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Elbow Stiffness: Biological and Neurological Considerations Recorded February 4, 2020

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- [Fawn] Okay, today's webinar is Elbow Stiffness: Biological and Neurological Considerations. Our presenter today is Paul Bonzani. He is an Assistant Clinical Professor in the Department of Occupational Therapy at the University of New Hampshire. He teaches the human movement and adult development courses in the required curricula in elective courses in upper extremity rehabilitation, orthotic fabrication and physical agent modalities. Additionally, he co-teaches a course on the psychobiological manifestations and management of the human stress response. He obtained his CHT credential in 1991 and continues to practice in addition to his educational responsibilities. Finally, Paul has authored multiple textbook chapters and review articles on varied upper extremity rehabilitation topics including functional anatomy, tendinopathy management, elbow rehabilitation and mobilization orthoses. Welcome to Paul. We're so happy to have you.
- [Paul] Okay hello, everyone. Thank you for spending time with us this afternoon. We're going to spend the next 50 minutes or so talking about the elbow and about why it gets stiff, what are the biological and the biomechanical and neurological considerations of elbow stiffness. So let's go right to that. Let's think of the learning outcomes for this course. We're gonna be able to, you should be able to understand the physiological timing of contracture development. You'll be able to list neurophysiological occupation-based phenomena associated with the development of elbow stiffness. And then we will discuss some preventative strategies, but the emphasis of this class is not on preventative strategies. It's really on assessment and the development and we'll touch a bit on what we can do with management particularly of the neurophysiological components of elbow stiffness. So the biggest, the question we all have to ask is, why do elbows get so stiff? And that's the question we always ask and we're always told it's because of the elbow joint's congruency. So that seems to be the answer. It's a congruent hinge joint and therefore it gets stiff. But



the question you have to ask yourself is, does it really get stiff in both directions? Or does it only get stiff in extension? Does it get stiff in supination and pronation? Or does it really predominantly only get stiff into supination? And the answer is we know as clinicians is that the elbow tends to get stiff in extension. We develop flexion contractures and we develop loss of supination or pronation contracture. So the question is why does it happen only in one direction? And the bottom line of that is the elbow is built to flex. I'm gonna show you a couple of biomechanical features to show you why the elbow is prone to getting stiffness in the manner that it presents to us.

So the first one of these, I beg your pardon, the first one of these is to take a look at the distal humerus. And why do we start the distal humerus? The distal humerus is the biomechanical foundation for the elbow joint. The trochlea and capitulum are convex surfaces that create a stable platform by which the forearm bones, the ulna and the radius, rotate around these particular stable features. You'll notice that there's a little bit of a groove here in the middle of the trochlea. Let me take this little, here's my little arrow here. And you can see that there's a little groove right here which we call the groove for the sigmoid notch. This creates a tongue and groove type of arrangement for humeral olecranon or the humeral ulna joint. What happens is that creates a congruent joint with some stability in medial and lateral movements. We also see a relatively shallow fossa here. Let's move that right to this area. That fossa is the coronoid fossa. The coronoid fossa is the seating point for the coronoid process, a very important area of the elbow, and underrated in its importance. The coronoid fossa and the coronoid process creates stability in the elbow when the elbow is fully extended. If there's any injury in this particular area, the elbow can become unstable or it can respond by developing osteophytic formation in this area and that will prevent the elbow from flexing fully. So this area of coronoid and coronoid fossa very important in trauma patients. Another area we see here is the trochlea-capitulum groove. This is basically the area where the radial head will approximate when the elbow is in full flexion.



Let's move to our next slide. We look at the elbow from a posterior perspective, we see two important anatomic features. The first one is the deep olecranon fossa, and this is for the seating of the olecranon process. Now the olecranon fossa and the olecranon process are quite deep and they lock the elbow when the elbow's in full extension. But what the important thing to remember about this process is that this process is less involved in flexion contracture. Many times we think about the posterior component of the elbow as developing or being concerned with the loss of extension movement, and that's not typically what happens. Typically, the loss of extension happens with anterior injury, not posterior injury. The little area we see here is the sulcus for the ulnar notch. And this is basically the ulnar, this protects the ulnar nerve, and keeps the ulnar nerve in a posterior area. We also see a large medial epicondyle and a thinner lateral epicondyle. The larger medial epicondyle is actually a protective hood for the ulnar nerve and it gives the ulnar nerve a pulley by which to move from a posterior to a medial position in the forearm.

Okay, so why are elbows then built to flex? Why do I say they're built to flex? The main reason is because they have a 30 degree anteversion angle which you see here. The articular surface is not in a direct line with the metathesis of the humerus. The articular surface is anteverted 30 degrees. This is a very important skeletal feature in elbow stability and elbow function. What this really does is it takes the line of the musculature and it removes that line from a close proximity to the humerus. As we'll see a little bit later, what this does is it increases the moment arm, allowing for the elbow to flex efficiently. What it also does is it decreases the moment arm for the triceps, and disadvantages the tricep, and we'll talk a little bit about that. And you can see that right here. Where's my arrow? There we are. So here is that straight line. And here is that 30 degree angle out in the anterior plane, anterior to the sagittal plane. Okay. The next thing elbows are built to do is they're built to be in valgus. So why would an elbow be in valgus? Why is it important? After all, it's a simple hinge joint. Why should it be



inclined to valgus? Why does it form this carrying angle? Well, you'll notice a couple of points here. The carrying angle, the important point here to consider is this one, is that the difference between the male and female anatomy. The reason why that's an important point is because it's why we have a carrying angle. The carrying angle is larger in females and smaller in males simply because the upper extremity has to sit in valgus to allow for adequate arm swing during ambulation. Now we're going to see the importance of this in clinical assessment at the end of this presentation, but this is an important point what you do remember. This is a normal component of the elbow. It means that the elbow axis is not transverse. It's actually oblique.

Let's take a look at that, what I mean by that. So let's look here. In this depiction we can see that our axis of rotation is actually inclined. It's an oblique axis of rotation. It's not transverse. A formal hinge joint that did not have a valgus angulation would have their axis of rotation in a straight line. This would be similar to the knee. The knee is about as close as you get to a pure hinging movement from an axis of rotation, or anyway, closer than it is at the elbow. The elbow has this six to eight degree angulation between its actual axis of rotation in the epicondyles. We also see here another important component of valgus orientation which is a recession of the radial head. Now the recession of the radial head is important to consider for force transmission through the elbow. It's an injury prevention mechanism. The elbow does not sit congruent, the radial head, rather, does not sit congruently to the capitulum. This also allows for valgus as there's a space here that the ulna can slip into without bone-to-bone compression. When we move into varus which is movements towards the medial side, we have bone-to-bone compression from the end of the ulna into the trochlea here. And so we have much less valgus movement than we have varus movement, sorry, the other way around. Much less varus movement than we have valgus movement, and our end-feels are different. What we see here is the inclination angle of the trochlea. So the trochlea here has a six to eight degree inclination angle for its articular surface, and what that means is the elbow is built to sit in valgus. It's an important consideration



because valgus positioning of the elbow is necessary for arm swing. You'll notice that as you see here, let's go back a little bit here, we could see a varying difference between males and females. Males with smaller pelvises require less valgus clearance at the hip and therefore the adjustment of the upper limb to the lower body and to the lower hip, into the hip, creates less of a need for a valgus angle. In the female skeleton the valgus angle is increased because of the hip being wider for childbearing considerations. We can see that anatomically here and we can also see that in the relevant radial head anatomy which is here. So you can see that the radial head is also inclined into valgus about 15 degrees and that allows for clearance of the radial head and allows for different end-feel between varus and valgus maneuvers of the elbow.

The varus end-feel is bone-to-bone and therefore it feels hard and bony. The valgus end-feel is springy or capsular in nature because it's actually a ligamentous constraint. If we look here on to the ulna, we could see that the matching joint here of the ulna has an angle between the coronoid process here and the olecranon process and this is a retroversion angle of about 30 degrees. Further, there is a rigid line here that matches the ridge on the trochlea giving congruency of the elbow joint. So we have important processes for stability, coronoid in extension, olecranon in flexion. Now the olecranon process is a little less important for flexion stability. We can fracture quite a bit of our olecranon without any loss of stabilization into flexion. But if we fracture 1/3 of the coronoid or more, the elbow becomes unstable and the person needs open reduction internal fixation of the coronoid fracture. So this is quite a bit more important in our discussion of stiffness, because injuries in the anterior surface create a great deal of elbow stiffness, particularly around the coronoid. The axis of rotation into flexion into supination and pronation is a longitudinal axis. And that axis actually comes between the superior radioulnar joint and the distal radioulnar joint. We have a complex arrangement of anatomy here. The forearm rotation actually becomes less stiff at the elbow from pericapsular problems. We'll see that in a minute on when I start talking about the difference between pericapsular versus skeletal problems. The axis of



rotation, the limitations, rather, of rotation in supination and pronation are more inclined to be related to what's called synostosis or actual growing together or bony linkages between the radius and the ulna at the superior radioulnar joint. So let's take a look at this movie here on the mechanics of flexion/extension. We'll look at the differences of flexion/extension and pronation and supination.

And let's start with our little animation movie right here. Let me pull this back, okay. What I want you to think about and what I want you to notice here is the beginning of the movement into flexion. Notice the muscles here, the brachialis, here, the bicep, here, the pronator. Here's the the tricep and anconeus. Let's now focus our function, our attention, rather, on the brachialis and on the bicep. You'll notice that the bicep tendon is away from the, is away from the joint axis of rotation. There we go. So here's the axis of rotation. Here's the bicep tendon. And if you notice that at full extension and you can see right there at full extension, the bicep tendon is still away from this axis of rotation. Now that creates a moment arm that causes the elbow to be able to initiate flexion effectively and that only happens because of that anterior reversion angle of the trochlea. You'll notice that as the elbow begins to flex that that moment arm increases.

Let's run that again. I'll stop the video right where I want it. Right there and you'll notice that right here, the moment arm is actually quite big. The distance between the axis of rotation and the tendon insertion is actually quite large here. That makes the elbow very powerful in flexion, but look where it's at its greatest strength. It's at its greatest strength right there at about 70 degrees of flexion. So right there. Right there to right there. And then the moment arm begins to decrease end of range into full flexion and in full extension. What it means this is that the muscles are best inclined to work in that mid range area and that's where we get locked into our stiffness pattern. Now let's look at the tricep for a moment. Let's run this again, and look at the tricep initiation of movement. The tricep initiates movement in full flexion. Notice that the tendon is completely wrapped around the axis of rotation. The tendon here is extremely



mechanically disadvantaged. So when the elbow is trying to extend fully to initiate movement, the tricep is very disadvantaged. It's assisted by gravity fortunately in eccentric control of the bicep. But it's still very mechanically disadvantaged when we're initiating movement. However, for our stiffness discussion take a look at this. It's also mechanically disadvantaged right here at about 30 degrees of flexion. It's extremely mechanically disadvantaged. There's no distance to the moment arm right here at all. Therefore, we're trying to use a pretty strong muscle to overcome elbow stiffness, but we can't, because it's mechanically disadvantaged. I'll come back to this and show this a little bit more when we talk about the neurological considerations and co-contractions at the end of the presentation.

Okay, could we go to the next video? Now looking at this next video, we're looking at the the arc of rotation of pronation and supination, and we're looking at the muscle activation of pronation and supination. In this case, what do we see for pronation? We see a muscle that's crossing the joint directly. And we see a muscle that's actually, this is the pronator teres, and it's directing its force obliquely across a nice line of pull and therefore creating a strong mechanical advantage and a good biomechanically advantaged moment arm. But if we look at supination, where does supination initiate from? Supination initiates here from the bicep. You can see that the supinator muscle, which is the assister, an assisting muscle, but is also a very small and mechanically disadvantaged muscle. The bicep line of pull is mechanically disadvantaged for supination. So although the bicep is a prime supinator, we can see that the bicep compared to the pronator is actually a weaker supinator than the pronator is a weaker pronator. Your elbow is built for pronation, because function occurs in pronation. And the other feature of the bicep is the bicep is a bi-joint muscle. What do I mean by that? Well, the bi-joint muscle, the elbow has a long head, the bicep, rather, has a long head which loses force to the shoulder. So this compromised moment arm, I'll run this through one more time, and the distance from the joint insertion makes the bicep compromised for supination and the pronator advantaged for pronation.



Okay, can we go to our next slide? Okay. So what I was hoping to get from that is you could see that biomechanically the elbow does exactly what it's supposed to do when it gets injured. It maintains its flexion. It maintains its pronation, but it loses its supination, and it loses its extension. Now let's talk a little bit about osteokinematic and functional movement. So osteokinematic, as you see here, this is the actual ranges of motion of the elbow for flexion/extension, supination and pronation. But we also notice that there's a given functional extension range and a functional pronation/supination range.

So the goals in therapy are always to gain 30 degrees of extension to 130 degrees of flexion, and 50 degrees of pronation and 50 degrees of supination. The reason for that is those are the degrees of movement that the elbow needs to attain to perform basic ADL tasks. This comes from a number of studies, Morrey studies and biomechanical studies using electrogoniometers and assessing movement. So this is what's the accepted range of movement for good outcomes in elbow rehabilitation.

All right, now we need to look at another component of holding the elbow together, because this is a component that creates stiffness also in the elbow and that is the medial ligaments. Now the media ligaments are a source of stability, as I said before, to valgus forces. We call them the ulnar collateral ligament or the medial collateral ligament, most commonly, ulnar collateral ligament. They are three functional regions. This is their outline: anterior bundle, posterior bundle, and there's what's called cooper's or transverse bundle. Let's work off the model and I'll be able to show you what this looks like. So in this model we can see here that the purple arrow, the purple arrow is our anterior bundle. The red arrow is the confluence point between the anterior bundle and the posterior bundle. And the green arrow is the posterior bundle. Now the way ligaments work is in flexion at about 30 degrees of flexion the anterior bundle here becomes the main constraint to valgus. At about 90 degrees of flexion this confluence



bundle is the main constraint of valgus. And after 90 degrees of flexion, the posterior bundle becomes the constraint to valgus. Now what happens when the elbow is injured and these ligaments are not involved in the injury process, but they are strained or they become partially injured, say, in a dislocation or in a fracture, you get swelling and then pericapsular adhesion that actually involves this medial ligamentous complex. And the part that gets most involved is the part here in this anterior bundle. This bundle shortens and therefore when the elbow tries to extend, this bundle becomes a secondary constraint component of flexion contracture. So that's our main discussion about these ligaments. These are, of course, a main source of sports-related injury at the elbow. But in our discussion today of stiffness it is this anterior bundle that's the most important to consider.

This is what it looks like on cadaver just so that you can see it in a cadaver model. This is the anterior bundle right here. So can you imagine if this is injured, this shortens and then assists the capsular contraction in restricting extension. So that's the contribution of the ligaments on the medial side. On the lateral side, the ligaments tend not to contribute so much to the flexion contracture. But there is one component we must talk about in injury and that is this component right here, the lateral ulnar collateral ligament. So let's pull up our model here. I'm gonna leave that arrow up, and pull our model up here, and then go through the lateral ligaments and their contribution to stiffness.

This is the primary and accessory lateral collateral ligament. And you could see that its main constraint is to the radial head. This is the annular ligament, and it's a 270 degree fibro-osseous ring that constrains the radial head in the superior radioulnar joint, and its primary role is to stabilize the radial head. However, here's a very interesting subset of this lateral complex and that is the lateral ulnar collateral ligament, which you see right here. Now this collateral ligament, very important, because when you have injury of the radial head, this ligament is sometimes injured as part of radial head pathology.



You can see that this ligament, if it were to shorten, it would act as another constraint of the ulna attaining full extension. Further, if this injury is, if the injury extends to a complete ligamentous tear, the radial head becomes unstable and the ulna becomes unstable on the humerus, and therefore we have an increase in varus forces. So that's what this does. This controls varus. There's a complex injury here called posterior lateral instability of the ulna. That happens when this ligament is completely torn. I'll show you a case of that next in the follow-up. When we talk about treatment of this problem, we'll look at a case where stiffness develops secondary to that injury.

This is what it looks like in cadaver. This is the complex here. So here's your radial lateral collateral ligament complex. It's hard to discern the lateral collateral, the ulnar collateral, collateral ligament here. However, it is in that constellation. That's why sometimes the model is a better structure to look at to study the individual bundles. Here's the annular ligament here in green. And this area right here is the anterior capsule, which we're going to get to next. So again, here's your function of the collateral ligaments, varus and valgus stability, and then this is that injury I was telling you about before, the loss of the lateral collateral ligament associated with ulnar external rotation posterior lateral instability problems.

The final structure we need to consider for the development of contracture biomechanically is the joint capsule. Now we're gonna talk about the joint capsule and the physiology in just a minute, but the issue here is the anterior capsule of the elbow. The anterior capsule is a thin, basically, transparent, translucent structure. It's lax at 60 to 80 degrees of flexion, which corresponds to the open pack position of the elbow, which is 70 degrees of flexion and 10 degrees of supination. It is the main soft-tissue structure that's implicated in contracture following injury. Let's look at this structure and you can see why. The capsule is redundant. It crosses a great volume. It is a large volume. It crosses the anterior surface of the elbow. It becomes redundant in 70 to 80 degrees of flexion, meaning its largest volume. So therefore, injuries that hold the



elbow in a flexion position end up causing this contracture, this capsule, rather, to shorten and create the elbow flexion contracture mechanism. You'll also notice that you can see slightly if you look on the sides that that medial collateral ligament also has contributions to the capsule as does the lateral collateral ligament here as you saw on the cadaver, and therefore ligamentous involvement can have a co-causation of contracture with capsular changes. Here, this is the annular ligament here and the capsule kind of underneath it.

So here's your anterior capsule supinator and your anterior view of the elbow. You'll notice now in the posterior capsule we have a very different presentation. The posterior capsule does not extend over the olecranon. It does not become redundant. It actually folds underneath the olecranon. And therefore, this is one of the reasons why when the posterior capsule is injured, it doesn't stiffen very much. It doesn't shorten very much, because it's not redundant across the olecranon here. And therefore, stiffness and deflection is not very difficult to remediate and is also does not have, usually, a significant capsular component.

When we talk about forearm rotation, I mentioned this briefly, it's the integration of superior radio- and inferior radioulnar joints. We have to discuss the interosseous membrane, because the interosseous membrane becomes involved in stability of the forearm. So let's look at that. So our final bit of anatomy to consider is interosseous membrane. The interosseous membrane is a fibro-osseous structure that creates a syndesmosis joint here at the forearm. What it does is it links the humerus, I'm sorry, I beg your pardon, it links the ulna to the radius. There's a strong component or biomechanical function, rather, of this ligament is to maintain this arrangement when the forearm bears load. So its basic function is to disperse axial loading through the hand over the radius, so that the radius is unloaded from the wrist by the time it reaches the elbow. So a classic example I like to give in my classes is if I bear weight on my arm and I weigh 100 pounds, the 100 pounds of axial force going through the



wrist is divided into an 80/20 relationship with 80% of the force going to the radius and 20% of the force going to the ulna. As that force transmits axially, the interosseous membrane stops the radius from proximal transmission, translation, rather, and holds the radius in place, and in doing so, it transmits force from the radius to the ulna, so that force transmission at the ulna starts at 80/20, but by the time it hits the elbow, it's 60/40. 20% of loads are unloaded off the radius and loaded onto the ulna. This is very important for injury prevention mechanisms. When this is injured in pathology, we get instability of the radius and that's actually quite a significant injury. That's typically associated with radial head fracture or significant dislocation severe Monteggia fractures where the ulnar is fractured, the radius has fractured, dislocation of the radial head and that can extend into an Essex-Lopresti lesion right here into the interosseous membrane. That does create a great deal of stiffness. The area I'm talking about is here. And so if I have injury and dislocation here, injury and dislocation here, these bones can come apart and tear this ligament here, and that's called Essex-Lopresti and that's quite a severe injury of the radius, of the elbow, rather with significant stiffness in the postoperative course. So there's your mechanical reasons for developing contracture.

Now why do we get physiological reasons for stiffness? Well, let's get to it. There's two types of contractures that develop physiologically, One is the intrinsic contracture. This is pretty straightforward. This is when we have instability creating loose bodies in joint arrangements and dislocations, et cetera, small subluxations that create shearing forces in the joint. The most common type of contracture development, however, is extrinsic contracture, and that's typically soft-tissue contracture or exostosis, which I mentioned, which can happen between the radius and the ulna, or it can happen at the radius and affect flexion. Let's talk about that. The intrinsic contracture here is typically associated with a high force repetitive motion injury. It can be related to loose body and we typically take, in orthopedics, loose bodies are excised. The contracture occurs in this region right here, in this area in the capsule. It happens when loose bodies block



either flexion or extension, and then we need to excise the loose bodies, or the surgeon needs to excise them. Plicas are when the synovium here or here, typically posteriorly, thicken, so the synovial lining here thickens. And the plicas need to be excised to create freedom of movement. These intrinsic contractures are typically limited to sports medicine practices and they are in high force repetitive athletes. The more common contracture, however, as I described, is pericapsular or exostosis. That contracture starts, this one common one that we see here is called heterotrophic bone. Heterotrophic bone is, you can see, it's outlined here in this X-ray, and heterotrophic bone is an extrinsic contracture, only in this case, it's a hard extrinsic contracture, it's one of bone and it will block flexion. So the extrinsic contracture here blocking flexion. However, the more common extrinsic contracture is pericapsular and that is, again, related to this anterior capsule.

Now why does this develop contracture? So there's one of the things to tell you. Whenever you have posttraumatic stiffness of the elbow that develops pericapsular adhesions, the anterior capsule is contracted 100% of the time. It's always the main culprit. Now let's talk about this for a moment. This happens because while the capsule has a high concentration of fibroblasts, it has cytokines that are released typically upon injury in this area that rapidly convert these fibroblasts to myofibroblasts. This has a higher propensity to myofibroblastic formation than any of the other soft tissues we see around the elbow, and therefore, we get significant contracture of this capsule, shortening of the capsule, and I mentioned the confluence before.

So here's that cellular process. When I have an injury and the inflammatory process begins, and you can see here, neutrophils beginning the inflammatory process. We will get this chemical cascade from phagocytosis that will cause the development of fibroblasts into the region. And those fibroblasts will then typically heal a wound. But in the anterior capsule of the elbow what happens is this remarkable process where these cytokines are released, and you see them here, that's not the blue cell. It's actually



blue cell here is the myofibroblast. These cytokines is these little outlines that look like dots all around it, and those are chemical messengers. What they do is they tell this fibroblast here to turn into this myofibroblast here. What you're looking at here is what happens in 72 hours. You go from no fibroblastic activity to fibroblastic activity to proliferative myofibroblastic activity in 72 hours. So right after an injury of the joint capsule, the elbow just takes off metabolically and creates this change in the capsule. And so the capsular change looks like this. It goes from, where's my arrow? There it is. So it goes from a regular dense collagen fibril to this absolutely unorganized bunch of cross-linked collagen molecules. Now why am I showing you the collagen molecule here? I wanna show you this molecule, because this is the basis for why we develop stiffness in these collagen tissues. Collagen molecules sit in a crimped formation. They sit in a, basically, like a spring at rest. And what is supposed to happen is when we move, these are supposed to unwind and go to full length. And when we are in normal tissues, this is what they look like. They go to this nice longitudinal length. They lengthen out beautifully, and you don't really get any significant restrictions in movement. Only until the collagen fiber has fully unwound, then you start see increases in stress and strain as we continue to add stress to the tissue. But what happens because of these myofibroblasts is this collagen actually stays in a shortened cross-linked position and isn't allowed to lengthen out, and therefore this irregular dense collagen creates significant restrictions in motion, and this is happening in the anterior surface of the elbow. So this brings us, so now we see the biomechanical and we see the physiological reasons for why we develop stiffness.

The final reason we develop stiffness is because we have neurological and muscle issues. Now I went over the muscle issues a little bit, but here's a way to look at the muscle issues in cadaver. This is your bicep. This is your brachialis. Here's your bicep right here, the elbow site. Here's your brachialis muscle right there. Here's your brachioradialis muscle out here. You can see them crossing the axis of rotation anteriorly making a biomechanically advantaged structure. And here is your tricep over



here. Your tricep inserting right there creates a biomechanically disadvantaged structure. Now neurologically, your brain is actually designed to match these biomechanical considerations. Your brain finds it much more, your brain readily activates your bicep. Think for a moment. Somebody who's done neurological rehabilitation, if you've worked with a stroke patient, does the person typically develop, even if it's terribly pathological movement, they typically develop bicep function at some point in their recovery unless the stroke is horrendous and has sort of a flaccid feel to it. And that is because the brain has protective mechanisms to protect flexion. Not only is the joint built to flex, not only are the muscles mechanically advantaged for flexion, but the brain is organized to allow flexion, because the body needs the elbow to flex for you to feed itself. So it's an evolutionary construct that causes the preservation of the movement of flexion. Now this interferes with rehabilitation and we're gonna go a little bit on how it interferes with rehabilitation. Now remember our torque, right? Remember, our torque is at its highest force at 90 degrees of flexion. So what we get and what we see here is something called the biceps co-contraction phenomena. I'm gonna take you through a case, and you'll be able to see the biceps co-contraction phenomena in action. This phenomena, I'll talk about this a little more briefly, a little more detail, in part two in how to remediate it, but here's how to observe for it and how you can see if it's interfering with the elbow stiffness that's presenting in your patient.

So let's go through this case. Let's look at this fairly significant injury. Here's a gentleman who sustained in an automobile accident this fracture. Where's my? There we go. He has a midshaft humeral fracture. He also has a Monteggia fracture, which is a grade I, which is a proximal ulna fracture near the dislocated radius with no fracture. That looks like this down here. That's what a dislocated radius looks like. So this is his injury. But I want you to see him postoperatively. Look at the joint space. It's actually not so bad. There's no exostosis. There's no calcification with a fairly good joint space and excellent reduction of his fractures. Let's watch him move. Let's watch him



standing. Now before we watch this video, take a look at his posture. Now this is the arm that's involved. This is the arm that's not involved. Here you can see his obvious elbow flexion contracture. But what I really want you to notice is the associated changes in his body. His left shoulder is elevated compared to the right shoulder. His left hip is elevated compared to the right side. What he is doing is actually motor learning an abnormal gait pattern. The genesis of the motor learning is elbow stiffness. Let's watch his motion. Can we see this video now, please?

All right, let's take that back, that's a little far. Let's look at this video. All right, let's watch him move. And you can notice even when he steps, even as he's walking, the weight shift is causing him to hike his pelvis. You'll also notice that as he swings his arm, his arm swing is exaggerated. And you'll notice how far back he has to pull his arm to bring his hand into an effective position. Remember that proprioceptively, his brain is telling him that his arm belongs at his side. And point of fact, proprioceptively his brain is telling his hand that his hand belongs at the greater trochanter. Now since his elbow is stiff, he can't return his hand to its normal posture unless he compensates by bringing the elbow into an overly extended position. The only way to do this functionally is to actually elevate the scapula and elevate the shoulder. So by doing that, he creates a movement pattern of posture that is actually reinforcing the contracture. So it's an interesting neurophysiological perspective. He actually is reinforcing through motor learning his abnormal movement of his elbow. Let's see this in action for a functional task. Can we look at the next video, please?

Now the next video shows him restricting his movement due to biceps co-contraction. Let's watch the beginning of this throwing activity. I'll run this through and then I'll stop it and we'll rerun it. Okay, now let's point out a few things. Okay, let's stop it and rerun it from here. What I want you to concentrate on now as you watch the video watch the amount of extension in the left arm in this video. Watch the compensatory movement at the shoulder as he's trying to engage in a functional tossing task or in a tossing task.



And you notice that he really, look at the lack of extension here. He's truly throwing with the scapula. He's actually protracting and elevating his scapula to be able to complete the task. Now you're gonna notice that he's gonna be quiet for a little bit. And I'm gonna cue him and give him cognitive instructions to concentrate on. I want to see what happens. Now look at the difference in his extension pattern. You can see that his arm is more fully extended. And if you watch this from this point forward, you'll see that it actually happens in almost a cogwheeling fashion. There, notice it in this next throw. You see the extension, you see the bicep release. He is using cognition to help overcome the bicep co-contraction phenomena. Let's look at this in a placing task. Can I see the next video, please?

If we watch this placing task, we can see the compensation much more clearly. It's a little less of a dynamic task. All of his movement's happening from the shoulder. So not only does this fellow have stiffness of his elbow, but he has neurophysiological overlaying stiffness from his bicep co-contraction phenomena. So we need a variety, now think about this for a moment. We need a variety of inputs for this gentleman to be able to help him overcome, not just the stiffness in his elbow, but the actual biceps co-contraction. One of the mistakes we make as therapists, in my view, is that we'll spend a lot of time with this elbow only focusing on the stiffness of the joint, and not correcting the abnormal movement pattern we see in the scapula. It's extremely important to get this part under control to be able to start addressing the actual contracture that's in the elbow. Take a look at our next video, and you'll see the difference in movement. Let's come to the next video now, please.

You'll see the difference in movement with someone who does not have a co-contraction phenomena. Okay, lemme show you his X-ray first, sorry, I lost that little bit. Let's look at his X-ray first. This is a gentleman who has a much more significant injury to his elbow. This is a grade III Monteggia. If you look here to see his, I'll take you through his X-ray, he has an intra-articular radial fracture. He has an intra-articular ulna



fracture. You can see right here is his ulnar fracture. The intra-articular nature of the ulnar fracture is right there. He has three screws in his radial head into the area where I told you contracture development happens at its most significant nature. Same surgeon, same surgeon, so same surgeon, same therapist. Take a look at his motion. Let's go to the next video, please. And you notice in his video he's two weeks out from his injury, maybe two and a half. He's doing a little bit of overhead motion with a little touch of overpressure. We'll talk about that in treatment. And watch his extension now. And more importantly, watch the lack of substitution at his scapula. You'll notice that he extends beautifully. Now his actual joint stiffness is about the same as you saw in the previous video, but his movement pattern is completely different. That is because he has cognitive control of the biceps co-contraction phenomena. So the point in therapy is this, and I'll leave you with this for today before we take questions. The point in therapy is the therapist must, in planning treatment for the stiff elbow, discern whether the problem is purely mechanical, is mechanical with soft tissue overlay. Most of the time that's the case, mechanical and soft tissue overlay. And is there a component of this neurophysiological biceps co-contraction phenomena that is contributing to an abnormal movement pattern, and if so, it's my opinion, that the therapist has to address the abnormal movement pattern at the same time they're addressing the stiffness of the elbow joint itself. Okay, so I'll take questions at this point. I know we're getting near the end of our time. So if there's any questions or concerns, I'd like to hear them at this point.

- [Fawn] Hi, Paul. While we're waiting for questions to come in, do you wanna go back to those two flexion and extension in supination/pronation videos and just review those one more time? I thought those were really good, and you said that you might come back to those. In that way, we'll give a little better time for people to login.
- [Paul] Sure thing. So we're talking about these little videos, the animation videos, correct?



- [Fawn] Yeah, and actually we can just pull those up. You didn't have to go back.
- [Paul] Yep, oh sorry, there you are, okay. Yeah, if you pull those up, I can talk over those for sure. Okay, so when we look at flexion, sure, no worries. So here's the flexion video. When we look at the flexion video, the key thing to remember about this video is the contribution of the muscle action. Now when we look at this video here, think of it this way. If the bicep co-contraction phenomena is occurring right here at this position, you can see that the bicep is gonna be a constraint to full extension. The bicep is so mechanically advantaged at this 60 to 70 degree ratio that when this bicep muscle fires, it's almost impossible to overcome it. There's one thing we see the clinic all the time. We'll see a stiff elbow and we'll see the bicep tendon sitting aggressively and activated while we're trying to gain extension. The problem is the typical answer for the therapist is stretch a little bit more or do a little bit of contract/relax phenomena. While the stretching is probably counterindicated, the contract/relax phenomena is a very good intervention to think about that point. I could then do an isometric contraction of the bicep and then do a PNF type of relaxation to allow this to lengthen out a little bit further. What actually does is it gets the bicep out of the way, so I could get down to the stiff capsular tissue. Now this is important because if we don't relax the bicep, things like using three-point splints and our static progressive and dynamic splinting will become less effective, because the time is then spent overcoming the bicep co-contraction and not actually addressing the stiff tissue at the capsule. That's my little review there.

Okay, well if there's no questions, then I'll wrap it up by saying the interventions for biceps co-contraction look a little bit different than they do for standard stiff elbow. In part two of this video, of this presentation, we're gonna be going through joint mobilization, soft tissue mobilization, exercise intervention and orthotic fabrication to address the problems we talked about today. So if there's anything else, you can email



me a question at any time in the next week, and I'll be more than happy to reply and try to help out and clarify any points from today.

- [Fawn] Thank you so much, Paul. I think that was a great introduction to elbow stiffness. We do have another course coming up next week. It is gonna go further into this, so I appreciate that. Please feel free to reach out to his email if you have questions. We should have this up on the site shortly, so hopefully, you might able to review this before the next live event, and then that way you are prepped for that. So again, I thank you so much, Paul.
- [Paul] You're so welcome.
- [Fawn] Have a great day, everyone. Hope you join us again on continued and OccupationalTherapy.com. Thank you.

