decreased finger flexor muscle stiffness, our findings of a persistence in post-contraction force errors following muscle stretch must likely be attributed to factors associated with muscle activation.

Other central factors may involve post-tetanic potentiation (p.t.p.) of monosynaptic pathways (Hagbarth, 1962; Suzuki & Hutton, 1976) or, perhaps, a resetting of the 'sense of effort' or 'corollary discharge' (Granit, 1972). The magnitude of p.t.p. does co-vary with the magnitude of motoneuronal activation and does decay over a time course similar to the post-contraction error herein described (Hutton, 1984). It might be questioned, however, if the frequency of motoneuronal activation from Ia afferent fibres during contraction is high enough to induce p.t.p. (see Hagbarth, 1962). Less is known and can be said about how changes in sense of effort or corollary discharge might also contribute to our findings.

Implications

Consistent errors in the execution of movement have been a subject of considerable experimentation over the past century (see Granit, 1972, for a review). Likewise, different terminology has evolved to identify the nature of this (these?) phenomenon (phenomena?), e.g. 'Kohnstamm phenomenon' (Kohnstamm, 1915), 'postural per-

sistence' (Selling, 1930), 'after-contraction' (Hick, 1953), 'kinaesthetic after-effects' (Hutton, 1963; Cratty & Hutton, 1964). However, little attention has been given to the underlying physiological mechanisms associated with these reported phenomena.

The nature of these errors as studied in the present investigation shares common features with previous observations and, as such, may reveal that potentiation of spinal reflex pathways could partly explain the basis for contraction-induced positive biases in reproducing learned criterion forces. It is interesting to note that when subjects are required to perform a fine manipulative task following exercise or a static contraction, they experience considerable difficulty in precision of movement over several minutes (Davies & Pratt, 1976; Davies & Ward, 1978). This outcome is entirely in line with the notion that the motor command for a precision movement has not been adjusted for a contraction-induced potentiation of the γ-loop and, perhaps, other excitatory neural pathways (Hutton, 1984).

More recently Craske & Craske (1985) have argued that 'musculature after-contractions' may reveal oscillator mechanisms in the motor apparatus, but they disregard how oscillatory movements might also be generated by after-discharge from proprioceptors. It should be recalled that Sherrington was able to elicit stepping movements in cat hindlimb by 'rivalry' of antagonistic reflexes, presumably as an outcome of 'double reciprocal innervation' (Sherrington, 1913; see also Hultborn, Wigstrom & Wangberg, 1975; Tyler & Hutton, 1986). It is quite conceivable, therefore, that after-discharge induced in two antagonistic pairs of muscles could give rise to oscillatory motor responses. As yet, we have not explored the specific effects antagonistic muscle contractions may have on the observance of contraction-induced errors. One might predict that antagonistic contractions may reduce post-contraction errors in muscle force due to reciprocal inhibition. This prediction awaits further experimentation.

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