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Hand

Increasing Dorsal Tilt in Distal Radius Fractures Does Not Increase Median Nerve Strain

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Background: Although extensive research shows an association between distal radius fractures and the development of median nerve related pathologies such as carpal tunnel syndrome, none directly track how the resulting angular deformity relates to likelihood of development of median nerve pathology.
Methods: Median nerve strain was measured with a custom-built system using a camera, optical markers, and a proprietary segmentation algorithm. After initial validation of the system in a cadaver model, our system was used to assess strain in 10 cadaver arms with a simulated distal radius fracture and increasing dorsal angulation. The measured strain at each angle was then analyzed using a linear regression model.
Results: The linear regression model in the validation experiment demonstrated a regression coefficient of 1.00067 ($P < 0.0001$) with $r^2 = 0.899$, thus validating the use of the optical tracking system. The average strain at maximum dorsal angulation (50 degrees) across all specimens was −0.2%. Linear regression analysis of the effect of increasing dorsal angulation on strain in the osteotomy model yielded a regression coefficient of −0.000048 ($P = 0.714$), $r^2 = 0.00129$, suggesting no significant correlation between increasing dorsal tilt and median nerve strain.
Conclusions: Increases in median nerve strain at the wrist are negligible with increasing dorsal tilt in a distal radius fracture model. It is therefore likely that other factors, such as increased pressure within the carpal tunnel, are the primary cause of median neuropathy in distal radius malunions. Therefore, correction of dorsal tilt may not be required to improve neurologic symptoms. *(Plast Reconstr Surg Glob Open 2022;10:e4177; doi: 10.1097/GOX.0000000000004177; Published online 24 March 2022.)*

INTRODUCTION

Distal radius fractures (DRF) comprise up to 20% of all fractures treated in the emergency room, with data showing an increase in the reported incidence over time as the population ages.[1] Although the rate of operative treatment is on the rise,[2] there is a growing recognition that patients over the age of 65 years may not require surgery, even in the setting of significant fracture displacement.[3] Despite this, a previous work has shown that almost 20% of patients treated nonoperatively may develop subacute or chronic carpal tunnel syndrome within 3 months.[4] In comparison, a study performed in the Netherlands by Bongers et al reported the incidence of carpal tunnel in the general population to be 1.4 cases per 1000 people or 0.14%, suggesting a significant increase in the risk of developing carpal tunnel syndrome following a distal radius fracture.[5] The patients most at risk for this delayed onset of carpal tunnel are also the patients most likely to be treated without surgery—elderly patients with a low energy fall.[6] In elderly patients with carpal tunnel symptoms after distal radius fracture malunion, surgeons must choose whether to perform a corrective osteotomy, a carpal tunnel release, or both. Therefore, a better understanding of the pathologic changes to the median nerve and carpal tunnel are needed to inform surgical decision-making and clinical counseling.

Prior work has established a link between distal radius fractures and the development of carpal tunnel syndrome or other median-nerve-related pathologies; however, the precise cause of this correlation is unclear.[7,8] The pathophysiology of carpal tunnel syndrome has been attributed to mechanical damage from chronic or acute compression,

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combined with relative ischemia from focal compression or blockage of venous outflow.[9]

Animal studies demonstrated that increased strain on nerves may lead to changes in vascularity and nerve conduction properties.[10,11] However, the relative contribution of increased strain versus compression of the median nerve to symptoms of subacute or chronic carpal tunnel syndrome after a distal radius malunion are unknown. A better understanding of the effects of increasing dorsal angulation on median nerve strain may help inform criteria for operative treatment or at least aid in counseling between various treatment options for patients presenting with delayed carpal tunnel syndrome following distal radius fractures. In the present study, we hypothesize that increasing dorsal angulation in extra-articular distal radius fractures increases the strain on the median nerve in a cadaveric model.

MATERIALS AND METHODS

Calculation of Strain Measurements

Strain is defined as the deformation in a material due to stress. It is a ratio of the change in length of a material compared with the original length. Strain in the median nerve was assessed using a previously-validated custom-built system consisting of an overhead high-resolution camera and optical markers.[12] A combination of super glue and green dye was utilized to create markers, which were then affixed to the nerve at 1 cm intervals (Fig. 1). A digital photograph of the markers was then segmented using a custom MATLAB program to isolate the markers based on their color profile. Figures 2 and 3 demonstrate this segmentation algorithm. Figure 2 represents the image from Figure 3 run through program. The program then identified the center of each optical marker and assigned it two-dimensional coordinates. By measuring the change in the distance between the markers (ΔL) compared with the initial distance between them (L), the strain (ε) was able to be calculated using the formula

$$\varepsilon = \Delta L \ / \ L. \quad (1)$$

Takeaways

Question: Does dorsal tilt of the distal radius in a distal radius fracture lead to strain on the median nerve?

Findings: Increases in median nerve strain at the wrist are negligible with increasing dorsal tilt in a distal radius fracture model.

Meaning: It is therefore likely that other factors, such as increased pressure within the carpal tunnel, are the primary cause of median neuropathy in distal radius malunions. Therefore, correction of dorsal tilt may not be required to improve neurologic symptoms.

Ex Vivo Validation

Ex vivo validation of the above technique was performed on median nerves harvested from fresh frozen cadavers. A 10-cm segment of median nerve was excised from four cadaveric forearms thawed for 24 hours before testing. The nerves were cut at the proximal volar wrist crease and 10 cm proximal to the wrist crease to allow removal from the specimen. Once extracted, nerves were placed in a custom 3D-printed jig (Fig. 1), which fixed each nerve segment on both ends with clamps and allowed for stretching of the nerve by fixed lengths, as determined with a digital caliper. The nerves were stretched in increments of 0.635 cm (0.25 inches), after which the calculated strain from the optical system using equation (1) was compared with the known strain, based on the induced change in length.

Distal Radius Fracture Model

An oscillating saw was used to create a dorsal osteotomy to mimic an extra-articular distal radius fracture in 10 cadaver arms, five matched pairs that were cut at the proximal one-third of the humerus. The osteotomy was centered 2.5 cm proximal to the dorsal ulnar corner to create a reproducible metaphyseal fracture. An osteotomy was also performed in the ulna to allow ease of dorsal angulation. The median nerve of each arm was then exposed through a longitudinal volar approach with

Fig. 1. Image of the extracted median nerve in custom jig used to stretch the nerve in increments of 0.25 inches (or 0.635 cm).

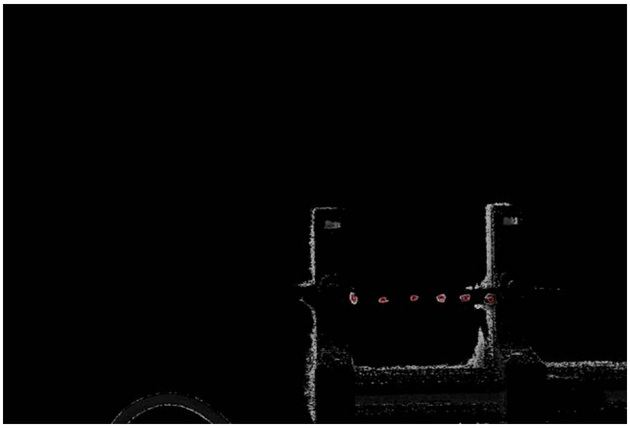

Fig. 2. Image of the median nerve taken from an overhead camera and processed though MATLAB program.

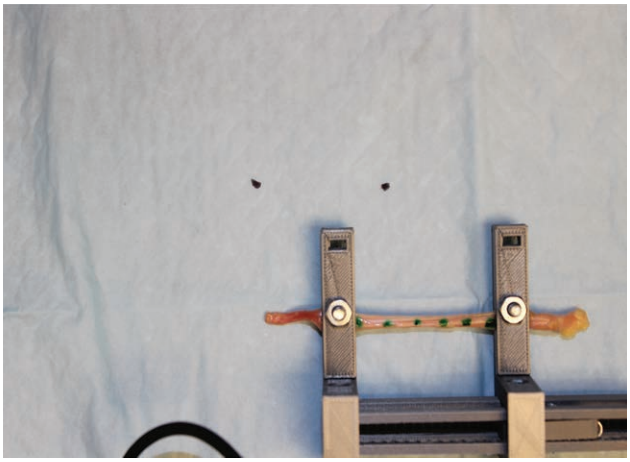

Fig. 3. Original image in Figure 2 before MATLAB processing.

minimal neurolysis, beginning just proximal to the volar wrist crease and extending 10 cm proximally. The transverse carpal ligament was kept intact to minimize volar translation of the median nerve at the wrist. Optical markers were applied to the nerve to facilitate measurement of strain, as described above. Four markers were placed at 1-cm intervals. The arms were mounted into a custom-built 3D-printed jig to allow measurable and reproducible dorsal angulation of the epiphyseal fragment compared with the radial shaft (Fig. 4). The fragments were secured with screws affixed to the epiphyseal and intact diaphyseal pieces, which were then fixed to the jig. Angulation was measured with a digital goniometer that was also attached to the jig to ensure precise measurements (Fig. 5). A physical goniometer was used to verify the angle readout on the digital goniometer affixed to the device. The change in distance between optical markers was recorded via an overhead camera that captured the relative positions of the markers on the nerve at 5-degree intervals increasing from 0 degree to 50 degrees of dorsal angulation. Of note, the position of the markers on the nerve was measured before creation of each osteotomy to use as a reference point, and each median nerve was gently pulled to assess for excursion through the cubital tunnel. A level was used

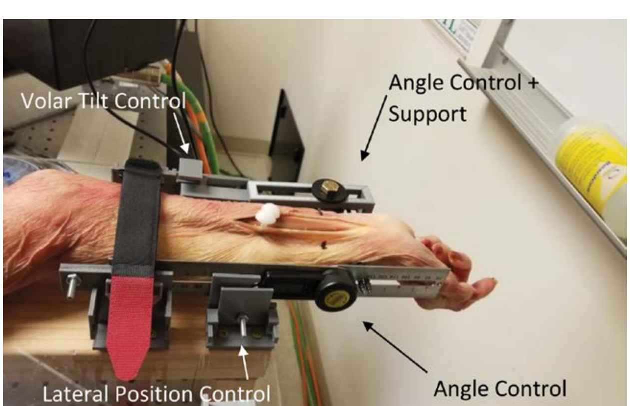

Fig. 4. Cadaver arm placed in custom jig used to fix the artificial DRF at various angles.

Fig. 5. Digital goniometer on the jig, which allowed for tracking of DRF angulation.

to ensure that the screws affixing each fragment remained in plane and parallel to each other to minimize effects of torque or rotation around the screws.

Statistical Analysis

Statistical analysis was performed in R-studio using a linear regression model, a two-way ANOVA, and a two-way random-effects intra-class coefficient model. A linear regression model was found to be the most appropriate to analyze the data in this study due to the comparison of continuous variables. A two-way ANOVA was used to evaluate the interaction of angle and cadaver specimen on strain. An intra-class coefficient was used to further validate the method of strain measurement.

RESULTS

Validation of Strain Measurements

The result of the ex vivo median nerve strain analysis showed a regression coefficient of 1.00067 ($P < 0.0001$) of change in nerve length on measured nerve strain with r^2 = 0.899 (Fig. 6), suggesting a high correlation between the known applied strain and the measured strain. The intra-class correlation coefficient was 0.948 ($P < 0.0001$) with a 95% CI of 0.837–0.985, suggesting excellent agreement between the two methods of strain measurement.[13] These results validated the use of the optical tracking system for measuring strain in the distal radius fracture model.

Effects of Dorsal Angulation on Median Nerve Strain

A linear regression analysis of the effect of increasing dorsal angulation on strain in the osteotomy model yielded a correlation coefficient of −0.000048 ($P = 0.714$), r^2 = 0.00129 (Fig. 7). The effects of each angle on the strain in the arms can be seen in the box plot in Figure 8. The average recorded strain at 5 degrees was 0.5% ± (1.7%) and the recorded strain at 50 degrees was −0.2% ± (2.4%). The median strain value across all specimens and angles was 0.059% and the mean was 0.15% ± (2.1%). The results of a two-way ANOVA analyzing the effect of dorsal

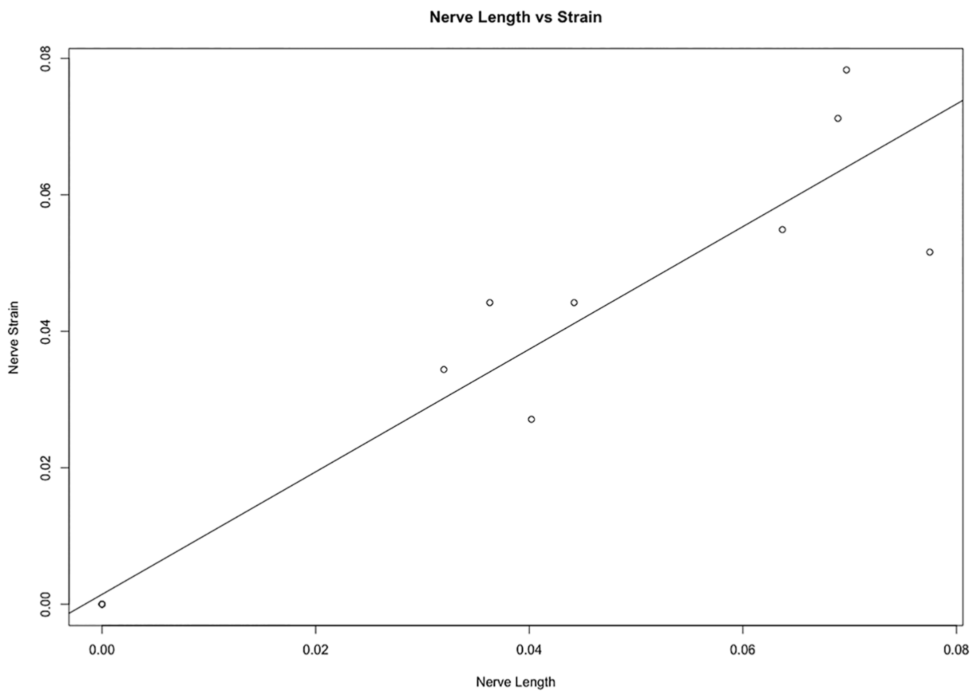

Fig. 6. Scatter plot of nerve length vs. nerve strain in validation study with line of identity.

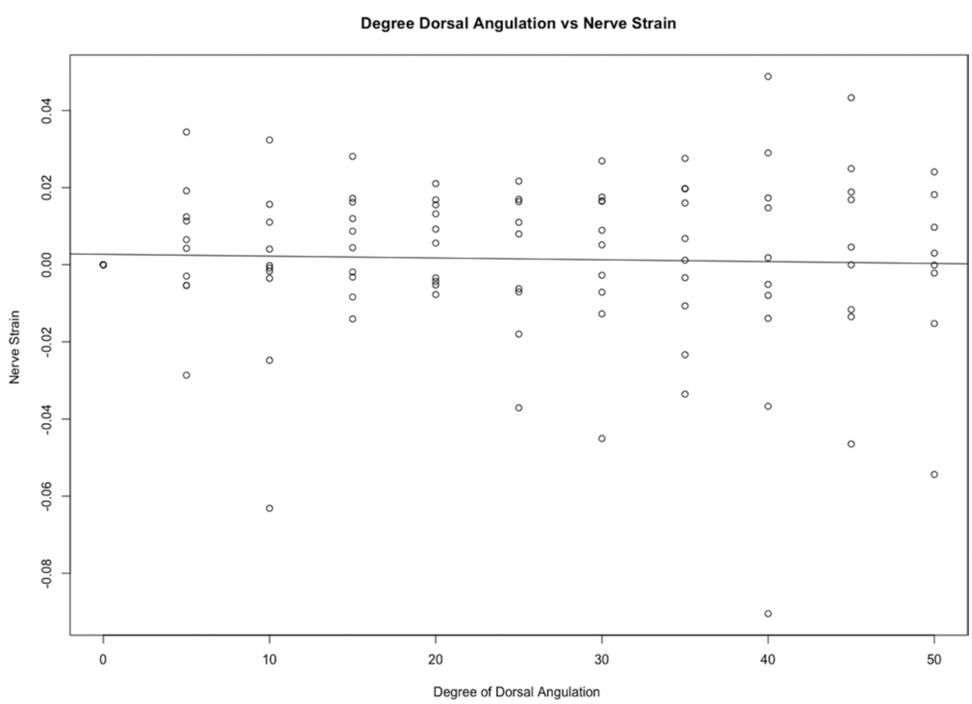

Fig. 7. Median nerve strain vs. DRF dorsal angulation with a line of identity.

angulation on strain in each arm found no significant effect of angulation on strain or specimen on strain. The F statistics were 0.137 ($P = 0.712$) and 1.318 ($P = 0.254$), respectively. Furthermore, there were no significant interaction effects between the two, F = 2.407 ($P = 0.124$). The results of the regression suggest that dorsal angulation has a minimal effect on the strain of the median nerve.

DISCUSSION

The high correlation in the validation study between known median nerve change in length and measured strain by the optical system confirms the accuracy of this tool in measuring strain of nerves. Previous work using this system has evaluated ligament strain as a function of joint kinematics with absolute error in strain measurements

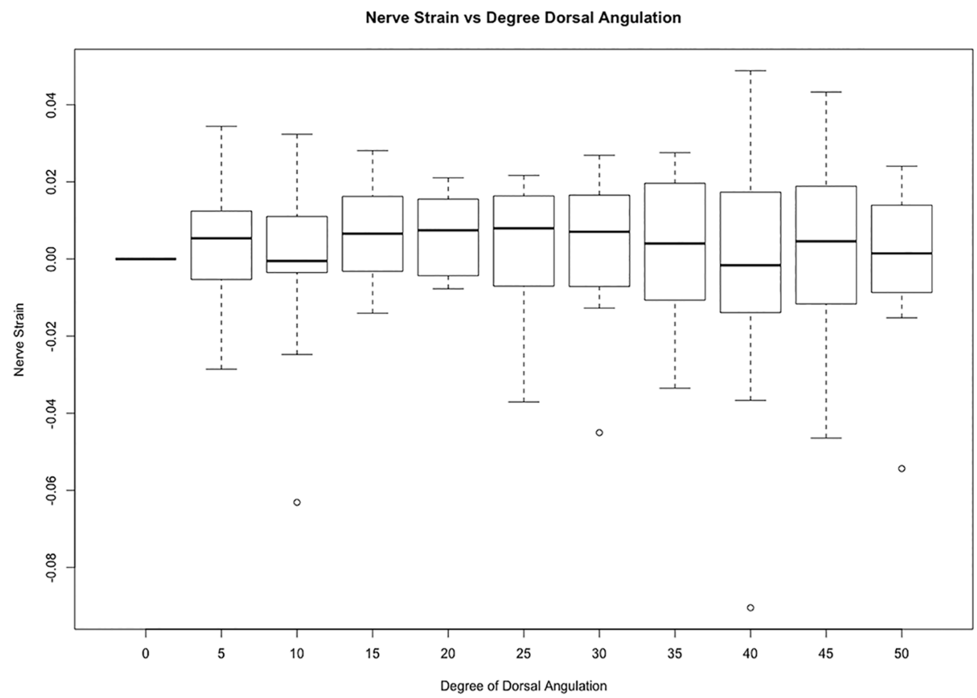

Fig. 8. Box plot of median nerve strain vs. DRF dorsal angulation.

ranging from 0.025% to 0.142%.[14] Video analysis has similarly been used for ex vivo determination of nerve strain, with reported in situ strain of 11% with an SD of 1.5% in a rabbit tibial nerve model.[15] In an in vivo model, increases in strain of as little as 6% led to a reversible 70% drop in compound nerve action potential amplitudes, emphasizing the detrimental effect of strain on nerve function.

A recent work evaluating the histologic effects of rapid stretch injury in a rat sciatic nerve model showed progressive histologic damage correlated to degree of nerve strain.[16] In the elastic region of the stress strain curve, a sustained 12% increase in strain showed no disruption of axonal or endoneural tubes (no evidence of axonotmetic injury). Higher degrees of injury as measured with increasing strain showed a correlation between endoneural tube disruption and nerve stretch. A similar study observing sciatic nerve strain in rabbits found that stretching the sciatic nerve by 2.1 mm a day using external fixation produced a reduction in amplitude, increased latency, and substantial histological damage after 2 weeks.[17] Furthermore, a brachial plexus study in a rabbit model found that once the brachial plexus had an induced strain of 8.1% (± 0.5%), compound muscle action potential was no longer evoked.[18] Therefore, significant increases in strain within a nerve have been shown to result in clinical dysfunction and histologic derangement.

While strain has been shown to be detrimental, the above findings suggest that increases in strain may be minimal in the setting of a distal radius malunion. This suggests that other factors, such as increased pressure within the carpal tunnel, may be the primary cause of median neuropathy in distal radius malunion. Little research currently exists looking at the pathological and physiological changes to the median nerve in the setting of DRF; however, our research affirms the body of evidence that pressure in the carpal tunnel is the major contributing factor to carpal tunnel in the setting of DRFs.[19–22] These results are further bolstered by the fact that in some cases, acute carpal tunnel syndrome can actually be brought on by correcting a residual DRF malunion, suggesting that the absolute angle of the malunion is not the precipitating cause.[23]

The lack of an appreciable increase in nerve strain despite the increasing angulation suggests that the nerve must be able to accommodate significant changes in angular position. Prior work has shown that the median nerve is capable of at least 19.6 mm of excursion through the carpal tunnel with free movement of the wrist alone, 9.2 mm in extension and 10.4 mm in flexion.[24] Therefore, it is likely that any change in the position of the median nerve in the forearm, even in the setting of extreme DRF malunion, can be accommodated by increased excursion through the carpal tunnel.

The implications of this research on the treatment of DRF and related neuropathy in a clinical setting are numerous. The above findings support the clinical practice of carpal tunnel release for patients with nascent distal radius malunion, and suggest that osteotomy may not be necessary to relieve median nerve symptoms. Similarly, significant dorsal angulation at the time of presentation should not be a factor influencing the decision for prophylactic carpal tunnel release in a patient treated with open reduction and internal fixation of a distal radius. Instead, the overall energy of the injury, as determined by translation of the fracture fragments and soft tissue swelling, may be better predictors of pressure increases in the carpal tunnel and potential for acute carpal tunnel syndrome. In fact, a retrospective clinical analysis of factors leading to acute carpal tunnel syndrome after distal radius fractures identified fracture translation (and not dorsal tilt) as the only factor predictive of an increased chance of developing acute carpal tunnel syndrome.[25]

Our study is not without limitations; chief among these is that it was performed in a cadaver model, which limited our ability to measure pressure within the carpal tunnel, as this would likely be significantly affected by perfusion and soft tissue swelling. However, given that our study is primarily concerned with indications for treatment in delayed onset carpal tunnel rather than acute carpal tunnel, it is less likely that soft tissue swelling is a major contributing factor to nerve pathology months after the injury. Similarly, cadaveric nerve tissue may behave differently than living tissue; therefore further work could be done using in vivo imaging methods in the clinical setting, likely with either diffusion tensor weighted imaging or ultrasound.[26,27]

Another limitation is that the osteotomy was made through both the distal radius and ulna, more consistent with the less common distal both bone fracture, and not a simple distal radius with ulnar styloid fracture.[28] The decision was made to perform the osteotomy through the ulna to allow creation of a more severe angulation of the distal radius metaphysis, and eliminate potential tethering of the soft tissue from an intact ulna. However, the current model likely represents a worst-case scenario with regard to median nerve strain, and it would be expected that an intact ulna would be further protective for the nerve. Similarly, the fracture was created with an oscillating saw via an open wedge osteotomy, and does not account for possible direct mass effect of displaced fracture fragments. Although relatively rare clinically, the astute clinician should be attuned to this possibility, particularly in the setting of patients presenting with evidence of acute carpal tunnel syndrome.

Furthermore, it has been demonstrated that change in wrist and finger flexion can affect strain on the median nerve.[24] In the present study, the wrists and fingers were left at resting position and were not artificially extended or flexed. It is possible that volitional extension of the wrist or fingers in an active patient could produce strain on the median nerve beyond what was seen in this study. Further research needs to be performed to establish how wrist and finger position affects median nerve strain in the setting of a distal radius fracture.

Finally, the median nerve was not fixed proximally due to concern for creating nonphysiologic strain, as the anatomic length tension relationship was unknown. To assess for the need to anchor the median nerve proximally with sutures, we exposed the nerve at its most proximal point and flexed and extended the elbow, observing for any movement in the proximal median nerve. In a cadaver arm used to test the apparatus, traction was applied to the median nerve near the wrist with no resultant observable motion of the nerve at the proximal humerus osteotomy. However, if we failed to observe any extant micromotion of the nerve, it is possible it influenced the strain measurement distally.

Additional research is warranted to evaluate the change in carpal tunnel pressure and median nerve strain as well as excursion with increasing dorsal angulation of DRF malunions. Furthermore, the degree of strain required to cause histologic changes to the median nerve in a human model is unknown, at present, and requires further study, as does the effect of age on median nerve elasticity. The results of our study taken in the context of the existing research suggest that the pathological changes which predispose patients with distal radius malunions to carpal tunnel syndrome are not likely due to increased dorsal tilt alone. A better understanding of the effects of fracture displacement, severity, and soft tissue damage could help inform clinicians when making operative decisions about surgical treatment of distal radius fractures.

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