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OPTIMAL CONTRACTION INTENSITY DURING PROPRIOCEPTIVE NEUROMUSCULAR FACILITATION FOR MAXIMAL INCREASE OF RANGE OF MOTION

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ABSTRACT

Sheard, PW and Paine, TJ. Optimal contraction intensity during proprioceptive neuromuscular facilitation for maximal increase of range of motion. *J Strength Cond Res* 24(X): 000–000, 2009—An informal review of literature on the use of postisometric relaxation (PIR) type proprioceptive neuromuscular facilitation (PNF) indicates that the force of contraction requested from the athlete ranges from 10 to 100% of maximum voluntary isometric contraction (MVIC). The purpose of this study was therefore to determine if an optimal contraction intensity to elicit maximum positive change in range of motion (Δ ROM) exists. This research question was tested across a convenience sample of 56 (37 male and 19 female) university athletes. Target contractions during PNF interventions were set at 20, 50, and 100% MVIC. Pre- and post-PNF intervention hip flexion range of motion (ROM) was measured on a unilateral straight leg raise. The target MVIC of 20, 50, and 100% elicited mean pre-post intervention Δ ROM of 8.4, 12.9, and 11.6°, respectively (all $p \leq 0.0001$). Differences in pre-post intervention Δ ROM between target contraction intensities were also significant ($p = 0.016$ to ≤ 0.0001). A peak Δ ROM of 13.3° was found at a PNF contraction intensity of 64.3% MVIC. Where optimizing increased ROM in healthy athletes is the desired outcome of PIR-PNF application, coaches and trainers should elicit contraction intensities of approximately 65% MVIC.

KEY WORDS joint flexibility, muscle stretching exercise, manual therapies

INTRODUCTION

Proprioceptive neuromuscular facilitation (PNF) is commonly reported (1,5,22,24,25) to be the most effective stretching protocol for eliciting maximum increase in range of motion (ROM). Many

elements combine to create a PNF protocol: agonist and/or antagonist contraction, duration of contraction phase, duration of relaxation phase, ratio of contraction to relaxation durations, contraction intensity, number of contractions, and timing of movement from end of contraction to new point of bind. Contraction intensity has been monitored and controlled by electronic/mechanical feedback in very few studies (3,9,16), the majority relying instead on “feel” to estimate contraction intensity.

A range of contraction intensities, from 10 to 100%, are proposed in the literature when undertaking PNF protocols: 10 (15), 20 (4,9,15), 50 (3,4,20), 60 (9), 75 (3,20,23), and 100% (2,6,9,10,18–21,26). Of these studies, only Feland and Marin (9) explicitly attempted to distinguish the efficacy of different feedback-controlled contraction intensities on change in range of motion (Δ ROM). We feel that while the fixed (via real-time visual feedback) nature of their contraction intensity is an example of good experimental control, it is not a true reflection of PNF as applied in clinical or training scenarios. We hypothesized that when the therapist-requested contraction intensity is not participant controlled by real-time feedback, a range of contraction intensities and a range of associated changes in ROM would result. We further hypothesized that the resultant data would be indicative of a correlation between PNF contraction intensity and post-PNF Δ ROM. We tested these hypotheses in the case of PNF applied to the hamstring group of university-level field sport athletes. The main purpose of this study was therefore to determine if an optimal contraction intensity to elicit maximum positive Δ ROM exists when contraction intensity is not controlled with electronic/mechanical feedback.

METHODS

Experimental Approach to the Problem

A randomized, crossover, experimental design was used for this study, eliminating issues of inter-group variability. Each athlete participated on 4 nonconsecutive days. Day 1 was a familiarization session at the end of which maximum voluntary isometric contraction (MVIC) was established. At weekly intervals, athletes returned for their 3 randomly

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assigned, Latin square–derived, experimental intervention sessions.

The independent variable of target PNF contraction intensities set at 20, 50, and 100% MVIC was imposed to elicit actual PNF contraction intensities close to those targets; with the athletes being blind to their efforts, it was assumed that, in contrast to the study of Feland and Marin (9), the actual intensities produced would display a degree of variability. The change (D) in the dependent variable of pre- and post-PNF intervention angles of hip flexion for a straight leg raise (ROM, in degrees) allowed for an estimate of the efficacy of the interventions in eliciting increased ROM. The measuring and recording of the second dependent variable, the actual PNF contraction intensities, allowed for a between-intervention comparison of contraction intensities produced. A line of best fit applied to the scatter plot of the Δ ROM against the actual contraction intensities allowed for an estimation of the best actual contraction intensity for eliciting optimal increases in ROM.

Subjects

After obtaining ethics approval from the Institute for Sport and Physical Activity Research Ethics Committee in compliance with their requirements for human testing, volunteer athletes gave written informed consent to participate and completed medical screening. Maximum hip extensor ROM was established using a unilateral straight leg raise with the athlete declared dominant limb. Athletes reporting history of traumatic hip or knee injury in the past 6 months ($n = 5$) or demonstrating hip flexion exceeding 80° ($n = 11$) were excluded. The remaining convenience sample of 56 athletes (male: $n = 37$, 21.1 ± 1.3 years, 179.9 ± 9.0 cm, and 78.6 ± 9.7 kg and female: $n = 19$, 21.5 ± 1.0 years, 168.1 ± 7.8 cm, and 64.5 ± 10.7 kg) completed all familiarization and testing sessions. All athletes were British University Sports Association soccer (Division 2), rugby (Division 2), or netball (Division 1) players in weeks 3 through 9 of their competitive seasons.

Sample size was determined following Hopkins' (13) equation 3

$$8S^2/d^2$$

where S is SEM and d is the smallest worthwhile effect.

The value for S was estimated at 13.2° (14) derived from test/retest MVC values of 12 university field sport athletes (male, $n = 6$ and female, $n = 6$) all of whom were later recruited as participants for the study. The smallest worthwhile effect was estimated at 5° Δ ROM following the PNF trials. From these values, the corresponding sample size was estimated at $n = 55.7$ where $\alpha = 0.05$ and $\beta = 0.80$.

Procedures

Establishing Preintervention Maximum Voluntary Isometric Contraction. Athletes underwent the procedure described by Stoll et al. (27) to establish their MVIC strength for hip extension, as follows: starting from the straight leg raise position, athletes were required to repeat two 5-second

MVIC with a 30-second passive rest interval between attempts; no incentive was provided by the tester in order that measuring conditions be standardized; the higher of the 2 contraction forces, as recorded by a calibrated strain-gauge dynamometer (ErgoMeter; Globus, Codogno, Italy), represented the athletes' MVIC.

Random Assignment, Recording, and Blinding. Target PNF contractions were set at 20, 50, and 100% MVIC, with sequence of application randomly assigned against a Latin square design for protocol sequences. Athletes' names were drawn from one "hat" and protocol sequences from a second. Contraction intensity was recorded throughout the duration of each contraction with the strain-gauge dynamometer for later analysis. Athletes and researchers were blind to the athlete efforts.

Proprioceptive Neuromuscular Facilitation and Measurement Protocol. The following PNF stretch protocol was used: active motion (hip flexion) to first point of bind, build to target contraction (isometric hip extension) over 5 seconds, hold target contraction for 7 seconds, active reset to new point of bind, and relax for 12 seconds. This sequence was repeated for 3 contractions. Isometric contractions were resisted using anchored straps, with the strain gauge in-line, rather than by the researchers, who monitored the sessions and reset strap tensions between contractions.

Using a fluid-filled goniometer (Clinical goniometer; MIE Medical Research, Ltd., Leeds, United Kingdom), ROM was measured at first point of bind preceding first contraction, after reset to first point of bind following first and second contractions, and after final 12-second hold of the third contraction. The difference of the final ROM measure minus the first ROM measure was designated Δ ROM.

Reproducibility of Measurement Variables. Within a pilot study, 12 university field sport athletes (male, $n = 6$ and female, $n = 6$), all of whom were later recruited as participants for the main study, were tested on 2 occasions, 1 week apart. Good reliability between the initial measure of hip flexion ($^\circ$) for a straight leg raise to point of first bind on the 2 test days was found for the procedure with coefficient of variation $<5.0\%$ ($CV = 3.1\%$).

Statistical Analyses

Descriptive data are presented as means \pm SDs . Assumptions for parametric testing were confirmed before significance testing. Statistical significance was set at $p \leq 0.05$.

Range of Motion Comparisons. Differences in pre- to post-PNF intervention Δ ROM for the subject population within each of the 20, 50, and 100% target MVIC PNF contraction interventions were explored using paired t -tests. Following Vickers' (28) argument that analysis of covariance (ANCOVA) is the most appropriate method for analyzing results, which compare a baseline measure with a postintervention measure, differences in pre- to post-PNF intervention Δ ROM for

the subject population among the 20, 50, and 100% target MVIC PNF contractions were explored using ANCOVA; the postintervention measure was set as “dependent,” and the preintervention measure was set as “covariate.”

Optimum Contraction Estimations. To derive a single representative value for the dependent variable of actual PNF contraction intensity, the mean contraction intensity recorded from 6 to 12 seconds was averaged from the 3 component contractions at each of the 20, 50, and 100% target PNF contraction intensity levels. From this, 4 scatter plots of the mean actual PNF contraction intensity against the cumulative Δ ROM following the 3 component contractions were obtained; 1 each at 20%, 50%, and 100% MVIC and 1 for the pooled data of the 3 intensities. Linear and second-order polynomial lines of best fit were generated for each of the 4 scatter plots, as were R values. A paired sample t -test comparing the R values of the linear and second-order polynomial lines of best fit was used to determine if a significant difference in agreement existed between the 2 lines of best fit. Optimum contraction intensity (%MVIC) was then derived as the value produced from running multiple iterations of the second-order polynomial from the pooled data against 0.1 increments around the peak ROM as degrees ($^{\circ}$).

RESULTS

Pre-Post Intervention Changes

Significant pre-post intervention Δ ROM ($p \leq 0.0001$) were found at the 3 target contraction intensities of 20, 50, and 100% MVIC (Figure 1). Actual mean contraction intensities were target 20% ($30.5 \pm 5.8\%$), target 50% ($55.2 \pm 6.1\%$), and target 100% ($78.3 \pm 13.3\%$).

F1

Between-Intervention Comparisons

Analysis of covariance showed significant Δ ROM differences between 20, 50, and 100% target contraction intensities following the 3 interventions ($p \leq 0.0001$). Simple contrasts demonstrated that significant differences ($p = 0.016$ to ≤ 0.0001) were to be found at all target contraction intensity pair-wise comparisons (Figure 1).

Optimum Contraction Estimation

The linear lines of best fit for the 20, 50, and 100% target contractions demonstrate moderate correlations between the contraction intensity achieved and the Δ ROM elicited ($R = 0.518, 0.471, \text{ and } -0.490$, respectively; Figures 2–4). The pooled data produced a weak correlation between the contraction intensity achieved and the Δ ROM elicited ($R = 0.377$; Figure 5). Second-order polynomial lines of best fit were significantly better than the linear ($p = 0.034$) at 20, 50, 100%, and pooled target contraction intensities where $R = 0.522, 0.538, -0.558, \text{ and } 0.688$, respectively (Figures 2–5). The second-order polynomial from the pooled data ($y = -0.0037x^2 + 0.475x - 1.906$) gives an optimal contraction intensity of 64.3%, eliciting a maximum Δ ROM of 13.3 $^{\circ}$ in hip flexion measured against a unilateral single leg.

F2 – F4

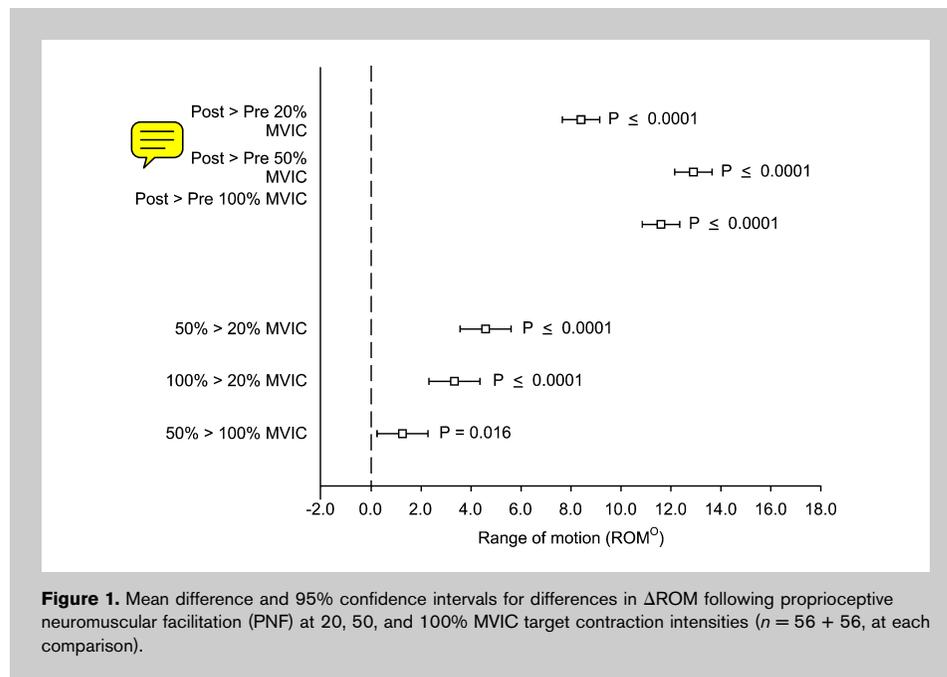
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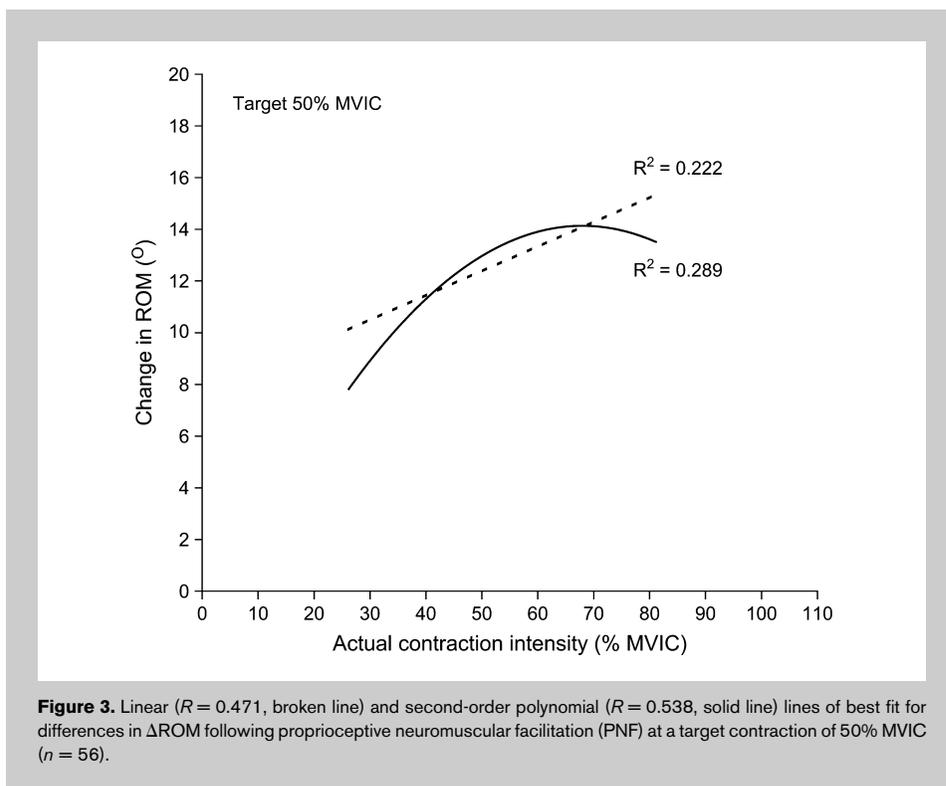
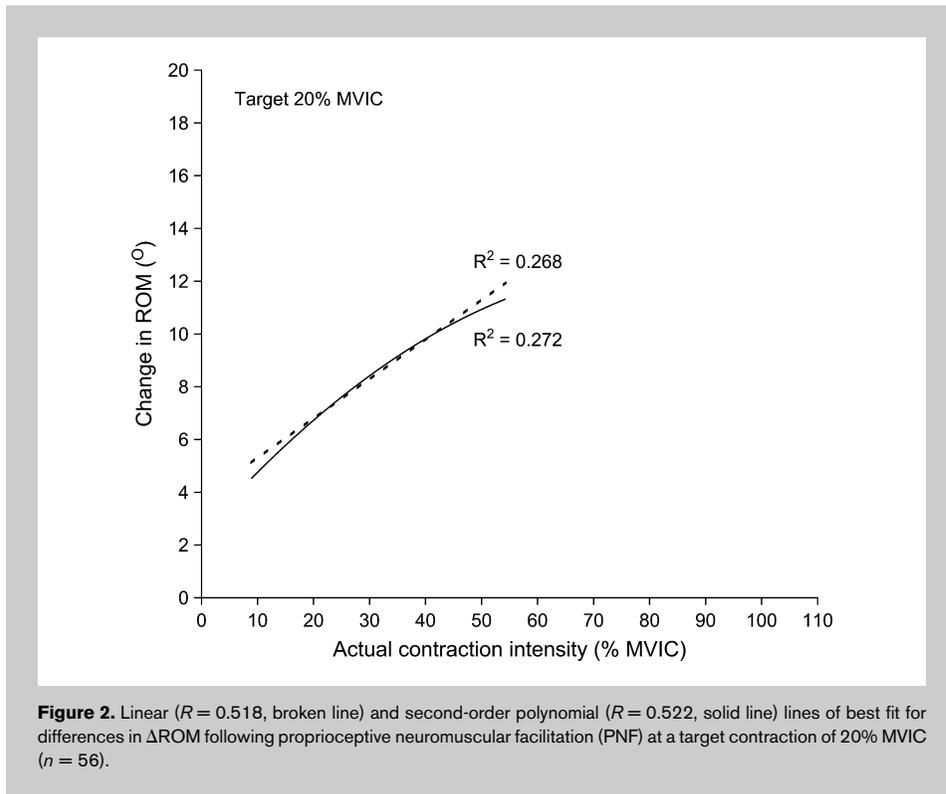
DISCUSSION

Chalmers (5) has argued that the excitation of the inhibitory effect of the Golgi tendon organs (GTO) following resisted isometric contraction during PNF is an insufficient rationale for the magnitude of Δ ROM following postisometric PNF. The primary contention is that the inhibitory effect is too transient (approximately 1 second) to carry through the PNF protocol. The early work of Gottlieb and Agarwal (approximately 0.5 (11) to 0.8 seconds (12)) support Chalmers (5), but Enoka et al. (7) suggest that the inhibitory response does not return to baseline for up to 50 seconds if minimal intermittent stimulus remains present. This would allow sufficient time for a “cumulative” inhibitory response to accrue, allowing for some of the observed Δ ROM following PNF. The work of Etnyre and Abraham (8) also supports Enoka et al. (7), arguing that there may be more than 1 inhibitory response suppressing the motor pool excitability. The work of Moore and Hutton (18) further confounds the issue with acknowledged contradictory EMG and Δ ROM findings. It may be that the involuntary paroxysmal

F4

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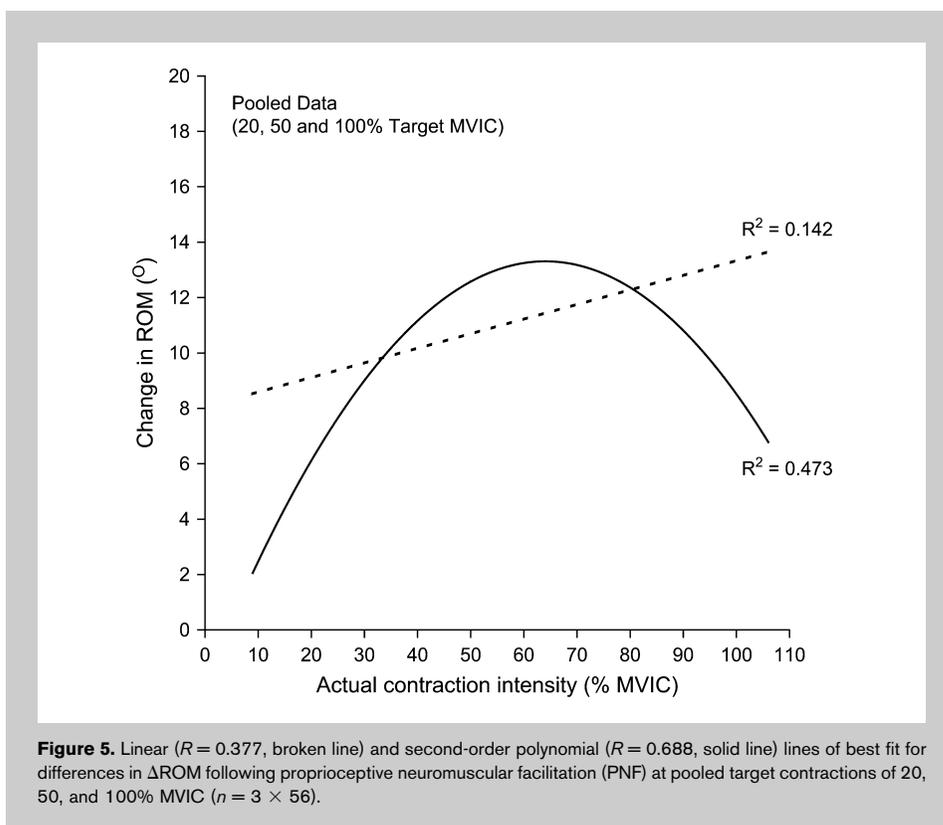
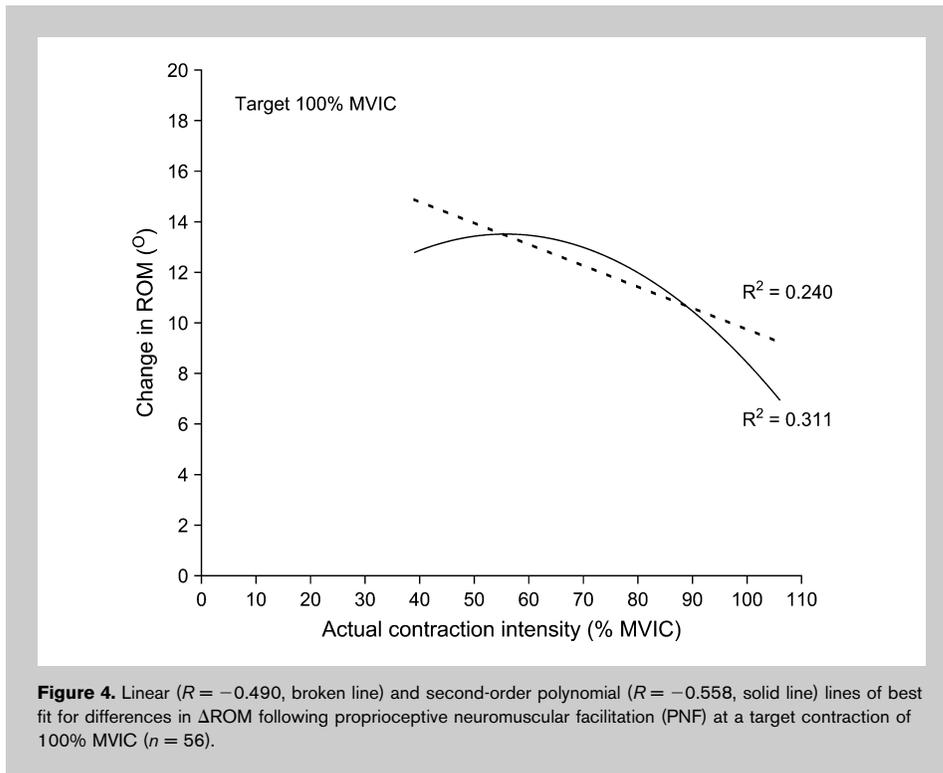




tremors reported with some of their subjects are indicative of muscle spindle fiber excitation in conflict with GTO inhibition. Enoka et al. (7) imply that previous animal models may support this conjecture.

Of particular interest in the work of Moore and Hutton (18) is the significant correlation between lower subject discomfort, decreasing EMG activity, and increased perceived efficacy of the stretch protocols. The implication is that alterations in stretch perception may play a role in the efficacy of PNF. Magnusson et al. (16) demonstrated an 18–21% decline in passive torque, and Mitchell et al. (17) demonstrated an approximately 9% increase in active torque at a constant stretch tolerance.

In light of the above, the positive slope of the curves of Figure 2 (target 20% MVIC) may be indicative of increasing GTO inhibition leading to increased Δ ROM but with insufficient stimulus to optimize the efficacy of treatment through adequate alteration of the stretch tolerance. The strong positive slope may also indicate that a muscle spindle fiber-evoked protective shortening of the muscles has not been stimulated to dampen the response. The linear curve of Figure 3 would argue for continued inhibition from the GTO and possible optimization of the stretch tolerance. The second-order curve may indicate that at higher contraction intensities, the contractile excitation of the muscle spindle fibers is overriding the inhibition of the GTO. Both the linear and, more so, the second-order polynomial curves of Figure 4 argue strongly for the further excitation of the muscle spindle fibers and a decrease in



stretch tolerance as MVIC is approached. Finally, Figure 5 demonstrates that a simple, linear, “push harder, stretch further” model of PNF is inferior to a more complex “multifactor interaction” (GTO inhibition, muscle spindle fiber desensitization, and increased stretch tolerance) second-order model ($R^2 = 0.142$ vs. 0.430).

If we accept the above rationale for a multifactor interaction at work in PNF (which certainly needs to be tested with both EMG and discomfort measured), then Figure 5 represents an interaction of GTO, muscle spindle fiber, and stretch perception components across a continuum of contraction intensities:

1. At lower contraction intensities (< approximately 50% MVIC), small increases in ROM are linked to viscoelastic changes and suboptimal GTO inhibition.
2. At higher contraction intensities (> approximately 70% MVIC), muscle spindle fiber excitation overrides GTO inhibition, decreasing potential viscoelastic changes and moderating the positive influence of stretch perception as MVIC elicits local discomfort.
3. At moderate (approximately 50–70% MVIC) contraction intensities, viscoelastic changes are enhanced by optimized GTO inhibition and stretch perception tolerance.

Within the moderate range, the optimum contraction intensity is estimated at 64.3% MVIC for an optimized Δ ROM of 13.3° in healthy athletes.

PRACTICAL APPLICATIONS

The efficacy of submaximal contraction intensity for PNF

application reported by Feland and Marin (9), and echoed by Sharman et al. (24) in their review, is further supported by the present study. Although no evidence that maximal contraction intensity PNF actually causes injury is available in the literature, caution against the use of higher contraction intensities is becoming more common (3,4,9,15,24). On the basis of our findings, we recommend that when athletes and trainers perform PNF, they use a submaximal contraction intensity approaching 65% MVIC to maximize Δ ROM while limiting the potential risk of injury that may accompany PNF application at, or approaching, 100% MVIC.

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