

ICSC01 Head Injury in Sport – Section 1 Sports Concussion

ICSC01 Head Injuries

Section 1 – Sports Concussion

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Video Lesson: 02:15:42

Welcome everyone to the FICS sports related concussion management online module. My name's Brett Jarosz. I'm a fellow sports and exercise chiropractor, based in Melbourne, Australia.

The purpose for this particular online module is for the sports chiropractic world to be able to further enhance our skills, our ability, in the management of concussion. So far, we have gone through the online modules, going through the new update from the consensus statement with the SCAT6 and the SCOAT6 and understanding our way to navigate through those assessments. What we would use in that acute concussion assessment, on the sidelines, on the field in your office in that acute setting, mainly in that first 72 hours, maybe up to a week we were using the SCAT6. And then when we are dealing with people coming into the office, 72 hours or weeks later, then utilising the new tool from the consensus group. The SCOAT6.

The focus for this module, is to try and further enhance our understanding and knowledge of those assessments, understanding the physiology, the pathophysiology, neural substrates, mechanisms, first principles, whichever way you that sort of makes sense to you. All those words say very similar things there. But when we do our assessments, what do those assessments mean from that first principal mechanisms, physiology, neural substrates perspective? What we want to try and do is go through these key points from the consensus. We will go through the reevaluation or the evaluation process, which is part of SCAT6 and SCOAT6.

Discuss this rest and exercise and understanding why we are describing it, understanding the different mechanisms of exercise, how they impact our physiology but then understanding how concussion impacts our physiology. Therefore, understanding why we want to use certain types of exercise at certain times and why we don't use other types of exercise.

And then understanding our rehabilitation.

I would like to start with my disclosures and biases so we can set the scene of the importance. You will hear a couple of quotes today about understanding the pathophysiology of concussion and how important that is for the 20 to 30 percent of people who sustain concussion injuries, but don't get better in that expected thirty-day time frame. So those people that then develop those persisting post-concussion symptoms, understanding the pathophysiology there, proves very important, and my bias of this is very much framed in that space because this is what I see in my private practice.

I don't get to see too much of the acute concussion injuries. I see the people who have not recovered, so my bias in this presentation is the bias that I see from my clinic.

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We need to understand this better to help serve our patients, our athletes, and our community. Then, of course, we will finish off with that return to learning, return to sport. But I will be jumping around through these parts, through the presentation, trying to constantly tie these together for our understanding.

The reevaluation process or more aptly, the evaluation process. The reason they put the “re” in front of it is for this. The thirteen “Rs” of the sport related concussion management model from the consensus group.

The concussion in sports group provide these thirteen “Rs” so that way we can flow through that concussion management, logical protocol, logical clinical flow, whatever words make sense. Recognize, reduce, remove, reevaluate, rest and exercise, refer, rehabilitation, recovery, return to sport or return to learn, reconsider, retire, refine. A lot of those things we have already gone through with the previous online update, and then today, we are going to focus our attention on those four key points.

Let us start with reevaluation. The purpose of the SCOAT6 and the child's SCOAT6 was to give healthcare professionals a standardised, expansive, and age-appropriate clinical guide to a multi domain evaluation in the subacute phase. 72 hours to a week post a concussion injury with a view to guide individualised management. While that statement there is obviously quite profound from the consensus statement, is because concussion is very much multi domain. We have cognitive components, emotional components, visual processing components of what we are seeing, but then we have the ocular motor components, the parts that move our eyes. We have the inner ear vestibular components that connect our inner ears to our eyes, but our inner ear is also to our spine. For postural stability and balance we have our motor coordination, so our cerebellar coordination patterns, our neck could be involved, and then some of the newer stuff, which we will really emphasise today is some of these autonomic aspects as well. So multi systems, multi domains.

And we must be able to assess all these components in a concussion injury so that we can best create this individualised management.

But we need to understand that physiology, pathophysiology mechanisms, neural substrates, we need to understand those things to truly provide an individualised management strategy. Otherwise, it becomes, I see this, I do this, and it becomes technical cookie cutter approach. I see people who have gone through that approach from doctors, and it fails. The emphasis here is really focusing on that third of patients that don't get better. If we can do a better job earlier to prevent that 30 percent, then we are helping serve our athletes and communities to hopefully prevent those persisting symptoms, or indeed if they are in seeing you with persisting symptoms, ask yourself, how do we do a better job of providing that individualised management?

The SCOAT6 is broken down there on the left-hand side. All the different steps that are involved in creating that logical clinical workflow so that you can work your way through a concussion assessment, and what we want to focus on today is those areas in bold, and I want to try and integrate this together. We know that the major concern with concussion

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is it is still based on symptoms. The idea that athletes' or members of the community, whoever it is that you have in front of you, when we are assessing a concussion, the primary assessment tool to determine concussion injury as well as clinical recovery is their symptoms.

But what we want to try and get a better understanding of is which of the domains, which of the physiological systems may be hypo functioning or dysfunctional or hyper functioning that is in turn driving those symptoms. Trying to look at the world through an objective lens, the testing, objective testing, and understanding that testing versus the symptoms, which is the subjective. And of course, remember patients can lie about their symptoms. They could be telling you, no, I feel fine, especially athletes who want to get back to the game or return to sport.

Try and look at the athletes from the perspective of, we understand symptoms very important, but we want to understand objectively what is going on in our assessment, what those things mean. We want to really get into the detail of these objective vital signs, the cervical spine assessment, our balance or postural stability, our VOMS assessment vestibular ocular motor assessment, graded aerobic exercise testing, and then our return to learn and return to sport.

The orthostatic vital signs there is emerging evidence that has associated concussion with changes in the autonomic nervous system. The postulated mechanisms include trauma to the hypothalamus limbic system, midbrain nuclei, and the regions of the brain responsible for autonomic function. Where we want to go back to is understanding our concussion mechanisms. Remember, it's an acceleration, deceleration injury to your nervous system. And of course, we hear those words acceleration, deceleration injury to your neck, we call that whiplash.

Well, a concussion is that, but it's just occurring to your nervous system. So same mechanism of injury. One's just being impacted to the orthopedic structures of the neck. The other one's being impacted to the nervous system.

When we create that acceleration, deceleration, we get that stretching, the areas that get stretched are primarily the areas through the midline of your nervous system. If we understand our anatomy, our first principles, physiology, mechanisms. If we understand what lives in the middle of the nervous system, it is all those hypothalamic, the midline limbic areas.

When we go through the middle of that cerebrum, limbic system, hypothalamus, get down to the brain stem, what lives in the middle. Well, that is all our eye movement nuclei. It is all our balance pathways and nuclei, our postural muscle tone, our pain systems, our pain inhibition systems. It's our ascending reticular activating system, the thing that gets us alert and awake, the autonomic nervous system. If we understand the anatomy that lives in there, which you would have gone through in previous online modules, then we understand that this makes sense as to why the research is showing that the autonomic nervous system is involved in concussion injuries.

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The big part now is this orthostatic tachycardia has been described as being associated with a substantial subset of concussion clinic patients. So orthostatic referring to getting upright, Tachycardia, obviously, racing heart. Now the numbers with this, and it's important here to understand that there are different forms of dysautonomia or dysfunction of the autonomic nervous system.

It just happens to be that orthostatic tachycardia tends to be one of the most common dysautonomias that occurs in concussion. The disorder, the orthostatic tachycardia here, is what they see with a lot of concussion patients that have got persisting symptoms and have got this issue with their autonomic nervous system. When they lay down, and then they stand up. We see that they have a heart rate increase from supine to standing of greater than thirty beats per minute, and it causes symptoms.

That is key to having the diagnostic criteria here will break down this even further in a second. In this example, from laying to standing, their heart rate is 50 beats per minute laying on their back, then when they stand up, their heart rate increases above eighty. If you are an adolescent, so less than eighteen years of age, that has to jump up by forty beats per minute, or it reaches an absolute value of a 120. They might be sixty laying down. All of a sudden, you see it jump up to a 124, and then drop down a little bit. They are upright, it hits 124, and then it comes back down to ninety. But because it reached an absolute value of a hundred and 20, that also counts.

But the importance with this diagnosis for orthostatic tachycardia, and the true name here is postural orthostatic tachycardia syndrome (POTS), they stand up their heart rate increases and is sustained by more than thirty beats per minute, but they don't get a drop in their blood pressure. It's the absence of postural hypotension. Heart rate goes up when they stand up by more than thirty and it's sustained. But there's no drop in blood pressure. What does a drop in blood pressure mean? It means systolic does not drop by more than 20. Or diastolic does not drop by more than 10. We are saying blood pressure stays level, stays normal, heart rate increases by more than thirty, plus they have symptoms. And what those symptoms are is they feel lightheaded. They feel dizzy. They feel faint. They feel presyncable. In the bad cases, patients even faint. When they are bad, they stand up, their heart rate spikes up, and these people faint.

Understanding that this light headedness, dizziness, pre syncope feeling is the common symptom, but they also develop headache or nauseousness or sweating, or you will see their hands, feet start to go purple from that Venus pooling. We start to see these different other signs and symptoms go along with it, spotty, black vision, feeling of heaviness in their legs. This condition is really, really common in people who have persisting symptoms. So please assess for these symptoms.

Now in the consensus statement, it was put forth this way to assess orthostatic vital signs in the SCAT6, which was to lay someone on their back for two minutes and then take their blood pressure once they have rested for two minutes laying on their back. We are there, two minutes pass, take your blood pressure, take your heart rate. You then get them to stand up, and then upon standing, after one minute, ask them if there's any symptoms while they are standing, and of course we take their blood pressure and heart rate. Now the

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problem with this is It's only for one minute. You will see the bad cases often show up here, but the more subtle cases you will miss, and you can see at the bottom of that table there, it gives you the clinical significance of what you're expecting to see and symptoms. The drop in systolic by 20 or a drop in diastolic by 10, that would be orthostatic hypotension. Which is still dysautonomia, but that's a different type. So, the blood pressure dropping in those examples one and two at the bottom of that table. Or the heart rate increases by more than thirty, and the blood pressure stays normal. That's pots. Or you might see that their heart rate drops. Blood pressure drops. And of course, we see these different variations of dysautonomia.

When we look at it from a pots point of view, blood pressure's normal, heart rate goes up by more than thirty. That's going to be one of the most common dysautonomias that we see. But we may miss it if we use this particular protocol.

What I am going to encourage everyone to do is to use the NASA lean test version instead of what's put in the consensus. The exact same thing. You lay them on their back and let them rest for a couple of minutes. Take their blood pressure and heart rate after two minutes of laying on their back, and then you have them stand up as you can see in the picture, have them with their back against the wall, have their feet one foot length away from the wall, both feet are there. They are leaning on the wall, and then you have them stand there for 10 minutes. Every minute that they are standing, take their blood pressure, take their heart rate, and ask them about symptoms. This is where we can start to see more of the subtle versions of these pots where it starts to gradually kick in after three, four, five minutes. If someone is bad, it will show up in that first minute. But often you must keep them there for a bit to see that autonomic failure or dysfunction.

On the left there, we can see some other prep instructions, because some people come into you and have already been diagnosed with pots, maybe prior to a head injury, or they have seen someone, and they have been diagnosed with pots. To make sure that we do the test appropriately, we want to limit their fluid intake to one liter for the day prior to doing the testing, limit their salt intake for forty-eight hours. No compression garments for the day of the testing, wear clothes, but no compression clothing. Then where it is appropriate and safe obviously, look at limiting or get them to stop the medications and supplements before the testing, as they could affect their blood pressure or heart rate. So that is how we want to do that NASA lean test. Let's go beyond what's in the SCAT and SCOAT6.

Let's do the NASA lean test.

Then when we move into the balance error scoring system, both within the SCAT and within the SCOAT6, we will understand how to do that from our previous online modules as well as your face-to-face training. As you can see in the video presentation "A, B, C", and "figure C" in the picture on the right, these are our modified balanced error scoring system tests. Feet together on firm flat surface. All of them with eyes closed hands on the hips. We have feet together, single leg on their non dominant leg. Ask the person which foot you would kick a ball with. They kick a ball with their right foot, they are going to stand on their left leg and then in figure "C" the non-dominant leg is the leg that's behind in our tandem stance. We have our error scoring system there. We know our rules of if they open their eyes, that's

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an error. If their hands come off their hips, that's an error. If they step out of position, that's an error. If they're leaning from the hips by more than thirty degrees, that's an error. If any of these types of things are happening together at the same time, it's just one error. It's not three.

We do the test for 20 seconds. Count how many errors are occurring in that 20 second. If they reach 10 errors, just stop the test, and just move on to the next one. Where it is appropriate, we want to get people doing the testing on foam.

That is no longer the modified “M” bess, the “M” bess, when you do the foam testing, now that is the full bess testing. We got firm and foam. Why do we want to bring this up and go beyond the modified “M” bess and do the foam, then do the full bess. Because of the different systems that this test is testing.

When we look at postural stability here, understanding that when you are upright, we are relying on vision, the vestibular system / inner ears, and our proprioceptive / somatosensory system. We have our muscles and our mechanoreceptors in our joints, and they are all giving us input up into our brain stem and to our cerebellum in order to tell us where we are in space. Why this becomes important is these things.

A real simple rule here is when you are assessing balance on that balance error scoring system, yes, you are counting the errors, I want you to watch for which way the person swaying in the subtle cases or which way they keep falling.

What happens with our balance? If we have a cerebellum, or vestibular or brain stem problems, remember your vestibular system, your balance nuclei, your vestibular nuclei, your eye movement nuclei, those pathways, they are in the brain stem. If we have dysfunction of one cerebellum, in this case, let us say it is the right or my inner ear, my vestibular system on the right or those pathways that go from those parts of the anatomy into our brainstem to then go to the brain or to the eyes or the spinal cord, wherever it is going to go. For those pathways from vestibular, cerebellum, into brain stem, what happens if one side's down, like doing weights and you watch someone doing bicep curl, and then we watch, this arm coming up, and everyone's watching the left, but its right arm taking a bit longer.

Everyone at the gym might say “you need to go and work on that right bicep, it's not as good as your left”. We will see that people with vestibular hypo function or cerebellar hypo function or brain stem hypo function will sway or lean to the side of hypo function.

When we do this test, not only counting the errors, but your count is also observing which direction are they swaying? Which direction are they falling? Do you consistently see they are going to the right, and then immediately in your mind, you say alright, I am making the error count, but I now also know that this person's either got something going on with their right cerebellum, their right vestibular system, or that right brain stem. Where those vestibular and cerebellar projections go, so primarily into the pons and the medulla there on that right side.

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Those things there are important for us to remember because we want to then put that together with other tests. The balance error scoring system that we brought up before, why do we maybe want to put someone on foam as well? When you stand on a hard flat surface, what is normal in a normal/normal person? Was supposed to rely on vision 10 percent.

Our vestibular system, 20 percent, and our proprioceptive/somatosensory system 70 percent. 10, 20, 70. Neck, ankles and feet, hips in that order of where the most weighting goes for the proprioceptive input, so hard, flat surface.

Norm is referenced as most normal environments, when you are walking around your house, standing in the shops, whatever it is, 10, 20, 70. But as soon as you put someone onto an unstable surface, when you put someone on foam, your nervous system reweights. And that's what this picture here is showing you. It shows you how your center of mass changes and the weighting of your nervous system shifts. When you are on an unstable surface, you now shift and rely 20 percent on vision, 70 percent on your vestibular system 10 percent on your body. So again, why do we as clinicians in our office want to start going, I want to go beyond the MBess, the modified, balance error scoring system.

Why I want to do the full BESS is because now when I put them onto that foam and unstable surface, it's now going to give me a different clue of different systems. If I am on the firm surface, I am primarily weighted to my body. Proprioception, but if I am on an unstable surface, I am now weighted to my vestibular system.

It is going to give us clues as to what systems we want to look at. So now you have done your balance testing, and you have seen that they are swaying to the right. We have now thought to ourselves, that could be that right cerebellum. That could be that right vestibular system. That could be that right brain stem.

Now you put them onto the foam and do it, and you see that they fall to the right and now you are starting to think, maybe this is looking more like that vestibular system is the key here. Well, I am going to have to do more vestibular tests to confirm my hypothesis because that is going to start to dictate where I create my individualised management. So far, are orthostatic vital signs for dysautonomia.

Our understanding now of that balance system and what we are seeing when we do balance testing, what it means. We are now going to do more than the modified VOMS that's in the SCOT6, and hopefully you will understand why when we get to the neural substrates. Based on concussion, concussion is still remembering symptom provocation testing. It is about the symptoms, and symptom recovery for determination of clinical recovery. As we can see here, their justification in the consensus statement is that symptom provocation with the VOR and the visual motion sensitivity tests appear to be associated with concussion. The modified VOMS have the same diagnostic accuracy and the applicability as the original VOMS, based on symptoms.

What you are going to see, within these changes to the concussion recommendations, is they have taken out some of the vertical testing, and the near point convergence. But from

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an objective point of view, that's almost like saying that concussion doesn't affect those areas, which is nonsense because 50 percent of concussions have a problem within the near point convergence. They have convergence insufficiency. We want to do all the testing, even though the modified forms are being used for assessment of symptoms only, and if you are just doing it for symptom assessment, the modified VOMS will be fine. But if we want to be truly objective and start to try and figure out where this concussion has affected that person that is sitting in front of you, we want to do it all. Remember the last point here, it is important to recognise that if symptoms are reproduced during the VOMS, that does not rule in the presence of a vestibular or ocular motor problem.

Really important, you need to practice these tests. Understand that when you do these tests, you may not have had a concussion. You may not have a vestibular ocular motor problem, but you do these tests, and you can feel symptoms from some of these tests, which is why we have got to do more tests and put the whole story together for that individual in front of us.

As we can see here, the table from the SCOAT6 on the left shows you have the various tests, and then we are asking about the four symptoms listed across the top there, headache dizziness, nauseousness, and fogginess. We ask the person to rate those symptoms out of 10 before you do the testing. This is the person's baseline. Then we go through each of the tests. On the left, you can see, we do smooth pursuits, we do horizontal and vertical. Now when you get to the Saccades, you can see that they have just got horizontal, but I want you to all do vertical as well.

Then you can see with the VOR, the VOR's got horizontally. They took the vertical out. But again, we want to do the vertical. They have removed near point convergence, as I mentioned previously, that we also want to have in, and then we have our visual motion sensitivity test. The general rule is we do the test, we observe while we are doing the test, to see for objective issues that may be going on and then post the test we are asking about the symptoms as well for the symptom provocation.

Video presentation placement: 29:50

Our smooth pursuits can be seen in the instructions in this slide, we are just moving the pursuits side to side at the rate of two seconds. Basically, from the side to the middle takes a second, and across the other side, we obviously make it flow two seconds from side to side. We do two repetitions.

One, and then two, we ask about symptoms, but I want you to repeat that in a vertical direction as well. We have got our testing there; you record those symptoms after the testing. The point on the bottom there, observe for saccadic eye movements or jerky eye movements. What should happen is your eyes should be able to stay on that moving target. If we see their eyes, doing something like displayed in the video, jumping off the target and back to the thumb, they are losing the smooth pursuit movement. We are looking for the blatant pathology things too, where the eyes show, one eye's moving, and the other one's not. Look for our pathology, but we are looking for dysfunction. Why does this matter?

Video presentation placement 31:03

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The confusing chart in the video slide on the right, I want us to understand what's involved in a pursuit of eye movement. The pursuits classically come from this parietal temporal cortex on either side. You can see in the example, if the target's moving to the right, my eyes are following it to the right. I want to keep this simple. The diagram in the chart is everything, let's keep it simple. When I follow my thumb to the right, pursuit is pulled by the parietal. Easy way to remember it, pursuit is pulled by the parietal. So, if the pursuit goes to the right, it is the right parietal, that is making it move that way. If the pursuit goes to the left, the pursuit is pulled by the parietal. So, it's the ipsilateral parietal lobe. Why is it important that we understand more of this? Hopefully, you are going to see the emphasis here, you do the pursuit and maybe you see jerky movements. They have got jerky movements going to the right. So, you are thinking "right parietal lobe". Maybe?

Maybe, because if we follow the rest of it, when I go to the right, the pursuit pulls it, but that pursuit parietal lobe goes down into your brain stem, and then it decussates across to the opposite cerebellum, and then from that cerebellum, it then goes into your vestibular system. When you look at that chart in blue, that is your VOR. That is your vestibular ocular reflex. Our pursuits use VOR.

If I follow to the right, that is the same as my head turning to the left. Left VOR, the eyes go right, a right pursuit, the eyes go right. This is why the anatomy matters because the pursuit to the right could be a right parietal problem. Could also be a left vestibular problem. This is why these little nuances are going to matter. It also could be obviously that brainstem. It could be anywhere in there, but I want to paint the picture there right now. Pursuits, parietal P and P, but the pursuits also use VOR.

In red there, area in blue, the common pathway shared by the horizontal VOR in the smooth pursuit. Really important, because if you rehab a pursuit, you see a pursuit, and the rules basically say this, if you see saccadic intrusions in the pursuit, you get told to give pursuit exercises. What if the problem is in their VOR? You need to give them VOR exercises to fix a pursuit. I hope that makes sense. We have to understand our mechanisms and principles here, so we can give the people the right exercises.

If we do vertical pursuits, it's no different except its primarily temporal lobe, and it uses your vertical VOR pathways. So same principles, pursuit to the right, right parietal. It's got the double decussating pathway. Parietal, ipsi brainstem, contra cerebellum into the VOR. That's my horizontal, obviously, reverses for the left pursuit. Vertical, both temporals down into that ipsilateral brainstem on each side into your vertical VOR pathway, which is what those things are showing you there, to your eyes. The takeaway, your pursuits are using the cortex, parietal temporal lobe, and then they are using the VOR. Key takeaway, parietal temporal cortex, and the VOR. Highlighted in red on the slide.

Now looking at our Saccades, (you will do this in the hands-on module) we have the right left, right left, right left, as shown in the blue italics on the slide, do it for vertical as well because the anatomy is different.

Video Presentation placement 36:31

As we do our testing, ask about the symptoms, and then we observe the objective things

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we see. When they are doing this, do you see them when they are supposed to look at the thumb here? I am just looking for visual purposes, they are supposed to look at the thumb, that thumb, that thumb. But watching the video you may see them lose focus. They decide they are pretty good, but when they go back to this side again they lose focus. Do you see that they overshoot the target? Can you see that in order to get to this thumb, they might be going from here and they might go here, here, here, here, and they have these saccadic corrections.

The more you practice this, the better you get at picking this up. We want to look for those objective issues with the eyes. The other little takeaway here that I want to give you, is the eyes from one thumb to the other thumb, you should basically just see them, be on one thumb and the other thumb, one thumb, other thumb, one thumb, other thumb. If you can see the eyes moving, a bit like a pursuit, if you can see that eyeball moving, their movement's too slow.

What does it all mean though when you are looking at those errors? Here we go. If I want to look to the right, a saccade is a voluntary eye movement, basically, the rest of your eye movements are reflexes. Your Saccades are the only voluntary movement you have, which is a bit like moving your hand, it's voluntary. We know that if I move my right hand, it's controlled by my left frontal, my right leg left frontal. If I want to look therefore to the right, it's left frontal. And if you want to remember it frontal, fast eye movements, "F" and "F" so parietal pulls the pursuit, and then we have got our fast frontal, to the opposite side. Activation of your frontal eye field and your superior colliculus generates contralateral horizontal saccadic.

Keeping it simple again. Frontal lobe. To your brain stem generators, then those brainstem generators, you can see in that fancy picture there. You got all those different paramedian pontine reticular formation, your nucleus prepositus, hypoglossi, medial vestibular nuclear. They are all the words, understanding our anatomy, frontal lobe, to that pontine medulla area in that brainstem, then move the eyes. That's our fast eye movement mechanism.

We want to look to the right, left frontal, to the right brainstem, pontine brainstem, primarily. I want to look to the left, right frontal, left pontine area. And again, why does it all matter? Let's look at this as the example. Let's say you saw someone falling to the right on your balance testing. You might be thinking, right cerebellum, right vestibular, or right brain stem. Now, suddenly, you maybe have done your pursuits, and as you did the pursuits to the right, you think that looks pretty good, and then you did the Saccades to the right. And now, all of a sudden, you saw that when they are going to the right, their eyes are slow or they are jerky, they are not fast, they are overshooting. Now all of a sudden, you are thinking, I saw them falling to the right. That could be that right cerebellum, right vestibular system right brain stem, and now I have seen that those Saccades to the right, that might be that right brain stem playing up, because when I go and saccades the right, left frontal, right brain stem, and they were falling to the right. I got two tests that are maybe suggesting that. Flip side of it, maybe the saccades are okay they are falling to the right, right cerebellum, right vestibular system, right brain stem, but when I did the pursuits to the right, that looked good. But when I did the pursuits to the left, remember which is left parietal, left-brain stem to the right cerebellum and right vestibular system. Now, this is

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something which has us thinking, this could be the vestibular system or cerebellum.

If it's starting to get overwhelming, it's okay. We are going to keep going through it over and over and over again, Just understanding the anatomy going through these slides. Fast, eye movements frontal, and then brain stem. But the importance of this is if we truly want to serve our patients and our communities better, understanding this to apply the most appropriate individualised management.

This matters. It really matters that we own this and that you own this understanding so that we can serve our patients the best way possible. And when it comes to vertical, the difference now, horizontal was contralateral sort of frontal lobe to that opposite Pons. When I do vertical saccades, it's bilateral frontal, to the midbrain. Different areas of the nervous system now. So frontal lobe to midbrain, always remember this. All your vertical eye movements end up coming from your midbrain. All your horizontal eye movements are generated from your Pons. So, these are going to be our two takeaways now.

So all of my horizontal has their component come out of the Pons? It just depends on what talks to it. Is it a parietal lobe that talks to it? Is it frontal lobe that talks to it, is it a vestibular system that talks to it? If it's vertical, it's the midbrain. If it's fast eye movements, the saccades, that's frontal, fast and frontal, vertical eye movements to the midbrain. If it's a pursuit, it's going to be that temporal lobe into your vestibular system into the midbrain. So vertical eye movements, for saccadic, frontal, midbrain.

For pursuits, it's that temporal area to the vestibular system to midbrain. So again, understanding these parts of our anatomy. We do these tests, observe for the deficits, and then from there, we put our story together of what things has this person got showing up as dysfunction. Now near point convergence, this was taken out of the modified VOMS. So, as you can see here, I have put it there as VOMS.

Because in the normal VOMS, it's included. Modified VOMS are taken out. I want us to be doing the near point convergence. Again, 50 percent of concussion injuries have convergence insufficiency. They have an issue bringing the eyes together. Again, we do three tests. As we have learned before, we get our distances of how close these people can get to their nose. We want to see that their eyes can get within six centimeters.

If they are not converging more than six centimeters, they have an issue. With all the VOMS testing, you ask about the symptoms, headache, nauseousness, dizziness, fogginess, rated out of 10, following your testing. But remember, we are trying to look at the eyes as they are bringing the thumb in, do you see that suddenly one eye stops converging on the thumb, and then we would stop moving the thumb and measure that distance.

Why does that matter? Well, your near point convergence is using your midbrain, your mesencephalon. Just some semantics here, there is three, four, not known thought processes of some of the pathways for, seeing it, which is occipital lobe, and then from that occipital lobe, other mechanisms using some cerebellar areas, some vestibular areas. There are question marks in the studies here? A lot of primate studies and monkey studies are used to understand where a lot of these pathways come from. The near point convergence

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is all of the in between like you have seen with the saccades and pursuits. Not truly known, but what could be agreed upon is those reflexes come from the midbrain. So that end point is from that midbrain. As you can see here, the mesencephalic reticular formation, and the reason I highlighted that, putting all this together now. The midbrain when we talked about saccadic, vertical eye movements all come from the midbrain. Convergence comes from the midbrain. The midbrain reticular formation also is in control or of the pathway of stimulating your sympathetic nervous system. And I will say that again, the mesencephalic, the midbrain, reticular formation is part of that system that sends a signal down to your IML to stimulate your sympathetic nervous system. What we often see is people who are a little bit anxious, maybe their heart rate's spiking, POTS or orthostatic tachycardia, you might start to see that there are issues going on with that person's midbrain. Now you might see that, don't know, but assess it.

Start to put that together and you will have done your orthostatic vital sign assessment, and you see that they have orthostatic tachycardia. Their heart rate's going up by a hundred and thirty. The person's reporting that they are feeling a bit anxious, sweaty fight or flight, a little bit wound up, whatever it is they are reporting these things to you.

You have seen they got tachycardia. You do your vertical eye movements, and you see that there are issues with all their vertical eye movements. You then do your near point convergence, and you see there's an issue with that. And now suddenly, you might think, maybe this person's got a problem with their midbrain.

How do we put all these things together? We keep doing our tests. We do more and more and more tests so that we can keep triangulating where the issues look like they are in this person's nervous system. Because if you just do one test, if you look at pursuits as the example, that could have been that parietal area. It could have been the pathway to the brain stem, it could have been the decussation from that brain stem to that cerebellum. Could be from that cerebellum to that VOR. It could be from the VOR, vestibular nuclei to the eye. Nuclear, oculomotor, the MLF, you can see the difficulty with that. If you do one test, it could have been any of those things show up. We have got to do every test that's possible that's in your tool kit, and then try and figure out all the tests and what test are showing dysfunction and where are the common places that have a relationship between each of those tests. We use that example. Vertical eye movements, near point convergence, heart rate, maybe we are looking at something midbrain. Someone falling to the right, maybe that could be a cerebellar vestibular problem. Maybe it could be your brain stem problem. Or which of my other test is showing that pontine area, it could be a saccadic movement, or it could be a pursuit movement.

Our VOR, at the understanding of the vestibula ocular reflex, now the modified VOMs took out the vertical VOR. Why that again is problematic is why do we want to take out a movement? We don't know if the person's got a problem with that vertical mechanism, remember that the modified VOMS are about symptom provocation. We want to go a step above that, and then look at the idea of the neural substrates. We do have VOR. We put a target up in front of the person, metronome at hundred and eighty beats per minute, the person's moving their head 20 degrees side to side, keeping their eyes fixed on that target.

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After 10 seconds of completing the test, wait for 10, ask them about symptoms. But while they are doing the test, you are looking to see, do the eyes stay on the target. If I just did that, you might ask them to, just turn your head to the right. The eyes jumped off, then they look back, he goes to the left, that looks pretty good. Goes to the right, the eyes jump off, then he looks back.

If you see someone, jump off, so they eyes move with their head and they come back, we call that a catch up saccade, and that lets you know whichever side they turn to, that example, they have me turning to the right, and then I jump back, that's letting you know that this right vestibular system is hypo functioning. It's decreasing function. Obviously, if I went to the left, that will be the same thing, on the left. I should be able to maintain my gaze the whole time and the eyes don't jump off.

This is the time to really emphasis why this is important. This reflex from your ears to your eyes is the first and only system that is fully developed when you are born. When that baby comes into the world, the vestibular system is the only system that is fully formed. It is the fastest reflex of humankind. That reflex from your ears to your eyes takes seven milliseconds, seven and a half milliseconds. The idea of you looking at the screen right now for the information on the screen, me moving my hand, for that to go through your eyes to your occipital lobe to see takes a hundred milliseconds. Slow. This is why when you put something up in front of you, and if you are looking at the screen, you can turn your head side to side if everything's working in your VOR, and you can keep reading the words that are on the screen.

But if you grab this screen in front of you right now and you moved at 20 degrees to second, you kept trying to read it with your vision. You can't do it. Your vision's too slow. We have to have an intact working VOR to be able to be upright and walking. We must have this reflex. You are going to hear me always say our priority in rehab is you must be able to keep someone's eyes still. They must be able to maintain gaze stability.

This test is really important. So let us go back. I turned my head to the right. The example I said before was my eyes then jump back up, a catch up saccade, when I went to the right, right vestibular hypo function. In rare circumstances, very rare, you may see the opposite where they turn their head to the right and their eyes then come back. They turn their head to the right, they overshoot and they come back. What we call a backup saccade.

In that particular instance, we are looking at a vestibular system that has increased function. Very rare that you see this. Now in certain circumstances, when we see it, we must understand that lets us know that that vestibular system's up, on that side, it's got too much juice. If I have this way and I have to catch up, I have dysfunction. Now the general rule with nervous system problems is that with the exception of, seizure disorders, epilepsy, seizure disorders, most vestibular problems are most vestibular. Most nervous system problems are decreased function problems. Epilepsy and seizures are, of course, increased nervous system problems. As a general, everything else seems to be decreased function problems.

Understanding most of the time, you are seeing a catch-up saccade, which lets you know that we have hypo function of the appropriate vestibular system. In this example, if we did

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a VOR, you saw that catch ups a saccade, like that, you think, that right vestibular system is not working too well. What other tests have you done that might also give you a clue. Well, you might have done the balance, especially on foam. Remember that is going to rely on the vestibular system more, and if that vestibular system's down, you might see that they also fall sway to the right. Now I have two tests that have seen it. I also now know that my pursuits also use that right vestibular system. So in order to make my eyes go to the left, with that VOR, you may have also seen that when they were trying to do pursuits, that their eyes were jerky to the left because that left pursuit uses the right VOR.

Hopefully, we are starting to see how important understanding of physiology mechanisms/neural substrates are. Of course, for our vertical VOR, it's the same thing. If I take my head forward, of course the eyes should stay on the target, but if my eyes went down and then look back up, it's a catch-up saccade. Obviously, if I am doing it that way, it should be able to stay on the target with my head going backwards, if my eyes went with my head and then I jumped back down, catch up saccade. What you just know is that that's just using the vertical pathways of that VOR to that midbrain. So exact same principles, and we put our picture together with the rest of our tests.

Video presentation placement: 54:30

There is our anatomy, which is somewhat already covered, when we did the pursuit. Head rotation to the right stimulates the right horizontal canal, which leads activation of the left lateral rectus, right medial rectus, so both eyes rotate to the left. I turn my head to the right, both eyes should go to the left. A lesion to the left horizontal canal leads to unopposed action of the right horizontal canal, which causes your eyes to be turned to the left.

The example here, that wording there of the second dot point. If this left horizontal canal, which should drive my eyes this way, another way to view it is that your vestibular system is like two magnets. And you know, when you bring two magnets together, and you can feel them pushing against each other, if I had a magnet here that's weaker than this one, so we will call this, big magnet, weaker magnet, because there is a lesion here. Well, this magnet is going to push, so therefore, this pushing from my ears should keep my eyes there.

But therefore, if I have got a magnet here normal, and this magnet here less. Now this one is dominant, what's that going to do? Well, that will push my eyes that way, because this one cannot push the eyes that way. So as a result, if my eyes are being pushed this way, your brain is picking up a signal now, but my eyes are being pushed that way, so your brain asks the question, am I moving? Because the only way to make your eyes go that way in a slow movement is to have a pursuit. I need to be able to follow something. So, your brain's asking, am I following anything? The brain says no. Then the brain is asking vestibular system, are you turning, are we sitting in a swivel chair, are we in a car doing burnouts and spinning to the right, have I got something going on turning my head to the right? And then suddenly, these systems go, NO. So, your brain goes, well eyes, what are you doing over there, get back there. Then suddenly this pushes your eyes again this way and the brain goes, are we doing any movement, are we following anything? NO. Get back there. That's nystagmus. The slow phase that comes from that vestibular system, brain going, gets back there because that is where I am supposed to be looking. That's your basic mechanism of a nystagmus, whether it's right, left, up, or down.

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Obviously, the vertical VOR, so a downward head acceleration chin down, makes our anterior canals make the eyes move up, and then of course if our head goes backwards, chin goes up, posterior canals drive both eyes downwards. Why all these matters are the most common cause of dizziness in the world is BPPV, benign positional paroxysmal vertigo. The crystals in the ear, and the most common one because of the anatomy is in your posterior canals. That is where 80 plus percent of all your BPPVs are going to be. Very rare to see them in the horizontal or the anterior. Gravity is going to cause them to go into the posterior most of the time.

If we understand the anatomy of the posterior canal, if I have crystals going into that canal, posterior canal is the same as doing that. Which means those crystals are going to cause my eyes to get pushed down. The brain doesn't want that, so the brain goes get back, so you see the eyes flick up.

Now, of course, crystals tend to be in one canal most of the time, so if the crystal is in the right posterior canal only that right posterior canal is going to cause your eyes to go down to the left. So, what are you going to see in BPPV, your eyes go down, like that with torsion, slow, bang, flick back up. And that is what you're seeing in your classic posterior canal, BPPV, a torsional upbeat nystagmus.

That is our mechanisms of how our anatomy works, but we want to look for it. We want to see it because then we understand what part of the anatomy is involved, and we triangulate that. Take away message, if someone's got BPPV, you fix that first. You fix all those crystals, otherwise, we have constantly got faulty vestibular reflexes being stimulated. And the number one clue that you are going to get about BPPV from a patient history is when they roll over in bed. That's the number one clue that you will get for BPPV. When I roll to the right in bed, I get dizzy. As soon as you hear that, it's not the neck. You need to look and assess for BPPV.

If you are going to do testing properly, you need to remove fixation. Spend the money and get video oculography goggles or goggles where you can put the person in the dark. You could use VR goggles for this, you could go to your optometrist or a cheap shop where you can buy thick round lenses/glasses “coke bottle glasses” to use on your patients because then they can't fixate. This allows you to see those little eye movements that occur when people have got BPPV. If they have got it bad, you will see it without the goggles, but in the subtle cases, we need to be performing our Dix Hallpike maneuvers, our supine roll maneuvers, etc we need to be performing them without the patient's ability to fixate. We must remove fixation, in the dark or frenzel lenses so they can't keep their eyes focused on something.

Really important. So your patient tells you, “I roll over to the right in bed or anytime I roll over to the right, I get dizzy”, you assess for BPPV. Even if it's not a concussion, that is the first thing you put into your armamentarium, and then, if you find it, you treat it with your appropriate Epley's maneuver, for the posterior canals, or we go through our barbecue roll for the rare horizontal canals, I personally use the modified gufoni maneuver for horizontal canals. That is some of our options for BPPV.

<https://www.interacoustics.com/balance-testing-equipment/visualeyes/support/gufoni->

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[maneuver](#)

Video Presentation placement: 01:01:10

Our final test here in this VOMS assessment and the modified VOMS is visual motion sensitivity. The other way to look at this, is this is a VOR cancellation.

You are using your fixation to cancel the VOR. So, remember, when I turn my head to the right, my eyes go to the left. If I turn my head to the left, my eyes go to the right, but if I give myself a target, my thumb in this test, and I turn to the right. My VOR wants to push my eyes to the left. But I have kept my fixation on my thumb, so what - that in turn – does, is it cancels the VOR. So even though they talk about this test being a visual motion sensitivity test, it's a cancellation test.

We use the VOMS principles, you do the test, 50 beats to the metronome, and we do those five repetitions. Five each side. One, two, three, four, five counts, 10. Wait 10 seconds, and ask about symptoms. While they are doing the test, you as a practitioner watch their eyes. Can they keep their eyes on their thumb, or do you see their eyes jump off their thumb?

Why does that matter? Because your cancellation is a reference back to the pursuit mechanism. When I turn my head to the left my VOR should push my eyes to the right. That's my normal VOR. When I do a VOR cancellation or visual motion sensitivity, if I turn to the left my VOR wants to make my eyes go to the right, but I can cancel it because if I have a left pursuit, that left pursuit is what pulls my eyes that way. So now VOR wants to make my eyes go to the right, and if I have an intact pursuit, I can now use that pursuit to cancel the VOR.

The importance of our physiology, neural substrates, in this example we can see, deficient VOR cancellation, ongoing to the left corresponds to low pursuit gain to the left. If you do your VOR testing, and they are doing those 180 beats per minute, they've got no symptoms, and you look at the eyes and the eyes are staying on, but then you did your pursuit testing before and you saw their eyes were jerky to the left, and you are thinking, "is that the parietal area or is that that right VOR?" What's going on there? But you do your VOR and find, It's not that. But then when you did your VOR cancellation, you realise that their eyes jump off. Now you have another clue, that the pursuit mechanism on that left may be the problem. So that pursuit parietal area down into the brainstem pathway, maybe the area that we need to rehab.

That section is full on, and there are hours and hours of modules and books that go into more details about this, but I want to give you a few takeaway key points.

- Pursuit
 - Parietal pulls the pursuit.
 - Ipsilateral side, to the opposite cerebellum and vestibular system.
 - Vertical pursuits, that temporal lobe into the vertical VOR
 - or anterior posterior canals, to the midbrain or vertical eye movements to the midbrain.
- Our saccades
 - if I look to the left, fast frontal. So fast eye movement is a saccade frontal, so contralateral frontal lobe pushes the eyes and uses the contra from the

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frontal lobe, ipsi to the eyes, pons for horizontal movements, swap it around if I go to the right.

- If it's vertical, both frontal eye fields, into my midbrain, all verticals midbrain.
- My near point convergence, midbrain.
- My VOR
 - right vestibular, left vestibular, into the eye movements
 - if it's vertical, anterior and posterior canals. So that way, anterior canals. That way, posterior canals to the midbrain.
- VOR cancellation
 - if I go to the right, it should be the right VOR pushing the eyes, but my right parietal pursuit should cancel it.

There is my anatomy neural substrates physiology for our eye testing. It's important to emphasize that the VOMS was not designed as a comprehensive tool for vestibular ocular motor function and may not encompass all the screening strategies necessary to examine all aspects of vestibular and oculomotor dysfunction.

It is useful as a screen tool, but that may not be appropriate as a replacement for comprehensive vestibular and ocular motor assessment. We should still be using, all our other testing tools, gaze holding, our video-type goggles, optokinetic nystagmus, subjective visual vertical, dynamic visual acuity, velocity storage testing. You have your advanced testing with video, V-HITs, you have your other types of, C-VEMPs, O-VEMPs, otolithic dysfunction, so you can see there's a lot of other vestibular ocular motor testing to be performed, saccades, anti-saccades, gap and express saccades, different types of testing. VOMS is good for the screening, so what they provide us in the SCOATS6, what we talked about today goes beyond the SCOAT6, and understand that there's still more that we can do to go beyond that even more to help our patients.

Video Presentation Placement: 01:07:32

When we look at the neck, the cervical spine assessment there, that is what is provided in the SCOAT6. Muscle spasm, tenderness, paravertebral tenderness, our range of motion. But what are we looking for primarily this? We are primarily looking for bad injuries, such as fractures and dislocations. From a concussion and Chiropractic point of view we want to go beyond these assessments that are within the SCAT and the SCOAT6. If we look at this clinical practice guidelines, it was published three years ago, ([Quatman-Yates et al, 2020](#)) it is a really, wonderful paper. It is a 90-page article that summaries all of the best practice evidence. This paper was written pre the 2022 concussion consensus, but the consensus doesn't outline all the evidence for every single test and every single rehab strategy. That is what this paper was focused on and shows that there's clear evidence to suggest that the cervical spine should be examined, but there is limited evidence on which procedures should be used in relationship to concussion. This is the dilemma. We know it should be examined, but what test should we use in relationship to concussion.

There are several different groups out there. Katherine Schneider, who is part of the consensus or the concussion in sport group, did several different studies. These are a couple of things from Katherine.

1. Schneider KJ. Concussion - Part I_ The need for a multifaceted assessment.

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Musculoskeletal Science and Practice. Elsevier; 2019 Jul 1;42:140–50.

2. Schneider KJ, Meeuwisse WH, Palacios-Derflingher L, Emery CA. Changes in Measures of Cervical Spine Function, Vestibulo-ocular Reflex, Dynamic Balance, and Divided Attention Following Sport-Related Concussion in Elite Youth Ice Hockey Players. *J Orthop Sports Phys Ther.* 2018 Dec;48(12):974–81.

<https://www.chrisworsfold.com/3-neck-pain-diagnostic-tests/>

When it comes to neck assessments, our extension rotation test asked the person to extend and then to rotate as far as they can side to side. Your manual spine exam is basically like what was in the SCOAT6 just then. You know, it's just your PA like motion palpation. From that PA motion palpation, just feel what you feel is the limitation of motion in one segment on the right or left. They just ask you to rate it as normal, slight, moderate, or marked, but use your feels there for that. Obviously, then palpate, is it sore? Is it tender? Over those same areas, and you can see the criteria that Katherine put there when the person's pain is reproduced, so familiar pain. It's greater than three out of 10.

If you are palpating a joint and you feel that motion palp is rated as moderate or marked, if you have those, it is highly predictive that it is facet joint mediated. Whether it's concussion or not, these tests are a nice group that you can put together to be able to decide whether it is facet joint related. Our extension rotation test, our manual spine exam, and then your palpation for tenderness.

Now the cervical flexion rotation test is when the persons on their back, you passively bring them up into full flexion, and then you rotate them to the right or to the left and as you can see in this slide, you are trying to see if the range of motion is reduced 10 degrees or more. With that blocked end feel, standard techniques we have done in our Chiropractic training feel that motion palpation.

If you feel that real blocked end feel and that significant loss of range of motion, we know that it has good diagnostic accuracy for that upper cervical cervicogenic headache relationship components there with that test. We have our extension-based ones, motion palp, tenderness, manual spine exam from the last slide, which is good for C2/3 below, and then you have this test, which is good for C1-2 area.

This is a great deep flexor and neck endurance test which is super important. There are relationships with our deep neck flexors for potentially concussion risk, especially in our high school age athletes and we also know it is a good predicting value in return to sport. Our testing, these two fingers hold them under the person's head, they perform a chin tuck, they lift the head up so that they are just above those two fingers, and they hold that for as long as possible. If that occiput ends up coming down and touching your fingers for over a second, or you start to see that they lose the double chins there, the skin folds, they start to lose that, you stop the test from a fatigue point of view. Head touching, the skin folds lost or if the person starts to get a pain reproduction. Otherwise, it is a test until failure, and we are trying to record the time that this person can hold those tests for.

Our head laser, classic whiplash type test. You will see this test and the next one becomes important as differentiating tests for vestibular problems or vestibular from neck problems. As chiropractors, naturally people think, chiropractors are only good for neck problems. But

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as chiropractors, we need to take that responsibility and go, is it a neck problem or an inner ear problem? So, vestibular problem or a cervical proprioception problem. We need to do a better job of differentiating that for the patient. To give them the appropriate care. So, joint position error testing, we got the head laser on, they have the target about ninety centimeters away from them. They close their eyes, turn their head to the right, come back, they don't open their eyes and you mark off where they got on the target. You passively reposition their head back to neutral, and you get them to do three tests on each side. Six, alternating or three on each side whichever way you want to do this test, and you get the average for the three trials on the left and the right.

Video presentation placement: 01:13:01

That chart you can see in the picture, the green and the yellow, anything that is outside the yellow, when you are performed at ninety centimeters from the wall, is considered dysfunctional, four and a half degrees. Obviously consider angles and our mathematics there, ninety centimeters away from the wall. Four and a half degrees, if you are sitting ninety centimeters from the target is dysfunctional. So that's our test for the cervical proprioception, knowing where my neck is, right or left.

The one you couple with that is the smooth pursuit neck torsion test. You do your smooth pursuits like you did before looking for your jerky eye movements. After you have done them normal in neutral, you then have the person hold their head still, and you have them turn their body under their head to ideally 45 degrees. If they have a neck problem, maybe only thirty, and you redo your pursuit testing again. You are looking for those jerky eye movements again, those catch-up saccades. The eye should be able to stay on the thumb smoothly. If you see those jerky eye movements, in particular when the eyes cross midline, that is what we are primarily looking for, and then of course can they reproduce their symptoms when their bodies turn under their head. Why do we do it with the body turned under the head? Because we are trying to stop a vestibular reflex, because as soon as you turn your head, you have created a vestibular response as well. Keep the head still, and we turn the body under the head to make more of the cervical afferents.

The cervical sensory motor, proprioceptive, checking the neck compared to that vestibular system. CSPR cervical spine, proprioceptive rehabilitation, for dizziness post-concussion.

- Hammerle M, Swan AA, Nelson JT, Treleaven JM. Retrospective Review: Effectiveness of Cervical Proprioception Retraining for Dizziness After Mild Traumatic Brain Injury in a Military Population with Abnormal Cervical Proprioception. *J Manipulative Physiol Ther.* 2019 Jul;42(6):399–406.

The results have suggested the patients with dizziness after a concussion who had abnormal cervical spine proprioception. The laser, joint position error, If that is abnormal, and or they have abnormalities with that smooth pursuit, neck torsion test, these people respond better to neck treatment and rehab rather than vestibular rehab.

You need to do your vestibular testing too because if someone's got BPPV, you don't do neck rehab, you do vestibular BPPV rehab. If you do smooth pursuits, when they are neutral, and you see that they have got jerkiness consistently in one or two directions. It's not a neck problem, it's a vestibular or central vestibular problem. If someone's has a nystagmus, so if

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you see that they are at the end and they have a nystagmus, it's a central vestibular problem.

You need to do the vestibular tests, and if you see that there are vestibular problems, whether it's BPPV or any of the central problems, as we can see here, exclusion criteria for any patients who had clear peripheral vestibular, BPPV or consistent central signs, so jerky smooth pursuits when they are neutral, not in torsion, or you see that there's like a nystagmus. If you see that, with or without visual suppression, you look at the ear. This is the importance of separating these tests, and as you can see here, properly determining whether concussion or cervical injury is the source of symptoms is vital because the management of each condition differs considerably. I want to emphasize a point here. Your nervous system is a learning machine, which is consistently learning and is built to survive.

The only thing your brain cares about is survival. It does not care about the fact that we can create this technology, I can record this and write these things, we can read it and learn and understand. Your brain just cares about, is this a threat? Is this computer threatening me? Are Brett's words threatening me? Everything is about survival.

If our brain is a learning machine for survival, it learns all the things that are negative to keep you safe. This is why you don't run across the road, or you don't put your hand on the hot plate anymore while it's on, why you don't jump in the lion enclosure at the zoo. This survival aspect is learned over and over and repeatedly. So nervous systems are great learning machines. The problem is that you can also learn bad habits.

Why does this matter for us from a treatment point of view? If someone's got a vestibular problem, and you don't assess their vestibular system, and you think, I am a chiropractor and I just treat the neck. You keep treating their neck, good adjustments, good soft tissue, good exercises, good rehab, and you keep treating this neck, and it is a vestibular problem. By ignoring the vestibular problem, you are training their nervous system to compensate from their neck.

So, you haven't really helped them, and you don't know this because you haven't assessed the vestibular system. Let's look at this example, we are going to say it's a vestibular problem, and all you do is treat the neck. You have a chance that you can train their brain to rely on their neck to override their ear. Where's the problem? Let's say they then go and have a car accident, or they develop DJD in their neck. Suddenly, that neck that you used to compensate for is now gone, and now these people go downhill.

So that is one of the risks. The other common type of thing that happens is we get all this great treatment going on the neck, really good treatment going on the neck, but they got a vestibular problem, and then as you are treating the neck, if they have a vestibular problem, the problem keeps coming back. They come in the next week and say, "It was great for a day or two, but then it just come back." or this treatment aggravates them more, because that's not the problem. The vestibular system's the problem, and the neck is the thing that's trying to compensate. All you are doing is treating or aggravating the compensation. So, we have got to make sure that we differentiate where the problem is.

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Video PowerPoint placement: 01:20:07

Where in the nervous system is this problem? Is it a vestibular problem, an ocular motor problem, a dysautonomia problem, a balance / cerebellar problem, a neck problem. Now the big one that has the most research is our graded aerobic exercise testing. This should only be performed when the athlete reports the general resting concussion symptom scale is not greater than the 7 out of 10. When the person you are assessing had a concussion and they are at rest, if they are reporting that their symptoms are 7 out of 10, you can do this graded aerobic exercise test, but if they are reporting that they are 8 out of 10 when they are at rest, wait to do this test until there are 7 or less. Once you do the test, which we will go through, then you can prescribe targeted heart rate treatment based upon 90 percent of the person's heart rate threshold.

Then if you would like, you can keep repeating this test every few days to a week to determine the appropriate heart rate the person needs to be working at. Where the literature is, the most evidence for any concussion treatment now is aerobic exercise. We want to understand aerobic exercise again for our group. So, the subsymptom threshold we are going to use is 2 out of 10. If someone hits a 3 out of 10, when they are doing aerobic exercise after a concussion, we stop the training. 2 out of 10 is fine, 1 out of 10 is fine, but if they hit a 3 out of 10. Stop the session and try again tomorrow.

Just repeating that, if we use subsymptom sub 3 out of 10, threshold aerobic exercise within 2 to 10 days after a sports related concussion, we know that it is effective for reducing the incidence of the persisting symptoms. If you get people exercising soon, you have got a chance of stopping them from getting persisting symptoms, but it is also effective for the people who have got persisting symptoms, to recover.

This is what is listed in your SCOAT6, and it a matter of ticking the box that you have either done it or not done it, and then just list which protocol you used. Now the Buffalo concussion treadmill test listed here is the one that has the most sort of research behind it. There are little variations of it that are now released, a Buffalo bike concussion test as well, but the buffalo concussion treadmill test is the one that's got the most evidence. And that is how we perform it, and this is covered in some of the other online modules. We perform that test to determine the person's symptom threshold and what their heart rate is when they get those symptoms or when they fail on that test. What is their heart rate when that test ends?

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So, understanding the physiology and the pathophysiology of concussion is especially critical for our 20 to 30 percent of concussion patients who get persisting symptoms.

Video Presentation placement: 01:23:23

Part 2 - Rest and exercise after that big reevaluation of the importance of it.

Let's understand aerobic exercise. Individuals with acute concussion have impaired cerebral blood flow, that's our whole stretching, sodium calcium coming into the cell, that creates that energy demand where the sodium potassium pumps are trying to get sodium out potassium back in. The calcium that's going into the cells creating vasoconstriction, and an energy mismatch.

I have an energy increase in demand to try and help those sodium potassium pumps, but that calcium going into the cell is causing decreased cerebral blood flow. What we can see here with concussions, we have impaired cerebral blood flow, cerebral oxygenation is impaired, abnormal blood pressure responses, dysautonomia. We know that what's going on with the concussion, so, the question we should be asking is “we need to be doing things to enhance cerebral blood flow, enhance cerebral oxygenation, help the autonomic nervous system”. The second dot point in this slide suggested the cerebral hypoperfusion might be responsible for the provocation or worsening of concussion symptoms. Decrease perfusion into the brain may be responsible for this provocation worsening of some symptoms. Increases in cerebral blood flow, cerebral glucose, brain derived neurotrophic factor, and heart rate variability are considered favorable biomarkers. If we see increases in those things, remember, HRV heart rate variability is a simple rule, and people always ask you about it. What do you want to see? The bigger the number, the better. As we get older, that number gets smaller, but the bigger the number in HRV, the better. The better tolerance you have in your autonomic nervous system. We want it to be bigger.

Increases in systolic blood pressure, mean arterial pressure, cortisol oxidative stress and reductions in diastolic blood pressure are considered detrimental. For example, an increase in systolic blood pressure during exercise may elicit a myogenic response in the cerebral arteries, thus stimulating reflexive cerebral vasoconstriction resulting in reduced cerebral blood flow in an already impaired state. The simple takeaway with that example is don't get people doing weights after a concussion. Because of the increase in blood pressure, then can cause a reflexive constriction, decreases the blood flow even more, enhancing symptom problems, enhancing that system dysfunction.

Understanding aerobic exercise now. Low intensity aerobic exercise, so that is 63 percent maximum heart rate or less, has been shown to increase cerebral blood flow and oxygenation and no adverse effects on exercising BPm cerebral blood flow cortisol, BDNF levels.

Moderate intensity aerobic exercise is less than 76 percent maximum heart rate, has shown generally positive effects on these symptoms. The top two points, they are low intensity exercise, maybe moderate intensity exercise, in terms of aerobic exercise, keeping someone like in that 65 percent less range has been shown to have those benefits, whereas you can see here high intensity has been shown to increase blood pressure, variable findings on cerebral blood flow, increase free radical production, cortisol synthesis,

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dangerous increases in mean arterial pressure, and reductions in cerebral glucose. So high intensity aerobic exercise would likely be detrimental for physiologically compromised concussed patients.

When we have a concussion patient, think about the American barbecue, low and slow, low level, low intensity aerobic exercise. When we look at the consensus statement, they have now come out and said strict rest until the complete resolution of concussion symptoms is not beneficial. This “cocoon therapy”, go into a dark room and wait till all your symptoms are gone, we don't do that anymore. Relative rest is monitoring your activities of daily living, resting where appropriate when your symptoms get up more than 2 out of 10, go back, rest and reduce screen time. Just get rid of screens for the first 24 hours. No TV, no phones, no computers in the first 24 hours. But after those first 2 days, so day 2, you want to start introducing activity of daily living within the symptom threshold. We recommend early physical activity that is tolerated. Individuals can systematically advance their exercise intensity based on the degree of symptom exacerbation experienced during the prior bout of aerobic exercise.

We can prescribe subsymptom threshold aerobic exercise treatment within 2 to 10 days after a concussion based on the person's threshold. Now this is where that buffalo concussion treadmill test is important. We do the test, find out what that heart rate is and where they get symptoms, where it goes to 3 out of 10 symptoms. As soon as that 3 out of 10 increases in symptoms occurs, the example being if they are 5 out of 10 when they are doing the exercise, that's okay, they are allowed to go to 7. So let it go up by 2, but if it hits an 8 on that test, we stop the test, we take their heart rate, and we see what that heart rate is when they develop those symptoms.

We then give them exercise at the 90 percent threshold. Let's just say they got symptoms at a 130 beats per minute. We are going to take 13 off that. They will be able to exercise at 117 beats per minute. We go through the prescription of that in a minute, but that's our subsymptom threshold exercise, which can be progressed systematically by repeating that test every few days to a week. Or they can just keep doing the exercise for 10 to 20 minutes every day. If that symptom doesn't increase by more than 3 out of 10, if it only goes up by 1 or 2 out of 10, or it doesn't increase at all, the next day, they can try and get a little bit more. And we want to start building people's aerobic exercise up.

That is how we prescribe the subsymptom subthreshold maximal exercises following your treadmill test. 90 percent of the threshold heart rate achieved on the treadmill test. They do 20 minutes a day for 5 to 6 days a week using a heart rate monitor.

Terminate the exercise at the first sign of symptom exacerbation, which is greater than 2 out of 10. If they hit a 3 out of 10 increases, they stop, or after 20 minutes, whichever comes first, you can keep doing the tests, as previously said, every few days to a week or every two to three weeks to establish a new heart rate model. The model I personally use in clinic quite a bit is to get the person to increase their heart rate target by 5 to 10 beats per minute every two weeks. And then get the person building that in the background, and then I will assess the vestibular, oculomotor, dysautonomia, and neck.

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In clinic, we are figuring out how we build those things up while they can just build this up on their own in the background. So, both alone and coupled with other impairment specific active rehab interventions, aerobic exercise training has been linked to faster symptom resolution and rate of return to sport and enhanced neurological recovery. So why is that the case? Because this low level to moderate level aerobic exercise enhances that cerebral blood flow. It enhances cerebral oxygenation, brings in all the good, favorable biomarkers that the brain needs when it's recovering in a concussion. Remember, we are in an energy crisis in concussion. We are using too much energy, but we don't have the blood flow coming in, that energy mismatch, that metabolic mismatch, the energy crisis.

Aerobic exercise at low level, at subsymptom subthreshold level, that is the thing that enhances cerebral blood flow, cerebral perfusion. These are the words I use with patients a lot. It is not about exercising to try and get fit, we are using heart rate-controlled exercise to enhance the blood flow into the brain, cerebral perfusion, to bring oxygen into the brain to help that energy crisis, that mismatch. Educating the patients of what's going on, so they don't go and do their exercise and tell you "I find it really hard to exercise at that heart rate" and you tell them you know. We need to let them know that it's to try and get your autonomic nervous system and perfusion to work effectively, and that's obviously what is not happening well in a concussion.

Video Placement: 01:33:13

Now when you look at the rehabilitation section of the consensus statement, you think about aerobic exercise as the first part of rehabilitation. Get people doing aerobic exercise straight away, then when you look at the consensus on concussion, this energy mismatches this metabolic mismatch, the energy crisis that's going on inside the brain is when we have that head injury, the stretching sodium calcium coming in potassium out.

That process is supposed to take about thirty days for it to resolve, maybe up to forty-five. This suggests that a lot of people may recover in 7 to 10 days from that process. But current advanced imaging is suggesting maybe 30 to 45 days. If we start doing this relative rest on the first day, and on day two, you start to get them to do some physical activity, aerobic exercise at a low level. And they are doing all that and trying to make their return to learn or return to sport, whatever the individual is. If we are dealing with athletes as part of being a FICS sports chiropractor, we are dealing with the athletes who we are trying to get ready to return to sport. If they are a student athlete, high school collegiate, whatever it might be, we are asking them to return to learn first. Learning takes priority for the student athlete. If they are professional athletes, of course, it is the return to sport.

Relative rest for 24 hours, maybe 48. Starting them exercising day two, day three. Aerobic exercise, low level, and if you are doing that, with that return to learn, return to sport program, but they are not getting better, or progressing, that is when the consensus statement outlines.

- If the person's got dizziness neck pain or headaches for more than 10 days, we want to start doing some cervical vestibular rehabilitation.
- If you have got dizzy balance problems, vestibular rehabilitation or cervical vestibular rehabilitation may be a benefit.

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Our job as chiropractors is to be able to assess properly so we know which thing we should be doing. Should it be neck, vestibular or should it be both?

We know that the inclusion of the sub symptom threshold aerobic exercise in combination with these other things should be considered. The first thing you are taking away; I get them doing aerobic exercise at an appropriate sub symptom sub threshold level as soon as appropriate. So, two or three days after a concussion.

In the case of recurrence of symptoms, when progressing through the return to learner return sport, reevaluating and referral for rehabilitation may be a benefit to facilitate recovery. All the things we just talked about before. Reevaluating for the dysautonomia for the oculomotor, for the balance, for the neck symptoms, evaluating all of that to determine what's the appropriate treatment for this person to help rehabilitate them from their concussion injury.

Dysautonomia, I want to just give them a few little things for these POTS, and I just want to give you things straight out of the literature that we know that we can sort of do safely, whether it's in hospital or in home, and then we will obviously build into understanding our other rehabilitation strategies that we have built up today. So, concussed athletes often have altered autonomic nervous system balance, which is reflected by higher heart rate during steady state exercise, versus controls. The primary autonomic nervous system control center is in the brainstem.

I talked about it before, the mesencephalic reticular formation is involved in this. Your other pontomedullary reticular formation is involved in these areas. We have our cardiovascular centers, our breathing centers located within those reticular formation structures in that brain stem. These areas may be damaged in a concussion, particularly if there is a rotational force applied to the upper cervical spine. The upper cervical spine, think of fighters, they will try and rotate the head on the skull to knock someone out. If there's rotation involved there, if we get this shearing which primarily affects this brain stem structures and with a concussive force, which is significant enough, we could get stretching through that area, and in turn could lead to those autonomic nervous system areas being stretch therefore being affected. The altered autonomic regulation after concussion is believed to be due to changes in the autonomic centers in the brain. So that hypothalamic limbic area and or uncoupling of those areas with the brain stem and then our peripheral receptors. Our arterial baroreceptors and the heart.

It is proportional to TBI severity and improves during TBI recovery. The worse the head injury is the worse the dysautonomia is. Super important, there is a large subset of concussion patients who have persisting symptoms that tend to get orthostatic tachycardia. We are testing that, with our NASA lean test, we go beyond what the SCOAT6 says. Stand them against the wall, keep them there for 10 minutes, take their blood pressure and heart rate every minute and monitor for symptoms.

If you see an orthostatic tachycardia presenting to you or POTS, postural orthostatic tachycardia syndrome, the evidence suggests that the autonomic nervous system dysfunction because of diffuse axonal injury, including brain stem structures and pathways

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mediating normal cerebral vascular auto regulation could account for many of the symptoms commonly seen post-concussion. We bring in this idea of tilt table training, so we have aerobic exercise. Now is the time we need to think about this clinically. Aerobic exercise, we said we want to start introducing day two or day three after a concussion. What if the person has also got orthostatic problems? What if they have a problem just standing upright and their heart rate spiking? You are going to try and give them upright aerobic exercises? This is where you have your patient who tells you “I tried to do all of my buffalo concussion treadmill test, and I tried to do all that aerobic training and it wasn't working”. Were they assessed for orthostatic problems? And often times you find that they are not. As we said, there are a large subset that have these symptoms missed. These types of people, if you want to do aerobic training with them, you need to make them do it recumbent. They can't be upright because they will be struggling to get blood perfusion to their brain, because they are upright. This is why you need to do all the tests, otherwise, you don't know. These people must be in a recumbent bike. In some circumstances, I have had people lying flat on their back, and in those upper body ergometers that you can buy, I have them put one of those up on a chair, so they are laying on their back on their ground, feet up on a chair, and we get them to do their aerobic exercises like that.

That might be how we need to institute aerobic exercise, and while we are also trying to get this orthostatic tolerance up, this is how they can do it in a hospital setting. They just put them on an inversion type table, a high low type of table, and then we can do tilt table training. You can see the model they do in hospital; they lay them on their back for five minutes, monitor their heart rate blood pressure, tilt them up to 70 degrees, and they are trying to keep them there for 30 minutes. They will be measuring their blood pressure and heart rate every 5 minutes. If they start developing fainty feeling presyncope, take them back down onto their back, monitor their heart rate and blood pressure, and they basically just get them doing this repeatedly and training their autonomic nervous system to try and deal with being upright. This is what they are doing in the hospital. So, if the patient can complete, the in-hospital tilt training, once a day without fainting for three straight days, they then refer them home to do home tilt table training, which looks like this.

You look at the picture on the right, it looks like the NASA lean test. Basically, one foot length out from the wall, upper back against the wall, and they try and stay there for 15-30 minutes. Now you may be asking, maybe, what's the reason for having your feet away from the wall? Because they are leaning on the wall, they are not getting muscle contraction, they normally get from their legs which pushes blood back up. We are trying to get those autonomic reflexes to keep the blood back up, not rely on the muscles from the legs, to shunt it. The idea of having your feet away from the wall and leaning on the wall is designed to take some of those reflexes away. So, we are not using those muscle responses, those muscles squeezing the blood. We are just using autonomic reflexes. And as you can see there, fifteen to thirty minutes twice a day, safe place without risk of injury, stop the session of the occurrence of the first symptom

An alternative to it is just using an inversion table, and then you can see the general process there of having them on a tilt table, they lay on their back, bring them up to 45 degrees for five minutes, see if they can tolerate it. If they can handle 45 degrees, you are progressing to 60 degrees. They can handle that for five minutes, progress to 70, then to 80, then to 90.

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As you can see, for a max of 20 minutes at or above 60 degrees if they have got tolerance. Any of these things, if you get symptoms of intolerance at any level, you just try and bring them back down to horizontal to allow them to recover. But we have got to build up that person's ability to be upright and that autonomic nervous system to perfuse the blood into the brain and keep good cerebral oxygenation, energy delivery. That's what this is all about. Remember, POTS and that heart rate spiking, the heart rate spiking is not the problem. The heart rate spiking is your nervous system trying to help you keep blood into your brain.

For those that are prescribed medications or practitioners who are trying to treat and slow the heart rate down, if that is what you are focusing on, you are treating the person's compensation, and that can make them worse. We need to do the training to train their autonomic nervous system to be able to effectively keep that perfusion in the brain. Don't go after trying to decrease someone's heart rate. That's the person's compensation to try and help them. Don't take away what they are trying to do to help them. You got to try and help their system work properly so that their heart rate can start to naturally drop down on its own.

Now the vestibuloocular rehabilitation we know is associated with reduced dizziness, improved balance, and faster return to sport when it is used in conjunction with your aerobic exercise, or if it's used on its own. It's expected that when you do this type of rehabilitation, you may get transient increase in person's symptoms. I like using that 3 out of 10 rules when I am doing my VOR and oculomotor and other vestibular exercises, but the key point there that I have bolded there, third point down. Patients with posterior and lateral canal BPPV should be treated with canal repositioning maneuvers.

Treat that, clear those otoconia out of the semicircular canals if they are present, and then you are assessing to see whether your patient needs to be doing other vestibuloocular motor rehabilitation. If you identify something on your tests. We talked about the VOMS, the balance testing and we talked about differentiating the neck with your joint position error - with the laser on your head - and the smooth pursuit neck torsion test. If we identify that there is a vestibuloocular motor problem, we want to provide that targeted rehabilitation strategy, and you then give that that matches what the person needs from your assessment, and this is the key, you can do any rehabilitation exercise as you like, as long as they are appropriate for the person's assessment. Where we fail is when we just give people exercises because you think, they have got a VOR problem, so you give them a VOR exercise, thinking it may work. But It may not. I have got a greater probability of being able to help someone. If I do my tests, I understand that a right VOR is that right the vestibular system. I understand that if my eyes jump off and they go to the right, that's a catch up saccade, and that is a right vestibular problem. I am probably going to want to bias that right vestibular system and I am not going to be wanting to bias the left side because otherwise I keep the imbalance there. The same as if I have got a weak bicep and I am doing bicep curls, and this right bicep keeps failing, but if I keep doing barbell curls, the imbalance remains. I am going to go and start doing some dumbbell work and bias that side a little bit more. It's no different with your nervous system than it is when we're doing that muscular type rehab.

Keeping your eyes still is the primary focus when it comes to this. If you see that someone's

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eyes are jumping off when they are doing a VOR, that is where you go. We must get gaze stability first. Your eyes must be able to stay fixated. If your eyes move off a target, if your eyes are just constantly moving with a little nystagmus or you can't keep your eyes still, if your eyes are moving less than two degrees, you will feel like you have blurred vision. If your eyes are moving by more than two degrees, you can start to feel like you are getting double vision, and every time your eyes move, at the time they move, which is why they must move so quick, at the time they move, your brain doesn't know where you are. That is why they must move quickly. So, if my eyes are constantly moving, though, constantly moving, because I have got a problem with my vestibular reflexes, if I have got a problem with that then my brain is constantly going where am I? And that is a threat.

Because if you don't know where you are, that means you can fall. And if you fall, that means you can hurt yourself, and your brain, because it's built to survive, falling is equal to the risk of death. So, everything comes back to survival. If my eyes are constantly moving, I don't know where I am, if I don't know where I am, that means I could fall. If I could fall, that means I could die. Very simple survival talks there from a brain perspective.

If you don't know where you are, and there the risks, how do you think these people feel? They feel anxious, and so what you will see in clinic is you will see a number of people turning up to you with high levels of anxiety, and when you do your screening forms and you do your SCOT6 and you do those anxiety questionnaires like a GAD7 or a depression anxiety stress scale (DASS), if you do those, or even just chatting to the people, people tell you, I'm anxious. And yet, what they will tell you, I have gone and spoken to these different people about my anxiety, trying these medications, and they don't work.

But then you go and do a whole bunch of vestibular testing, and you finally get vestibular problems. And that vestibular VOR type testing, if that has got a deficit, then you can start to see that these people are anxious. All because of that threat system being wound up. And this is why it is so important that we assess and understand that vestibular mechanism, understanding the different tests, but the important fact is they can keep their eyes still. All we need to do as you can see here, gaze stability training requires a patient to maintain their visual focus while moving their head to facilitate the recovery from the VOR impairment.

We have several different options. Gaze stabilization of VOR x 1. It is designed to increase the gain. If I went like that and my eyes jumped off as I turn my head and I jump back, catch up saccade example or day to day has been this right vestibular hypo function. The other word in neurophysiology, is that would say, the right side got a decrease in gain because every one degree that my head moves, my eyes should move equal opposite one degree. If it's two degrees per second, my eyes should move two degrees per second. That's a gain of one. That is the neurophysiology terms.

When you see the design to increase the gain of the vestibular system, it's designed to make that hypo function, that decrease gain, come back up to level. If I want to do these gaze stabilization exercises, which is just this. If it was to the right, if that was the dysfunction, I am probably going to want to bias doing a VOR like that. Maybe even turning my head to the right, closing my eyes coming back to the middle, so that way I am biasing that right

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side. Rather than just doing this. Because then I am doing right, left, right, left. How could I bias that person's dysfunction? If you understand the principles, it doesn't have to be a standard approach. I could put someone in a swivel chair, and I just turn them to the right.

The only problem that I have with that is that I don't have my eyes staying still. And so, we can see we have to make the clinical call of what does this person need I need that person's eyes to stay still versus if I spin them to the right in the chair, the eyes are flicking because they are not keeping their eyes fixated. How else could I do it?

Video Presentation placement: 01:52:44

I can do a times two. Times two meaning just two things are moving. Again, design increased the gain, so that on the right side, if we use that as example, I could make my eyes look at my thumb. I could make the thumb go to the left. While my head goes to the right, then close my eyes, and bring it back.

And so now I have the vestibular system I have my eyes. If I am just doing my head here, I have my eyes fixated on a target. So, I have my eyes staying still. If my thumb moves, my eyes are still staying still on the thumb, they are just following it, and then I get that double input. So, we know other ways to look at it. Is a VOR x1, one thing's moving, the head, but you could also view it that that's going to increase the gain by one.

A VOR x2, I have the thumb and head moving, two things moving. You could view that, which is going to increase the gain by two. It's going to help it even more. This is where we can start to play with, what is it that I want to try and emphasize a little bit more?

Do I need to emphasize this right side that little bit more because that one version is just not getting enough change, so maybe I will need to get it a little bit more energetic movement. Flip side of this, is I may need to get the other side down a bit. When you were doing this testing, you may have seen on your left that they went that way and came back, and there was a backup saccade. There was too much function, too much gain on this side. Or maybe you were trying to do all the increased game things on the right, without any result, and you thought how do I decrease this left side? In this example, I might make them do turns to the left while they keep their eyes on their thumb designed to decrease that gain. This is about understanding different exercises, but the most important thing is understanding our tests.

How do I hit those areas? You might give someone a right VOR because that you saw that when you were doing your balance testing, they were swaying to the right. So, I might do a right side to try and drive up that right balance posture reflex. Starting to understand, I might use an eye exercise for something that doesn't even look like it's got anything to do with the eyes, but it had everything to do with balance.

So how do we do this? Other options, you got your VOR x1, x2, x0. But from a home point of view or in clinic, moving people's heads passively. Because it's a reflex. If I actively, do it, that actively doing it is also recruiting my neck. So maybe a swivel chair keeping my body still or standing and using my legs and turning the whole body to the right, because now the neck's not being involved, and the ear is being involved more.

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This is where the problem with the rehabilitation is. I can't give you standard approaches for everyone because we must do our test and then logically think our way through the symptoms. Do I need that neck involved? Is it okay to have the neck involved? Do I need it to be more, pure inner ear? How do I make it more, pure inner ear? Well, I need to do it more passively so it's more of a reflex, maybe in a swivel chair. Maybe we can do it standing, but we want to progress those exercises as you can see. Progressed from passive to active.

Progress from non-weight bearing. Where's that relevant? Or what if someone's got orthostatic tachycardia?

I might need to have them laying on their back, and I do those VOR movements on their back because they can't tolerate being upright yet. Think about the individuals in front of you and remember there is no standard here. Please understand what the right thing is to give for this person at this point in time.

Our pursuits, you might have found that there was a deficit in the pursuits to the right, but we talked about it earlier. The pursuits to the right, right parietal temporal, ipsilateral brain stem to the opposite cerebellum into that vestibular system. I might need to be doing left VORs to help a pursuit to the right first.

And once that VOR is working well, then I can start to bring in the pursuits if that's appropriate. As you can see, practice following the thumb with the eyes, the head stays still in the previously determined abnormal directions, make sure the VOR's appropriate first, then you can progress to these, like, the head eye coordination where we can follow them in the same directions, VOR x2. Non weight bearing, seated, to standing, depending upon, orthostatic tolerance, balance, abilities.

As saccadic eye movements, Same thing, have we got frontal things here? Now here is where this becomes interesting. Someone has a cognitive problem after a concussion. I can't think, I'm really struggling to concentrate. Well, that's frontal, classically. You might start giving them small little saccadic eye movements like in the picture because you now know those saccadic eye movements come from the frontal lobe. Now I am not saying that's going to fix someone's cognitive aspects, but this might be a way that we can start stimulating that frontal area and in turn, help these persons cognitive areas start coming back online in a very lower level. Find the directions those saccadic weren't working too well, and then we prescribe the exercises according to what that person needs. Is it a frontal thing? Is it a brain stem thing? Is it vertical? Is it horizontal?

Our near point convergence the best test is the Brock String. Get that string. Get three beads on it, and you can see different instructions laid out on the PowerPoint slide. Get a string, they try and bring that bead as close as they can to their nose where it's in focus. Have the third bead arms distance away from you, and then put the middle bead in between those two beads, close one, far one, one in between. Then all the person's doing is as they look at the string, they look at the bead get the bead in focus, and as they are looking at the bead, they try and make an "X". So, an example, the lady you can see there, she has a green, a yellow, and a red. If she is looking at the yellow, and then if we look at the bottom picture,

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let's substitute the green there for a yellow. That's what she should see. She should see the yellow bead. She should see it's in focus. She should see the string makes an "X" and she should see two red beads behind it. As you get that bead in closer and closer, you won't get an "X" here closer. You'll just see a "V". Hitting that bead, and you will see the beads behind it. So that is an important thing for fusion.

Why is this important? Because this is reading. So people who have often had head injuries, concussions, even the people who come into clinic say, "I am getting a headache when I am reading. I don't know what is going on. I went to the eye doctor, and they said glasses are fine, I don't need any of that and my visual acuity is fine". It's 20/20, we do this near point convergence and suddenly you find that their eyes can only converge at 18 centimeters, and they are supposed to be within six.

As soon as you start working on this convergence, now suddenly, they can see a string, they can see an x, and they are working with that. They come back to you and tell you, "I can read, and I don't get a headache, or I don't get tired when I'm reading anymore".

This movement is so important for our ability to read. For 3D depth perception, it is important. This is why we want to keep that in the actual testing of the VOMS. Here's the bit that added another layer. Too in-depth to go into for this, but I want to flag it with you. We said that convergence comes from the midbrain. I said that the midbrain is also involved in that sympathetic nervous system.

If you have got a patient who's maybe got that orthostatic intolerance and that racing heart, maybe making them do vergence exercises or vertical eye movements because they come from that midbrain. If you make them do that, that could make them spike their heart rate more. That may not, you need to test it, but it's something to put in the back of your mind and think if someone's got dysautonomia, we have given you a priority that the eyes must be able to stay still when we come to rehabilitation, that is always number one.

Now let's go back a step. We said that we want to implement aerobic exercise, as soon as possible, like day two, day three after a concussion. Because cerebral blood flow, oxygenation, glucose, all those things, that's what's going on in concussion, the energy mismatch, the metabolic mismatch, the energy crisis. That is the priority. If someone's got dysautonomia, that is our priority in concussion rehabilitation.

We must get that aerobic exercise to enhance cerebral blood flow, and we must get people to be able to get upright. Some of these rehabilitation exercises may not be appropriate to be done with the eyes. Until we have that dysautonomia remedied and working properly first because that is the cerebral blood flow hypoperfusion issue, and maybe if I make people do vergence or vertical eye movements, and that starts driving someone's sympathetics more, I could get increased blood pressure, increased heart rate, and now I am making the problem worse. So, the importance of us understanding our anatomy and substrates and what it is we are trying to do.

Video Placement 02:03:43

1. Quatman-Yates CC, Hunter-Giordano A, Shimamura KK, Landel R, Alsalaheen BA,

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Hanke TA, et al. Physical Therapy Evaluation and Treatment After Concussion/Mild Traumatic Brain Injury. J Orthop Sports Phys Ther. 2020 Apr;50(4):CPG1–CPG73.

Now for our neck rehabilitation, we all have a lot of different skills in this space. We know from this study by Quatman and Yates these might be the exercises that may be good to be done after a concussion. Neck strength and muscle strength imbalances have been shown to be associated with concussion risk. We talked about that deep neck flexor aspect, especially in those adolescent high school age groups involved in sports, especially females too.

May be valuable for practitioners to provide cervical musculoskeletal interventions with the goal of decreasing risk for subsequent concussion injuries. We know that deep neck flexor endurance testing is a good test for us to be using as a marker for return to sport. We want them to be able to hold that position for at least a minute. And if they wear a helmet for their sport, they should be able to do at least a minute with their helmet on. We might start a bit low giving them biofeedback so they can learn how to use it. Use a sphygmomanometer under the back of that neck there below the occiput.

Gentle chin tuck without the SCMs activating, started at 22mm of mercury, 10 x 10 second holds, and they slowly progress, but it's not shoving the head into the ground. It's performing that chin tuck. It is the skull flexing on the neck, not the retraction of the head. Working on that biofeedback.

As they get better at that, we can start to load it with bands to provide more resistance, build up the endurance of how long they can hold those things for biofeedback, 10 x 10 second holds. As they start to know how to do it, start to load it up with bands and start increasing the time, or the resistance that's on the bands. But the endurance is the key. These things (deep neck flexors) are going to need to last all day. We would rather have them being able to hold it for two minutes rather than being not able to hold a black band for 10 seconds. We want to go with the endurance first, then we can start building the load. Build them appropriate to what they need. Do they need to be on their back first? Do they need to be seated? Do they need to be standing? Make the clinical determination based on who's in front of you and what their tests show.

This one's an important one. We will see a lot of people when we are doing this joint position sense testing. What we want to do is we want to make sure that when we start this rehabilitation, we want to take people's vision away. We want them, when they are doing joint position training, eyes are closed. You have them turn their head, they come back to the middle, and then let's just say they did this, then they open their eyes and they will say, "I overshot".

But this is where it is, remember where this is, they come this way and they come back and undershot that time. We want them to learn how to reposition themselves via that neck. Obviously, their ears are involved with that one as well. Because, here is another little clinical pearl for you, the human brain compensates with vision a lot. So, when you have a neck problem or a vestibular problem or a balance problem, what you will see is that a lot. And you do need some more of the advanced imaging, advanced sort of testing modality.

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So, force plates to enhance your BESS testing, some of the goggle-type testing to remove vision, so to measure some of these things. It just gives you more of an objective number. But what you can almost know for certain, is that people with neck issues or vestibular issues, they will start relying on their vision. They will compensate from their vision. They will upregulate their visual system. You will hear the stories in the history taking, when the patient tells you “I need to turn on the light when I get out of bed at night, because I bump into things”. Because this person relies on their vision, these are the types of people that don't want to drive when it's dark because they are relying on their vision so much.

These are the people who may be walking down the aisles in the shopping center, and they go to the shops, and just everything sets them off, because everything's moving in their visual world, and their vision's too slow to process it, because their ears and their bodies should be doing it reflexively.

It is important when we are doing this neck training you start with eyes closed, and they learn how to know where they are without their vision. If you find that when you were doing that joint position error testing before, you know, listen they come back here and they showed that they were outside of the yellow on the target. So outside it is four and a half degrees.

On the right, when they turn to the right and come back, but on their left, they were fine. If we just said it was just neck and you cleared out all the vestibular testing, you might be doing some manipulation, whatever your chosen technique is. You may be doing soft tissue, needling, taping, whatever it may be. Maybe doing that through their neck, and then you are getting them to do, proprioceptive retraining on the right with eyes closed first.

Once they have built up their accuracy, then we can build them to working with eyes open, tracing different patterns. So, then their eyes stay on the target, and the target is the laser, and they are following the laser with the eyes. What do you need to be able to do that though? You need gaze stability. To progress through some of these exercises, there might be another step that's needed elsewhere, but you might be able to do this neck rehabilitation, because you don't need your vision right now; they could be doing that while they are also doing some right gaze stability work to focus on being able to keep their eyes stable.

And then as we build along, we build each of the systems up, we can start to couple them. But your assessments are going to determine what it is that person needs. The return to learning and the return to sport, the way to look at this is going ahead of everything we have talked about. You do your evaluation. You are figuring out what is happening with that person, and you make your diagnosis, thinking this is a concussion.

And you have identified that there is something wrong with their balance system, with their vestibular system, with their neck, with their autonomic nervous system. You figured that out, and then you are giving them the advice of one day rest. And then on day two we will start to move them into activities of daily living, and we want to start getting exercise. Now don't view the exercises of return to sport. View the exercises that we are trying to enhance cerebral blood flow, and that cerebral perfusion. That's the purpose of the exercise.

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While that is going on, for our student athletes, remember the athletes. But in your offices, you are most likely dealing with the general population as well, so you may have some children, adolescents coming into your clinic, that were not part of the event you were working on the sideline for. These people have come in after already having sustained a concussion. As you are assessing them you are working through this return to learn strategy.

As you can see, it's all based upon 2 out of 10 symptoms. If it hits a 3 out of 10, that session's over. You rest, and you can go back and try it throughout the rest of that day or the following day, but the progression through the return to learn strategy, as we said, is symptom limited, but they have got to be able to get through returning to school full time before they are allowed to get back to sport. That is why I put this first, because they are getting back to school full time, all classes, all their homework without issue, is the priority over back playing sport.

Return to sport strategy. The quickest you can get through that entire phase is a week. So, the quickest that we can get an athlete returning to sport is one week but be aware that this whole process may take a month. With your testing, if you don't base everything just upon symptoms, because someone might say to you, I have no symptoms, but you watched the person get knocked out which is one of the key observable signs to remove someone from the field of play. They have been knocked out because of a head collision.

You remove them from the field to play. Hypothetically, let's use this example that the person has been knocked out and has no symptoms. And you go through this return to sports strategy, the catch is they have no symptoms. So, each day, they are going step one, passing that, step two, they are passing that, step three, they are passing that. They have no symptoms, so they have no symptoms at rest. This example they have no symptoms doing step three, so they get clearance to go back to sport. We can go to step four, step five, step six. But what if you did your exam and you found that they had a balanced problem where they are falling to the right, where they have got a vestibular problem, or they have got orthostatic intolerance. Now, we are saying, taking this hypothetical case as our example, the person has no symptoms, however most of the time, these people are going to have symptoms when you do the testing. But maybe they were lying because they want to get back to sports. So, they are trying to tell you they don't have symptoms, however this is where your objective testing becomes important.

Because we know that if someone's got a concussion, research has shown that there is an increased risk of a lower extremity injury or a subsequent concussion just because they had a concussion. We want to make sure that we have our objective tests and that we address those objective tests and get the person performing the tests as well as we can. So, we have a nice return to sports strategy here, which is based upon symptoms. But please remember we have our objective testing that we want to be doing as well to match up as we are working through that return to sport strategy.

FICS requires any of the doctors working at international events to update their knowledge of head injury and concussion every two years to remain current. 2023 we have upgraded

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everything into the SCAT6 and the SCOAT6. And this is our online module to start to introduce some of these rehabilitation strategies and understanding what these assessments mean, as we start to put some of this understanding together. Review these slides, open some of your neuroanatomy textbooks, go and do a couple of additional courses that you can do, to better understand other tests to upskill yourself to become further leaders in this space in a very topical area, obviously, concussion assessment and management.

Happy to answer any questions. I hope that you have been able to take some things out of this presentation and be able to implement them with your patients when you see them next.

To reach me you can email admin@ficsport.org

Take care, Brett Jarosz.