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Robert Curran
CUNY Brooklyn College

Sasha Sorr
CUNY Brooklyn College

Eva Aquino
CUNY Brooklyn College

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Potential wrist ligament injury in rescuers performing cardiopulmonary resuscitation

Robert Curran, Sasha Sorr, and Eva Aquino

Department of Exercise Science, Brooklyn College of the City University of New York, New York, United States
Address for correspondence: Dr. Robert Curran, E-mail: rcurran@brooklyn.cuny.edu

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Abstract

Wrist pain in rescuers performing chest compressions as part of cardiopulmonary resuscitation has been reported anecdotally and recently in the literature. Studies have indicated that rescuers apply as much as 644 N of force to the victim's chest with each compression, while standards require one hundred compressions per minute. Recent research suggests that forces transmitted through the rescuers' wrists of less than 10% of those seen during the performance of chest compressions significantly strain the scapholunate ligament. Biomechanical research should be performed to further evaluate this possible correlation. Compensation for worker injury maybe involved.

Keywords: Cardiopulmonary resuscitation, Emergency Medical Services, resuscitation

INTRODUCTION

Given the nature of Emergency Medical Services (EMS) work, it is not surprising that musculoskeletal injuries are the leading cause of reported morbidity. Occupationally induced musculoskeletal injuries are an inherent hazard of the EMS profession.[1] Wrist pain in rescuers performing chest compressions as part of cardiopulmonary resuscitation has been reported anecdotally and recently in the literature.[2] Studies have indicated that rescuers apply as much as 644 N of force to the victim's chest with each compression, while standards require one hundred compressions per minute.[3] Recent research suggests that forces transmitted through the rescuers' wrists of less than 10% of those seen during the performance of chest compressions significantly strains the scapholunate ligament.[4] The combination of this evidence suggests that chest compressions as performed during CPR may cause damage to the scapholunate ligament of the rescuers' wrist. Biomechanical research should be performed to further evaluate this possible correlation.

DESCRIPTION

Cardiopulmonary resuscitation for treatment of persons in cardiac arrest involves the administration of external chest compressions. Compressions are administered by placing the heel of one hand in the middle of the victim's chest, then placing the other hand on top of the first, interlacing the fingers. The rescuer must compress the chest of an adult about 1-1/2 to 2 inches (4-5 cm), with recent studies indicating that compressions of greater than two inches increased survival.[5] The chest is allowed to fully recoil before the next compression. This process is repeated at a rate of 100 times per minute. The position of the rescuer is most commonly full dorsiflexion to hyperextension of the wrist and hand in contact with the chest of the victim, which is being compressed by the rescuer's second dorsiflexed to hyperextended wrist/hand on top of it. The bottom wrist and hand then bears the body weight of the rescuer attempting each compression. A similar position, in which the result of a fall on the locked hand to the thenar eminence with the hand in extension ulnar deviation and the forearm pronated usually leads to a motive of injury in which the wrist is positioned in extension ulnar deviation and intercarpal supination. This type of injury often involves the rupture of both dorsal and palmar portions of the SL.[6] This injury pattern leads the capitate bone to protrude violently into the SL articular space.[7] Tension on the palmar aspect of the hand and compression on the dorsal is developed. This component of the dorsal compression leads the capitate into the SL cleft, acting as a mortar thrusting into the SL gap stretching and rupturing the portions of the ligament at a given angular position of the two bones.[6] Mayfield, *et al*. reported a laboratory study in which cadaveric wrists and forearms were used to demonstrate injury to the scapholunate interval. Frozen cadaveric specimens were used in simulating a fall on the outstretched hand causing wrist extension, ulnar deviation, and intercarpal supination. Under these loading conditions, the scapholunate joint was the first carpal joint to be injured. Ligamentous jury then progressed around the lunate to produce sequential instability of the scapholunate, capitolunate, and triquetrolunate.[8]

Anectdotally, rescuers report wrist pain after performing chest compressions. Trowbridge, *et al*. documented that all participants in a CPR research study reported wrist pain after performing compressions noting an association between the decline in compressions of adequate depth and wrist pain.[2] Several of the research participants had to stop compressions due to wrist pain. This is supported by as yet unpublished research by Curran and Dunbar. Of the numerous possible causes for wrist pain in the rescuer performing chest compressions during CPR, subfailure injury to the scapholunate ligament has not been previously proposed in this population and there is no evidence in the literature of research in this topic.

The intrinsic aspect of the SL ligament complex consists of the scapholunate interosseous ligament which connects the radial side of the lunate to the ulnar side of the scaphoid.[9] The ligament is comprised of 3 parts: An avascular, mostly fibrocartilagenous or membranous proximal portion along with 2 “true” ligaments, one dorsal and one volar.[10] The dorsal portion of the SL ligament is thicker than the volar portion, with transversely oriented collagen fibers. These dorsal fibers have been shown to be important in stabilizing SL translation, whereas the more obliquely oriented volar fibers are more critical in constraining rotation. When the SL ligament complex is torn, the scaphoid has a tendency to flex and the lunate has a tendency to extend, assuming a dorsal intercalated segmental instability (DISI) pattern, an observation that is supported both by *in vitro* and clinical data. Acute SL dissociation is the most commonly recognized and treated pattern of carpal instability. It usually occurs after a fall on an outstretched hand, axial loading, with the forearm in pronation and the wrist hyperextended. If the SL ligament is disrupted, the scaphoid and lunate may be rendered translationally and rotationally unstable, leading to a widened SL gap, as well as an increased SL angle.[10]

Microtrauma or subfailure injury in tendon and ligament may occur either as the result of overuse or as a

single traumatic event,[11] tendon and ligament microtrauma and partial tears may accumulate damage to the point that load bearing is compromised and complete rupture or secondary damage occurs.[12] In addition, ligament microtrauma may result in increased laxity, which in turn is associated with degenerative joint disease and osteoarthritis.[13] These studies suggest the importance of studying subfailure injury and intrinsic healing of ligaments. Ligament microtrauma may result in increased laxity, which in turn is associated with degenerative joint disease and osteoarthritis.[13] These studies suggest the importance of studying subfailure injury and intrinsic healing of ligaments. In one study of rabbit anterior cruciate ligaments, subfailure injury (~80% of failure stretch) altered the shape of loading curves and thereby increased joint laxity.[14]

Lee designed the first *in vivo* biomechanical study which compared different wrist positions with load application to determine the resultant effect on SL ligament strain by directly measuring the ligaments. Using MRI, the effect of wrist position on the strain imposed on individual sub regions of the SLIL during axial load was studied in both a neutral position and at approximately 90° extension. A previous study indicated that the force applied to each wrist during a pushup was calculated to be approximately 38% of body weight. Because subjects were unable to endure such forces for the extended period of time necessary to complete MRI imaging, only 10% of this force (25-35 N) was applied to the wrist during the actual test. Force was applied to the wrist via extension of an elastic band. The length of the band was measured before and after elongation and a previously determined force/elongation curve was used to determine the applied force. Six male volunteers without history of prior wrist injury or reports of wrist pain each underwent 3 MRI scans,[1] high-resolution scan in a neutral position with no applied force, and[2] lower-resolution scans in which the rig applied a force to the wrist in neutral and in maximal extension. The mean percent elongation in loaded extension was 48.4% for the palmar component and 4.8% for the dorsal component of the SLIL in Lee's study. The values obtained for ligamentous strain in neutral versus extension demonstrate that for a given axial load, theoretically, a greater degree of force is transmitted to the SL interval with the wrist in extension. In addition, while being loaded in extension, this force is distributed to preferentially cause lengthening of the proximal and palmar components of the SL ligament while the dorsal portion remains virtually unchanged.[4]

Chest compliance studies have shown that the force-depression relationship for a typical adult male is almost linear with a slight hysterisis.[15] For a chest depression of 1.5 inches it requires about 100 pounds, for 2 inches it requires about 125 pounds.[16] Baubin demonstrated that volunteers used approximately 644 N of force to compress a manikin chest the recommended 1.5. to 2 inches, though they calculated that a force of 411 and 548 N would be applied.[3] Tomlinson, *et al*. demonstrated that in most out-of-hospital cardiac arrests, adequate chest compression depth can be achieved by applying 50 kg force (490 N) to the sternum.[17] A common automatic chest compression device on the market applies approximately 50kg of force to achieve chest compression.[18] Chi, *et al*. study on CPR kinematics investigating 18 health care providers noted sternal compression forces of approximately 455 N, depending on the position of the rescuer, to achieve compression depths of between 42 and 44 mm.[19] Gruben reported a mean of 431 ± 40 N in 16 cardiac arrest patients.[20] In their study 38 mm was not reached in 9 of the 16 patients whose values were calculated by extrapolation. Chest wall elasticity varied greatly between individuals with a range in force from 10 to 54 kg in patients where 38 mm depth was achieved. Gruben *et al*. also found a large range from 245 to estimated 800 N for 38 mm, and estimated that >700 N would have been required in 5 of the 16 patients. The authors were concerned that it would be difficult, let alone unrealistic, for the average rescuer to achieve even the minimum recommended 38 mm compression depth during CPR.[20] The loads required to cause failure of the scapholunate ligament vary with the strain rate from as low as 75

N to approximately 359 ± 110 N.[21] For the SL the ultimate load to failure ranges generally from 170 to 260 N. 22-24 Berger obtained for the dorsal portion of the S-L a maximum load of 300 N, with the palmar portion 150 N and the membranous (proximal) portion 25-50 N giving in a composite of 500 N for the entire ligament.[22] Logan *et al*.[13] obtained 125 N for the S-L palmar portion and 62 N for the dorsal, giving at least 187 N for the whole S-L ligament.[23]

CONCLUSION

This article proposes that the forces transmitted through the rescuers' wrists in the performance of external chest compressions during CPR are suffice to cause injury to the scapholunate ligament of the rescuer, potentially resulting in further cumulative trauma, degenerative changes, and eventual disability. Further biomechanical studies specific to this particular population should be performed.

Footnotes

Source of Support: Nil.

Conflict of Interest: None declared.

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Publications