The Effect of General Anesthesia on Passive-Knee-Extension Range of Motion

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Context: Flexibility is promoted as essential to physical fitness, but the mechanisms limiting it are not fully understood. Objective: To investigate the effects of general anesthesia on hamstring extensibility. Design: Repeated measures. Setting: Hospital operating room. Subjects: Eight volunteers undergoing orthopedic surgeries unrelated to the tested limb. Measurement: Three measurements of passive knee extension (PKE) taken before and after administration of general anesthesia. The force applied during the measurements was consistent between trials. Results: Mean PKE range of motion (ROM) was significantly greater before anesthesia (75.0° ± 11.8°) than after (53.3° ± 17°; t = 5.6, P < .001). Pearson product correlation revealed a significant correlation between the mean difference in PKE ROM between treatment conditions and subjects’ body weight (r = .91, P < .05). Conclusions: The findings might be attributable to diminished neural drive to the antagonist muscle groups and suggest a more complex neural control of flexibility than simply neural drive to an agonist muscle. Key Words: flexibility, hamstrings, neural inhibition

For decades, individuals involved in sport and fitness have advocated flexibility as a primary component of physical activity. Although stretching to increase flexibility is promoted as an essential adjunct to physical activity and therapy, the mechanisms that contribute to increases in flexibility have not been clearly identified. The 2 popularly implicated mechanisms thought to influence flexibility are passive mechanical lengthening (muscle extensibility) and neural activity. Neural activity can be further divided into increased stretch tolerance and active contractile responses.

Passive mechanical lengthening is a change in the physical length or extensibility of the musculotendinous unit, and it is theorized that it changes through stretching. There are 3 properties common to viscoelastic tissue, of which the musculotendinous unit is composed. These include stress relaxation, hysteresis, and creep. Stress relaxation is increased extensibility with a corresponding change in elasticity and has been demonstrated
in vitro using a rabbit as a model.\textsuperscript{6} Similarly, viscoelastic stress relaxation has been demonstrated in vivo using human subjects.\textsuperscript{1-3,5} Hysteresis is a change in the load-deformation curve during loading and unloading, and creep is the continual lengthening of tissue until eventual failure during a constant load application.\textsuperscript{1-3,6} The complexities of these properties make generalizations to neurologically intact humans difficult.

Neurological responses are the second mechanism thought to account for increases in range of motion (ROM) from flexibility training. These responses can be further divided into active contractile responses and stretch tolerance. Stretch tolerance is the pain threshold during a stretch and the accommodation to the discomfort as the stretch is held for a period of time.\textsuperscript{7-13} Halbertsma et al\textsuperscript{7} demonstrated that maximal muscle moment and extensibility increased significantly while elasticity curves remained unchanged. Stretch tolerance might be related to stimulation of free nerve endings of types III and IV afferent neurons.\textsuperscript{4} Muscle spindles and Golgi tendon organs are the primary proprioceptive stretch receptors and account for active contractile responses.\textsuperscript{4,14}

Stimulation of muscle spindles initiates monosynaptic and polysynaptic reflexes and, in turn, elicits autogenic gamma and alpha motor activity within that muscle.\textsuperscript{4,15,16} Conversely, the Golgi tendon organ is stimulated by the contraction of a muscle and is responsible for autogenic inhibition of that muscle and excitation of the antagonist.\textsuperscript{4,15,16} The theoretical basis of proprioceptive neuromuscular facilitation (PNF) is the autogenic inhibition and reciprocal inhibition properties of these receptors.\textsuperscript{14,17-20} PNF stretching techniques have been shown to increase ROM acutely,\textsuperscript{17-19} but electromyography (EMG) studies have shown that these techniques increase local muscular activity rather than diminish it.\textsuperscript{17-20} The association of stretch receptors and free nerve endings and the interaction with the viscoelastic component remain the center of debate. Assessment of flexibility under general anesthesia might help delineate the factors affecting elongation of the musculotendinous unit.

Currently, there is nothing in the literature describing quantifiable measurements of muscle extensibility of anesthetized subjects. Anesthetized subjects have significantly reduced central-nervous-system and peripheral afferent activity and, to a lesser degree, reduced efferent motor activity.\textsuperscript{21-24} The purpose of this study was to assess the effects of general anesthesia on extensibility of the hamstring musculature, thereby gaining insight on the mechanism from which flexibility gains are obtained.

**Methods**

**Subjects**

For this study, 7 male patients and 1 female patient undergoing orthopedic surgery (mean age = 24 ± 6.9 years, mass = 82.8 ± 17.7 kg) volunteered for the experiment. Three men and 1 woman underwent surgeries to the lower
extremity, and in these cases, the contralateral leg was assessed for flexibility. Subjects reported no history of lumbosacral or lower extremity pathology affecting the test leg within the preceding 3 months. Subjects were able to achieve at least 120° of hip flexion with the knee flexed and were limited at least 30° from full passive knee extension (PKE) while the hip was fixed at 110°. These ranges of hip flexion and limited knee extension were chosen to allow for sufficient ROM if large increases occurred under anesthesia. Subjects were free of adverse neural tension, as demonstrated by a negative Lasegue’s test. The institutional review boards of the Centre County Community Hospital (State College, Pa) and The Pennsylvania State University approved all experimental procedures for use on human subjects, and all subjects provided informed consent before the testing.

**Apparatus**

Two fluid goniometers (Baseline Bubble Inclinometer, Fabrication Enterprises Inc, Irvington, NY) were fitted with Velcro™ hooks on the posterior side of the devices. The goniometers have a clear graduated disk overlying a clear circular tube half filled with blue fluid. The graduated disk can be rotated to place the zero at the level of the fluid when the segment being examined is in the neutral, or 0°, position. When the goniometer is rotated, the fluid moves to an angle equal to the angular displacement of the base, and measurements are made in 1° increments. Fluid goniometers have been shown to have high intertester reliability (intraclass correlation coefficient [ICC] = .92) and were chosen for this study because of the ease and speed of their application in the operating room. Two 10-cm-wide, terry-cloth-covered elastic wraps were used to secure the fluid goniometers to the patients. The terry cloth provided a secure base to which to attach the Velcro hooks on the back of the fluid goniometers.

A handheld dynamometer (Nicholas Manual Muscle Tester, Model 01160, Lafayette Instruments, North Lafayette, Ind) was used to determine the maximum force used during both experimental conditions. This dynamometer has been shown to have high intertester and intratester reliability, with an ICC exceeding .90, and was convenient to use in the operating room. The device had a range of 0.0–199.9 kg. The LCD display of the device would return a peak-force output for each trial in kilograms, with a sensitivity of 0.1 kg, and was accurate up to ±0.5% of full scale. The dynamometer was factory fitted with an application stirrup that transfers the force from the subject to the load cell inside the device. The calibration of the dynamometer was checked before each recording was taken according to manufacturer instructions.

**Procedure**

Each subject participated in 2 measurement sessions. The first session consisted of a screening exam and the no-anesthesia PKE ROM measurement, and the second session consisted of PKE-ROM measurement while the pa-
tient was under anesthesia. During the first session, subjects were screened for recent lower extremity pathologies in the limb to be measured, and the 3 no-anesthesia PKE trials were conducted. The first measurement session occurred 1 hour before surgery and before the administration of any pharmaceuticals. During the second session, collection of the 3 anesthesia trials began approximately 5 minutes after anesthetization, at the discretion of the surgeon and anesthesiologist. The same methods, testing apparatus, and procedures for collection were used during each treatment condition.

As part of the screening exam, the Lasegue’s test was administered, and if no signs of adverse neural tension were present, the ROM was then measured. The Lasegue’s test was performed on the test leg as described by Magee.27 A positive test excluded 1 candidate from the study.

Range of motion was measured on the leg of the side unrelated to the surgical procedure, with the subject lying supine. One goniometer was secured to the midshaft of the lateral leg using 1 terry-cloth strap. The distal edge of the second terry-cloth strap was placed approximately 2 cm proximal to the superior pole of the patella. The second goniometer was secured to the second strap on the lateral side of the thigh (see Figure 1). With the subject supine and the leg flat on the table, the goniometers were set at 0° by the examiner. With the knee bent, the hip was flexed to end-ROM or when patient discomfort was reached. Subjects unable to reach 120° of hip flexion were excluded from the study. A strap was used during the initial screening to secure the contralateral leg but was not used in the operating room, because this would have required placing a strap over the surgical leg.

Figure 1 Passive knee extension with the hip held in 110° hip flexion. Force of displacement is monitored with a handheld dynamometer.
of subjects having lower extremity surgery. Surgical protocol required that the goniometers and straps be removed after the pre-anesthesia measures were taken and then replaced in the operating room.

PKE was measured if the subject was able to reach 120° of hip flexion. With hip flexion maintained at 110° by one examiner, a second examiner then passively extended the knee until the patient reported the first feeling of discomfort. At that point, angular displacement of the knee was measured. All subjects that were included had PKE values equal to or greater than 30° from full extension at 110° hip flexion. Two potential subjects were excluded from the study because they were able to achieve less than 30° from full knee extension at 110° of hip flexion.

While the subject was lying supine, maximal PKE was measured by applying the goniometers in the same positions as described previously. The same examiner that positioned the proximal goniometer maintained hip flexion at 110° with the knee bent while another examiner positioned the Nicholas Manual Muscle Tester (NMMT) as shown in Figure 1. The distal edge of the stirrup on the NMMT was placed 8 cm proximal to the medial malleolus. The same examiner then applied a force perpendicular to the shaft of the lower leg with the NMMT while the hip-flexion angle was maintained. The knee was passively extended at a slow rate of angular displacement until the subject reported their first sensation of discomfort. The examiner applying the force immediately took a force measurement in kilograms (accurate to within 0.1 kg) with the NMMT. Simultaneously, the angular displacement of the knee was attained from the fluid goniometer on the lower leg. This was repeated 3 times for each of the 2 measurement conditions, and the average force of the 3 no-anesthesia trials was used as the limit of applied force during the anesthesia condition.

Data Analysis

The mean PKE for the 3 trials under each condition was calculated. Measurement reliability and standard errors of measurement (SEMs) were estimated by calculating ICCs as described by Shrout and Fleiss. A 2-tailed dependent t test was conducted to identify significant differences in PKE under the no-anesthesia and anesthesia conditions. The relationship between the differences in ROM between conditions and subjects’ average body weight was estimated using a Pearson product correlation. The level of significance was set at \( P < .05 \) for all analyses.

Results

ICCs and SEMs, calculated for each measurement condition, revealed high reliability under the no-anesthesia and anesthesia conditions, ICC = .99, SEM = 1.2°, and ICC = .98, SEM = 2.4°, respectively. A 2-tailed dependent t test identified a significant difference between the no-anesthesia and anesthesia
conditions \( t = 5.60, P < .001 \). The mean PKE of the no-anesthesia condition was 75.0° (± 11.8°), and the mean PKE under anesthesia was 53.3° (± 17.0°). The mean PKE of each trial under each condition is plotted in Figure 2. A significant correlation was identified between the average difference between treatment conditions and the subject’s body weight \( r = .91, P < .05 \). The body mass of each subject was plotted against the average difference in ROM from each condition (Figure 3). As body weight increased, the average difference between treatment conditions also increased.

**Figure 2** Range of passive-knee-extension motion across 3 trials with and without general anesthesia (mean ± SD).

**Figure 3** The best-fit line for the average difference in range-of-motion measurements between treatment conditions and body mass.
Our findings suggest that general anesthesia results in a decrease in PKE ROM. We expected to observe ROM increases under general anesthesia as a result of depression of neural activation of the hamstrings, but our findings suggest just the opposite. We believe that these results might be attributable to the depression of antagonist muscle activity under anesthesia. During our study, subjects were asked to remain completely passive throughout the PKE procedure; however, consciously or unconsciously evoked cocontraction of the musculature surrounding the hip and knee is likely to have occurred.

This assertion is supported by EMG studies that have demonstrated cocontraction of the quadriceps and hamstring muscle groups during passive and PNF stretching techniques. In those studies, it was not determined whether the activity measured was the result of consciously or unconsciously evoked contractions of the agonist, reciprocal inhibition caused by contraction of the antagonist, or muscle-spindle-initiated reflexes, all of which can produce measurable potentials that would be difficult to differentiate using surface electrodes. In our study, data were collected in an operating room, precluding the use of EMG to measure muscle recruitment.

Yamada and Yoshizawa investigated EMG activity of the hamstrings, quadriceps, gluteus maximus, and erector spinae during a passive straight-leg-raise (SLR) test of patients under and not under the effects of anesthesia. The authors report that there was concurrent hamstring and quadriceps activity toward the end-ROM during the SLR tests and that it was significantly less when subjects were anesthetized. ROM was not measured in their study; however, our findings support those of Yamada and Yoshizawa. The decrease in ROM during the anesthesia condition observed in our study corresponds to the decrease in neural activity observed in the aforementioned study. Unfortunately, the use of EMG was precluded in our study because of concerns of the surgeons and anesthesiologists.

Studies involving PNF stretching techniques have demonstrated increased agonist and antagonist neural activity with concurrent increases in ROM. Others, however, have reported that EMG activity recorded during stretching maneuvers is too low to have an appreciable affect on extensibility. It should also be noted that EMG activity recorded with surface electrodes might not be sensitive enough to detect small changes in neural activity. In addition, minimal EMG activity was used as a determinant of end-ROM and not measured as an outcome variable in previous studies. Our findings are in agreement with those mentioned previously that demonstrated that EMG activity increased during passive stretching maneuvers. Our findings suggest that inhibition caused by antagonist activity is required for maximal muscle extensibility. We believe that decreased neural recruitment of the antagonist musculature under general
anesthesia is responsible for decreased hamstring-muscle extensibility. Therefore, conscious passive stretching might not be entirely passive but might include inhibitory potentials generated from the antagonist musculature. This is also supported by the significant relationship of body weight and decreased ROM.

The magnitude of the difference in ROM was higher in subjects with higher body weight, further supporting our conclusion that hamstring-muscle extensibility is at least in part caused by antagonist-muscle contractions. In addition to the original data collection, we measured 3 subjects 1 time at tissue resistance defined by investigator experience. These single-trial measurements were taken after the 3 measures using a controlled force. They revealed slightly greater range of motion, but motion was still substantially less than the respective no-anesthesia-condition measurements. These results, although not part of the original study design, are consistent with our theory of reduced neural activity to the antagonist muscles, rather than gravity being responsible for the results observed. No statistical analysis was performed on these data.

This study did have limitations that should be appropriately addressed. We included subjects undergoing surgery to the lower extremity because of the small number of patients undergoing surgery to the upper extremity in our surgeon’s clinical practice. The inclusion of patients undergoing surgery to the lower extremity necessitated eliminating the same strapping procedure for the contralateral leg that we used in the no-anesthesia condition. During the anesthesia condition, another investigator manually secured the contralateral leg to the table. This difference could be a source of measurement error. Nonetheless, because of the strong correlation between body weight and change in PKE ROM, we believe that the potential error that can be attributed to this methodological difference is negligible.

Another limitation was the inability to control for which anesthetics were administered. Many variables affect the types and amounts of anesthetic administered during surgery. Surgeon, anesthetist, and patient preference are the major factors, but there are other, less controllable, factors, as well. Patients’ age, weight, allergies, and type of surgery all affect what types of anesthetics will be used for a particular surgery. The use of muscle relaxants was also based on several factors. Intubation is the primary reason for the their use, but surgeon and anesthetist preference also affect these decisions. Nonetheless, the consistency of subject responses under anesthesia suggests that variations in anesthesia had little effect on our results. Future studies might attempt to better control for these factors by recruiting subjects matched on similar demographic and surgical characteristics or by recruiting only those who will have a spinal or regional block performed.

It was hypothesized from the onset of this study that general anesthesia would allow increased ROM by decreasing central and peripheral sensory information elicited by passive stretching. Our results do not support this hypothesis. Flexibility might be limited not only by the extensibility of
muscle or other surrounding soft tissues but also by neural drive to the antagonist muscles in an effort to maintain a level of tone. Our results suggest that general anesthesia alters this mechanism. We conclude that neural recruitment of 1 or more antagonist-muscle groups facilitates hamstring elongation during PKE. These results suggest that hamstring extensibility might be best facilitated by contraction of the antagonist musculature. Further investigations into the role of neural constraints to ROM are clearly warranted.

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References