Academy of PHYSICAL MEDICINE

Transcript

Fascia
With Leon Chaitow

APM: We're in London because we have, once again, have the opportunity to interview Leon Chaitow. As I've said before, he doesn't need much of an introduction. He's been practicing osteopathy for over 55 years. He's been lecturing for over 35 years in colleges of chiropractic, osteopathy, massage across the world. His book tally now comes to something in the region of 80 numerous papers in various journals. He is, of course, the founder and the editor in chief of the Journal of Bodywork and Movement Therapies and he's here this evening to talk to us about fascia. Leon, a warm welcome, once again, to the Academy of Physical Medicine.

LC: Thank you, Steven. Thank you. I'm exhausted after this.

APM: Well, one of the things that Feeding the Fish do is that they do their…well, part of their act on Hoverboards and I'm hoping at the end, we might get you a Hoverboard as well.

LC: I look forward to that.

APM: And dress you up in some LEDs. You've got a couple of books on fascia, haven't you? Fascia and fascial dysfunction and I think you said there's some new developments.

LC: It's interesting that the first fascia book, which I don't know if it's showing on the slide, was 2012. That was with three co-editors and it's now getting to the end of its useful life because so much of it is out of date. In 5 or 6 years, it's gone. The other one which is this one, which is the fascial dysfunction which is what it…it's what it says it is looks at the assessment and management of fascial dysfunction. That was published three years ago and I think by next year, a year after, it's going to need a complete revision because so much new work is emerging which helps us to understand what happens when we treat the body, whether we're directly trying to address fascia or whether we're working on other soft tissues. There's no way of avoiding working on fascia. You may say, "I'm working on a muscle," or, "I'm

working on a joint." There's no such thing as a joint without fascia or muscle without fascia. So we actually don't know quite what tissues are responding but the research is beginning to show us how manual therapies and movement therapies affect the soft tissues generally in more detail than we knew before.

APM: And I think you…because you need to run through this basic science, we're actually going to do something which is slightly unusual for our productions which is that we want to run through your presentation on fascia and the development before we get into a little bit of practical and so on. But that shouldn't stop you asking questions as we go through because we will interrupt to take those questions and —

LC: Just to explain, the actual…my presentation on the basic science of fascia is a two-day presentation. I don't think we've got the time for that so I've selected a dozen or so key bits which sort of gives a broad spectrum glimpse at what's going on and hopefully, in a short period, I can cover that. I hope there'll be questions and then we can try and demonstrate what emerges from that in practice.

APM: But if people want to attend your two-day presentation and learn more then they can find the details of your lectures at leonchaitow.com or…

LC: They might be able to.

APM: Might be able to.

LC: Mainly, if I'm invited somewhere, I let the organizer do the promotion. So I don't promote. The next fascia course is in New York in May. If anyone wants the details, look at the web, you'll find it.

APM: Well, if we get sufficient interest in this and then maybe we can arrange something that's a little bit more convenient for our London audience and…

LC: I don't really tend to promote those things, as I say.

APM: because they come through another organisation

LC: They happen.

APM: Indeed, OK. So what would you like to tell us about fascia?

LC: So if we can look at the image on the screen, the title I've put together here, obviously, "Fascial function, dysfunction." The bits that I want to spend a little time on in a few minutes are the repair and rehabilitation, the homeostatic function. The more we learn about how connective tissue repairs itself, the more it becomes obvious to me that we don't need to learn

a whole host of new techniques. The techniques that are existing, maybe with a little refinement especially in terms of dosage in particular, how much force is used, the degree, the direction, the duration of compression or stretch, it really is critical but the actual techniques that we use don't need to change very much, more the refinement of how we use them. So I'm going to spend a little time on that but I want to get through some of the…the big picture first, just to touch on osteopathic links with fascia, Andrew Taylor still. These key points, to me, say all we need to know and especially this second bullet point here, the body is self-regulating, we are working with the self-regulating mechanisms of the body and in fascia, that will become obvious as we go along but the new research in that gives us a clue as to what's needed and the structure and function reciprocally interrelated speaks for itself. So I put that up just to remind us that he, 130 years ago, Andrew Taylor still was pointing to the importance of fascia. Nothing has changed except we know more about it now but he had great insights. Again, just a background, philosophical point on this character Sally, when we're dealing with dysfunction, whether it's pain or limited range of motion or general ill health, we're dealing with failed or failing adaptation and as adaptation fails, we have to look at what is the practitioner's role and as I see it, she helps me understand and patients…I show her the patients and I say, "Well, you're Sally. I'm Sally. We're all Sally," and in fact, if we're dealing with a shoulder problem, your shoulder may be Sally. It is dealing with adaptive demands and that's all this baggage, what's happened in the past, what's happening now and our role is quite simple. Remove adaptive demands or enhance function or treat symptoms. Basically, that's all we can do. So our role in working with fascia is how can we enhance its function, how can we reduce unnecessary demands on it and that's about it. So those are the philosophical backgrounds to it. Let's just look at a little bit of it. First thing I wanted to clarify is if I'm talking about muscle…let's say I'm demonstrating later on and I'm saying, "Well, we're working on upper trapezius." Upper trapezius doesn't actually exist. As an entity, it is…this is not upper trapezius but it is a muscular structure with all sorts of fascial layers and interfascial structures so that the…talking about a muscle problem, you're talking about muscle and fascia. Talking about a joint problem, you're talking about joint fascia, all the soft tissues. So the image here is just meant to show the continuity and the relevance of that, we'll see in a moment for as when a muscle contracts, it transfers load via these fascial connections. This particular one, let's say in the forearm, runs from…skin is up here somewhere then you've got the fatty layer and then you've got this fascial band running right down the bone. If this part of the arm, the muscle contracts, that will transfer load through that band of fascia, through that sheath and into other structures and that becomes a key feature of how we need to use fascia to understand it and this may be more relevant. So here, we have a cross section through the third lumbar. We have our thorax and with all our ribs and then there's a little gap below the 12th rib until we get to the crest of the pelvis and that…this is a cross section through that point. So here, you have the…maybe the 2nd or 3rd lumbar. Third lumbar, this is and here, you've got a whole range of muscles.

You've got psoas, quadratus, the paraspinal muscles, latissimus dorsi and then the various abdominal muscles and there's rectus abdominis in the front. All of those structures meet or their fascial connections meet at a little, tiny junction called the lateral raphe and the lateral raphe is seen on the photograph as…here where it is called L-I-F-T, LIFT, lumbar interfascial triangle. That is a triangular shaped column which runs from the pelvis right the way up under the ribs. The only bit of it we can access is here at the third lumbar level just between the 12th rib and the crest of the pelvis and why it's important is that load is transferred from all of these muscles through this seam, this column to all the other tissues so that when we get a contraction in one, load is transferred to others and this becomes critically important in our clinical work. When we look at this image, so here you have the thoracolumbar fascia which we saw a little bit of and if you look at this image, all of the directions of load from the connecting muscles, seratus and latissimus and the abdominal muscles and going down the legs to the hamstrings, this is a remarkable connecting hub and the relevance of that clinically…let me show you on the next slide. We are going to look at two little video clips here. This is going to last for about 10 seconds at the most and the next one I'll show you for even less time. This is a side view, an ultrasound side view. Here we have skin and then we have layers of superficial fascia then deeper fascia and different layers. We'll see what happens when that individual, lying on a mechanical bed with ultrasound imaging, looking at the lateral aspect, when it is flexed and extended and you'll see how fascia should move in a normal functional person with no low back pain and if I click that, why is it not moving? There it goes. So you see the sliding function as one layer moves on another with all those different muscles and… I need to find it again. Told you it didn't last long. So that's normal function. Imagine that this is what's happening in someone with back pain. Very, very little movement. Compare it again…I know they don't last. There must be a way of putting them on a loop but I don't know how to do that. So that's normal, watch the movement, sliding and gliding. Here we have lack of slide, glide. OK, if I can go backwards, one slide to the previous one, why might this thing not be sliding and gliding? Why might these different layers of these different muscles…which aren't all on the same plane. They all have to be on different planes. Why is that not moving and sliding and allowing movement? It could be because of reduction in the lubricants, hyaluronic acid. That's unlikely though or there could be fibrosis or there could be adhesions. It could also be because there's just so much tension, let's say from the hamstrings, from latissimus, from overworked abdominals. So many tensions from different directions that it can't move and that gives us a clue as to what…when we start looking at homeostatic function of connective tissue of what needs to be done. In this instance…

APM: Could I just ask a question though?

LC: Please do.

APM: You said that that tension could be the result of tension in latisssimus or hamstrings. Does that not then still mean it's the muscle you need to treat, not the fascia? Because that's simply connecting the two tensions.

LC: Let me just go a little further and maybe it'll become clearer. It's a good question. Let's see if you still want an answer to that in… let me go to the next one. I'll come back to it in a moment. Pectorals. OK, again, it's showing fascial continuity. We're going to do this to you later, Gary, who's our model for today. I'm going to show you how addressing…using the axillary folds here, it's possible to put tension into tissues that connect the cervical spine…the cervical area rather with the thorax, with the upper arm, all the way down to the diaphragm. So that's just getting a sense of the fascial continuity. This gets closer to answering your question. When the hamstrings contract, they transfer load through the sacrotuberous ligament, across the thoracolumbar fascia up to the contralateral latissiumus. That's the pattern or if latissimus contracts, its load is transferred in various directions but amongst others, across this gluteus maximus here and down through the tensor fasciae latae to the knee, suggesting that problems in latissimus or the upper area of the thoracolumbar fascia could be creating stress in the gluteals and be responsible for knee problems and vice versa. So it gets even more interesting. Well, for me it does. This is the work of Franklyn-Miller in 2009. His team took a fresh body, a fresh cadaver and they loaded spring…they put little spring loaded devices at the end of various muscles and they wanted to see, before the…while it was still relatively fresh, if they stretch something, where did that load get transferred to? When you stretch a hamstring, obviously the amount of force you're using is 100% of the force you're using. That's the load. Iliotibial tract, going sideways, therefore, 240% of that load. Ipsilateral lumbar fascia, 145%. So they put the whole thing the other way around. I reckon I find someone's got a tight hamstring. Is it a tight hamstring or is 240% of the load coming from tensor fasciae latae, iliotibial tract tightening or is it coming from above? So what I'm feeling in a muscle may not be in the muscle. It may be load being transferred to it. Does that sort of answer your question?

APM: It does and I'm sure we'll see…when you do your demonstration on our model, Gary, I'm sure we'll see exactly how you would approach that and to some extent how you —

LC: With some confusion. Now, OK, so this gives us a sense of load transfer and force transmission and how…that we have areas of coordination and that lumbar interfascial triangle is one of the key ones. It's the bit that you can just about feel if you come in obliquely, just above the crest of the pelvis and move towards what you might think you're approaching quadratus lumborum but you're not. You're getting towards that little triangle. You're a little away from it but unless you've got razor sharp…OK. This just looks at the living evidence. Here you have someone lying face down under controlled conditions and the various devices measuring what's happening. She's being

asked to contract latissimus. So she has something under her arm, a little pad and she's asked to squeeze that against your chest. She contracts latissimus and there's external rotation of the contralateral hip. That's force transmission. Now if you do that as an experiment in a workshop, you'll find that you can feel it on some people, not on others. It's not getting transferred. Something is stuck. The force cannot be transmitted because the layers can't slide. So I think this is quite a useful bit of in vivo evidence that it really does matter. This is just to give a sense of postural dysfunction and the various changes that are needed. If you look at this young lady over here, you can see that if she grew up without any interference in terms of posture, she'd end up looking very much like this. She already does but with appropriate treatment, she ends up looking like that. Rebalancing the soft tissues, the fascial structures…and this is one from…this is from Tom Myers' work and he just talks about unhooking the deep downward pull of the deep fascial line and so on. This is his particular model and he's done a chapter in the fascial dysfunction book on this and I just think it's useful to see. Here we have someone and she might come in…she could be Sally, that image we saw earlier. She's likely to have neck pain. She's probably got headaches periodically. Certainly, she's got protraction of the shoulder. She's going to have shoulder problems. The spine is not very happy. It'll give trouble in the end. Her breathing is not very good because the whole lower thorax is crowded. She's going to have abdominal problems and pelvic problems and probably because of the position of the knees. So you could look at her and say, "If not now, you will have knee, hip, back, pelvic, probably chest pain, upper back pain, neck pain and headaches."

APM: You know, I've always wondered myself that we tend to take from our textbooks what we regard as a postural norm, an optimal posture. How do you go about assessing what is normal for that individual? Because she's not necessarily going to naturally adhere to that anatomical picture —

LC: No and look at me now. My posture's not fantastic, is it? The problem is we can all adopt…I learnt a long time ago how to align myself, Alexander technique wise but within a few minutes, I'm back to here. We have habitual patterns. They do have long-term effects. I come back to the model of dysfunction and adaptation. Sally is adapting. If she came to me…if this was Sally and she came to me with those problems and she said, "I've got a neck pain," and I looked at this, I looked at her posture, I say, "Well, yes, we could play around with your neck and shoulders and you'd feel better for a week or 10 days or a month then you'd be back," because we're not dealing with the underlying feature. So we've got to remove the baggage from Sally which is her postural problem, is a long term…she's carrying that baggage and it means that she will not be as functional when she does whatever she does whether she's…whatever job she's doing, whatever sports she's doing. It will lead to problems. So I stick with that symptoms represent failed or failing adaptation. How can we remove the adaptive demands or enhance the

function? Part of that would be if she's willing to work on her posture, work on her breathing, work on…

APM: Is there a difficulty in this? What I drew from this is you're saying, "Well, let's treat this little girl because she's going to get problems later," but —

LC: Well, or her parents brought her and said, "We don't like the way she stands."

APM: Yeah but is there not a possibility that actually she won't get problems later because that's just normal for her body?

LC: Well, that is not normal posture. Head is forward of the center of gravity, is going to be stressing the neck. Her shoulders are already going to be problematic. Look at that poor belly. Her lower thorax is crowded. She can't breathe. So she's going to be an upper chest breather. So the symptoms, you can predict that this 8, 9, whatever year-old, by the time she's 20, 25 is going to have a whole range of symptoms. I'd rather her then but who's willing? I've had patients come in with worst posture than this, youngish men, women and with neck or arm or hand or knee pain and they want that fixed and I'm not happy to fix them. I'd rather they went to a fixer. I say, "If you want, we can try and take a…I will work on your neck but only if we agree that we're going to do something about the bigger picture." Anyway, that's how we choose to work. Let's just quickly…now we move from the big picture. Now we get to the small stuff and this is where the dosage material comes in. So these are fibroblasts. Fibroblasts are the cells that manufacture extracellular matrix and collagen and if you injure yourself, they leap into action and create the scaffolding for the wound repair and they are mechanosensitive. They aren't controlled by the nervous system or by anything else. They are responding to tensions and architectural shape. And so from fibroblasts, they become myofibroblast which are giving you that support. Here is what happens. These two fibroblasts started life the same size. The purple one is on a more rigid plastic and this one is on a softer plastic. They need a certain amount of tone. They attach to whatever surface they're on by these little pseudopod things, little attachment features. So they are holding on. This one is holding on to something firm, fairly rigid and it's growing big and strong and healthy. This one's on a softer surface, it's not. The message from this is…the title gives it away, Wipff and Hinz in 2009, "Myofibroblast work best under stress. They need a certain amount of tension load and deregulation of these activities leads to contracture." Now this gets a little bit technical but I hope not too much. This is where we get to the homeostatic function of collagen, how does it fix itself? We have a triple helix. We have like three strands knitted together that makes up a fibril and those fibrils linked together…if you can see on the screen, these dark links and as we get larger and larger, we end up with a rope-like structure. So the mechanical strength of the triple helix is due to the links between the helixes, helices and the links between the triple helix and the microfiber. That's

where its stability comes from but it is basically an unstable structure. OK, I'm not going to spend time on this except to highlight this article. If anyone wants to read up about the way in which collagen repairs itself, this is the article to read. It's very new and we're going to spend just a few minutes learning about it because it gives a strong indication as to what to do. These are fibrils. Each one of these is one millionth of a meter away from the next one, one micron away. So they're pretty small and these are the…this is looking at enzyme activity on them. When these fibrils are not adequately tensioned between themselves and by external forces and by internal hydraulic forces…I'm sorry. I lost my mic. Did that cause any…?

APM: No, you're still fine.

LC: I'm OK. I'll leave it there. So for stability, they require external forces, tensions, pressures and links between themselves and others and then internally, hydraulic pressure. Let's go to the next one.

APM: Out of curotsity, how is that picture being produced? What sort of imaging is that?

LC: It's electron microscopy and it's very highly…I don't know. You'd have to the article and ask them, I just liked it. That's all. I'm trying to move on and…there it is. Let's just look at this. Now, we all know what a rugby scrum is. I'm using this as my metaphor because here, we have the three front rows and the three front rows. They're locked together, that's a triple helix. And then we have other forces. We have the locks and the eighth man and the flanks, all creating, hopefully, adequate tension so that this thing doesn't collapse and this is what happens when it collapses. Fibrils unravel, they buckle, they cleave. Now that is the message from the previous slide. When there's inadequate internal or external tone on collagen fibrils, they collapse immediately. Enzymes come in and tidy up the mess and new collagen is created by the fibroblasts and you have a new collagen strand. That is self-repair. It is an ongoing process happening in you, in me all the time and it depends upon the stability of the tissues. If we have adequate tone, not imbalanced tone where there's excessive amount of force, let's say one group of muscle's hypertonic, the antagonist's hypotonic, the tension's unequal. In those tissues, there'll be constant repair going on and the chance of fibrosis. So we get the sense that if homeostatic functions are…if the amount of regeneration is reduced because we have more balanced tone, we're going to have healthier and happier tissues. Well, that's how I try to interpret this. So I think the rugby scrum helps us or it helps me anyway. The other part of the story is the osmotic pressure. If we do not have enough hydration, the same thing happens. So it's external forces and internal osmotic pressure. Collagen depends upon having water. So we can see a healthy tissue here and dehydrated, not very happy down there. Water plays a crucial role in stabilizing the structure of collagen molecule. So I always tell my patients, before they have treatment, "I want you to have a glass or two

of water." They're going to respond better. They're going to have a better outcome.

APM: Does that intake of water translate that rapidly into —

LC: Very rapidly. What gets into your system gets into…it's going to hydrate you within seconds. We go to something else which is a little bit technical. This gentleman, Popescu who died two years ago I think, Romanian researcher, looked at the connective tissue matrix, the extracellular matrix and he identified telocytes. Well, he called them telocytes. They didn't exist until he invented them. They were there but they were not recognized for what they are. This is one of the most amazing changes in our understanding of connective tissue in my lifetime. These things have been around forever. We didn't invent them. He didn't put them in but they were not recognized. What are telocytes? Connective tissue cells that are involved in homeostasis, found everywhere in the body in the connective tissue, communicate with surrounding blood vessels, nerve endings, smooth muscles, everywhere and have roles in tissue repair, etcetera, etcetera, as it says there. Now what you see here is a telocyte. It's got long strands of tissue. It is like a mobile hospital combined with a mini manufacturing enterprise. It makes…oh, as well as having a large communication network. It communicates with others of its like and with other cells which tell by chemical or other means that they need help. It manufactures things. I lost that. Now what it is? That's the next one. It manufactures whatever is needed. Let's say it's come across a blood vessel in need of help. It'll manufacture something and it'll send it off and deliver it in a little balloon of…isn't that beautiful?

APM: It is, yeah.

LC: It's remarkable. It will also reprogram the genetic behavior of other cells. It is truly one of the most remarkable —

APM: So if the blood vessel is damaged…I mean I've always taken a very simple approach, that actually the fibrins in the blood would then repair the whole —

LC: As well but the —

APM: But what does this add to the mix?

LC: The telopods. So the telopods are the little bits that stick out. OK, the long, strandy things. Communicate with surrounding blood vessels, nerve endings, smooth muscles, glandular elements, epithelia via direct homo and heterocellular junctions. Tissue, OK. Let's go to the next one and see if that answers your question. No, it won't because I've left out some of the slides that…what I would urge you to do is go and do a web search for telocytes or read the editorial I did in the Journal of Bodywork and Movement Therapies.

If you go to my Facebook page, you'll find I've got stuff on telocytes there. Just go back a few days, a few weeks, you'll find stuff. When these things are deficient, you have areas of disease and fibrosis whether…which is the chicken and which is the egg, I can't tell you. When there are not enough of them around, tissues become unwell or when tissues become unwell, there are less of them around. I don't know the answer to that and neither does anyone else. Research into them is within the last 10 years. You'll now find many hundreds of papers every year coming out. So if you go to this one, it's one of the first ones. 2011. So cells that have been with us forever weren't known about and they're connective tissue cells.

APM: Can we influence them?

LC: Can we?

APM: Can we?

LC: Yes. The point is they are mechanosensitive, so —

APM: So chemically sensitive and other things…

LC: They're chemically sensitive and mechanosensitive. So yes, we can. It's the same story. If the tissues are happy, they're happy and we can influence the extracellular matrix, the…as much as it's possible. So let's just see a little bit more of this. Maybe it'll make more sense. I'm trying to gallop through two days. Paul Standley at the University of Arizona Medical School, osteopathic department, they published a number of papers on…these are fibroblasts, four pictures. Let's look at them. These are fibroblasts that are spread on to a membrane which is slightly elasticized. So you have an elasticized membrane and, like putting butter on toast, they are spread. These are nice human fibroblasts. That slightly domed elasticized surface is attached to a cylinder which can be turned…where they can have intermittent vacuum effects. So the dome would be doing this, OK, if you wanted to. That elasticized dome can also be slightly stretched or slightly reduced in tension. They are trying to model two osteopathic approaches, myofascial release and counterstrain. This is what…to see what would happen. So here you have happy fibroblasts sitting on their membrane. Then they have 60 seconds of counterstrain which is simply unloading by 10% the tension in that membrane, for 60 seconds and they look to see what their chemical behavior was and so on, so nothing much of interest here then…hang on, let me just click on that. That should give…that's the control, 60 seconds of counterstrain. Then eight hours of repetitive strain which means this for eight hours and I think you can see that they don't look as happy. That's less important than that they were producing inflammatory products. They were actually inflamed. They're not connected to anything. No nervous system, no blood supply. They're simply fibroblasts.

APM: Is there any significance in the bright yellow dots in that?

LC: There may well be. I don't know.

APM: Not for the purpose of this.

LC: The next bit is that membrane is now put into 10% reduction in load for 60 seconds. Eight hours of repetitive strain, 60 seconds of counterstrain, not only do they look happy but there was a 45% reduction in inflammatory production. So if you use counterstrain, potentially…if we can translate this in vitro laboratory, what's happening is…what we think is happening in vivo, in the body, we're changing the chemical behavior but it gets even more interesting because this study was replicated. This was a counterstrain, unload the tissues. They did exactly the same study but this time, they added just 6% of load instead of unloading it, exactly the same result. If you added a bit of tension…the pictures are interchangeable. The same thing happens. So you can either load or unload the tissues and you'll change their behavior. Now, how do I explain this one? You know, a bioengineered tendon is a tendon that they grew in the laboratory. They took collagen from a cow and they grew a tendon. Let me see if I can get this to work. Human dermal fascia and it was mixed with fibroblast. So human fibroblast and cow collagen, they created a tendon and then they sliced it open. They created a wound in it and they wanted to see would the fibroblast heal the wound and they did. After creating a wound, different degrees of durations of strain were applied to the…in the direction of the long axis of the structure. So here, you have the wounded engineered tendon with no strain. That's the grey one. This is before treatment and there's the grey one. This is if you do nothing, that's the healing process over 48 hours. So the wound closes slowly if you do nothing and then different degrees of load, 3%, 6%, 9%, 12% and you can get…if you use 12%, you slow down the healing process. If you use between 3% and 6%, you speed up the healing process. Then it gets even more interesting if I can get this to move along. They looked at the time. So then we're using 6% now which is…and they used it for 1, 2, 3, 4 or 5 minutes and you can see that before treatment, the thing heals up all on its own if you do nothing and then B, which is about five minutes…45 minutes of myofascial release which would be a slight 6% load on tissues which is exactly what you do in myofascial release speeds up the healing process. Now if you don't find that exciting, I do. OK, let me move it along a little bit more. We're getting close to the end, you'll be glad to hear because I know —

APM: No, not at all. This is fascinating stuff. I mean a lot of this has been…people have been doing this by accident for a long time, of course.

LC: Trial and error, we found that 5% or 6% of load…you don't overload the tissues. You can get a different effect. This is from a different source. It's from tendon biomechanics and mechanobiology, a review of basic concepts in Journal of Hand Therapy. If you load tendons too low, you reduce their

tensile strength and all of these things are on negative and you…if you use moderate, 4% load, 4%, 5% load, tensile strength increases and…hang on, that's collagen degradation reduces. According to that arrow, it should be up there and the telocytes…the tendon cytes develop into tenocytes and they become healthy tendon. If you use too much, 8% in their study, tensile strength changes, again, and you get negative effect —

APM: Can you just remind us? I mean 4% and 8% of what?

LC: Of whatever tension there is on those tissues now and that becomes really difficult clinically. What is the difference between 4% and 8%? Very little. So you can do too little, you can do too much. At some conferences, I tend to finish this presentation with that old '60s song, Not Too Little, Not Too Much. There is a right amount and we tend to know that clinically but the important part of the story is that…we go back to that picture, that if we do somewhere around identify the barrier and then just lean on it, you don't have to push it. Now I don't quite know what you do when you're working with Bowen technique. I've seen it and I've felt it but I don't know if you have any sense of how much force you use. Not very much, as far as I understand.

APM: I'm not experienced in Bowen technique at all. Maybe if we have a member of the audience who is —

LC: Maybe they would know. Anyhow, this is about tendons, not necessarily about other structures. We're talking about very low degrees of load. Now, I'll try and show you when we do some practical what I think is the best way of identifying how much. This last bit is about isometric contractions, when you contract a muscle. So I have, let' say, short, tight flexor muscles which I do. I should be able to take my hand and I can force it to a 90^{o} angle but if I try and do it myself, I'm hitting a barrier around there. Because I'm a manual therapist, I use these a lot and I type a lot, so overtight. So if I take that to its easy barrier, I'm putting a certain amount of load on to the sarcomere. So here, we have actin and myosin. Here we have what we call the series elastic component. That's connective tissue and here, we have the parallel elastic component above and below. This is our best understanding of how sarcomeres work. So here I have my shortened structures and I now try and contract these isometrically. So I'm making a contraction and there's no movement. If there's no movement, the sarcomere stays the same length but it's contracting. This is getting shorter, therefore, other bits inside have to stretch. So isometric contractions are, in fact, stretches of the internal connective tissue. If you then actively try and stretch the thing, I think you'd have to stretch pretty hard to do that but you're getting a different way of modifying the behavior but the interesting thing is that isometric contractions, in and of themselves, have an analgesic effect. These are some of the mechanisms thought to happen with myofascial release and there are telocyte and fibroblast activation, hydrostatic changes in the pressure, various neurological things happen. Of course, there's a whole lot of

neurology going on. I'm just ignoring it for the moment because that's where all the focus is. A lot of it is about mechanical behavior. So there'd be central nervous system and other features which alter pain in turn and there's something else happening. You upregulate the levels of endocannabinoids. We manufacture our own painkilling cannabinoids. So a combination of neuromotor, mechanotransduction, piezoelectric, viscoelastic, hydrostatic, hormonal and other features all happening when we're working unless we're doing too much or too little. In this case, they're happening but they're not happening adequately.

APM: Modified gene expression was in there as well.

LC: Well, the telocytes will…very much so. I mean if you just go back to those four orange squares. If you take tissues and stress them for eight hours, their gene behaviors, they're going to be producing all sorts of nasty inflammatory stuff. If you then put them into unloaded state for 60 seconds, you switch off that part of their gene behavior and you're modifying their genetic outcomes. I put this up if people want it. It just gives you a sense of what the neural aspect of the whole story is, different receptors behaving differently. I'm deliberately not spending time on the neural features because that's far more studied. All fascia contains mechanoreceptors and pain receptors and nociceptors and I think that was it.

APM: It is.

LC: OK, so that was…how long did that take, 15 minutes?

APM: It's about 15 minutes.

LC: That's what I thought.

APM: Maybe give or take half an hour

LC: Were there any questions coming through on that?

APM: We had one come through as we were talking though which was taking us back to that postural question of mine which was on the issue of ideal posture and I'm sorry, I don't know who sent this question in. This viewer tends to talk about movement rather than static posture. I'm not quite sure what…I think you know what they mean there and what would you say about that? I mean how much is the movement —

LC: Surely. Static is what…when you're trying to just get a snapshot of now but if you really want to see how the person functions, you put them through a series of functional exercises and movements. I'm particularly interested in breathing. I can't assess breathing without assessing the breathing and that's touching and evaluating what's moving, what's not moving and if I want to

see…have functional movement, I'll have people standing on one leg and doing this or doing that. You want to see what's restricted and what's moving freely. That's far more important in everyday life than static posture but the two things are connected. You can get a sort of first impression. If someone has a real chin poke, head forward, round shouldered, slumped and they've been like that for 10, 20, 30, 40 years, I know very well what I expect to be short and tight. So when I ask them to make certain movements, what limitations they're likely to be but each…everyone will be different, depending upon their own history.

APM: Which leads neatly to the question which has just come in, again, from another anonymous viewer, do you think that stretching helps or hinders healing for fascia? I think it probably matters that much, doesn't it?

LC: Well, it is exactly how much. You can't stretch deep fascia. Deep fascia is unstretchable. There are number of mathematical models which have looked at it, the tensor fasciae latae…sorry, the iliotibial tract, I should say would require several tons to move anything but you could take the load off it, modify it and improve its slidability. That will help it function better. So you can't stretch deep fascia. The superficial areolar tissue, just below the skin stuff and the stuff between the deeper layers, you can modify that, we believe anyway, usually through movement and movement combined with compression and…so what is stretching? If I do that, I'm stretching. If you do it to me, it's passive stretching. If I do it to myself, it's active stretching. So I would think a combination of a practitioner's input to modify areas of fibrosis and/or restricted superficial fascial function and then the patient would be…through yoga, pilates, tai chi, stretch…other things, functional exercise, various things to improve their own functionality under guidance or on their own. I don't know if that answers the question but certainly, you can't look at posture and say, "That is static and I can make all my assessment." It's a first lead in as to what's going on.

APM: You already mentioned your particular interest in breathing which you talked a lot about that in the last broadcast that we did. A question we have here is do you think breathing has an effect on fascia? And I suppose you could look at that the other way, of course. What is the effect of fascia on breathing and can you explain your thoughts on that?

LC: So if we go back to those little slides on the homeostatic response, how internal fascial structures…because you've got fascia in the lungs, you've got them in the…you've got all over the place. As you are breathing, the rhythmic movement of that, even the circulation of blood is a pulsation. All of those are movements which are picked up by the local fascia. So I think Paul Standley's work, originally, before he went in to work at the osteopathic school and did this research was looking at the effect in arteries when they used stents or mechanical structures to try and open up blocked arteries and they found that the additional pressure from the…whatever the metallic

implant changed the flow of blood and that actually created more problems than it solved in many instances. So his original research back in the, if I've got it right, '70s…no, in the '90s was in that direction that blood flow itself is modifying connective tissue behavior, so is breathing. So all internal movements are having an effect on the local fascia and the actual action of thoracic expansion and contraction, the diaphragm movement up and down, the reciprocal behavior of the pelvic floor, all of that has fascial implications. Just one thing that I'll be doing with our model, if you'll let me…there's a lovely study. I may have mentioned this in the last talk, Norwegian research, I've got a feeling I did. They reviewed posture and structural changes in people with an upper chest breathing pattern, women with an upper chest breathing pattern and they found that they all had pelvic floor dysfunction. The most common pattern of dysfunction they found was hypertonic, short, tight psoas. Now psoas comes up from your hip area and it travels past the pelvic floor up to the lumbar spine and merges with the diaphragm. So it is a structure which connects everything from below up. So it's not known as a breathing muscle but it…

APM: But it's presumably…from what you said earlier on here, actually tension in psoas could be magnified in the diaphragm —

LC: It is. The other thing in breathing is the two muscles that always activate on breathing. On inhalation, scalenes always fire. On exhalation, quadratus always fires. So you've got that firing, firing, firing, firing and overactivity, shortened, tightened, tug, pull, including some muscle and fascia, you're going to change behavior, the fascial behavior there.

APM: We mustn't keep our model waiting much longer. Can I just ask one more question of several that have come in before we go across to have a look at Gary? Someone has asked about Anatomy Trains which is —

LC: We haven't touched on Tom Myers.

APM: I mean the question they have is do you think these are the same fascial patterns that have been discussed as postural fascial patterns and…?

LC: Look, there's a very recent paper which I did not put up because I felt I needed to limit this which evaluated, through dissection, the accuracy of some of these fascial patterns. I don't know if I've got one in here. Do I have that bit? No, it's not in these slides. Myers took the…and there are about 8 or 9 other models. Myers is not the only one. There are French ones, Mesier and others that have tried to make sense out of these fascial connections. I think this recent paper, and I can't remember the name of the author, did this series of dissections to try and see if all of the connections that Myers points to in his various chains of muscles and fascia are demonstrable on dissection and most of them are but not all of them can be, in all people because we have idiosyncratic connections. So I'm not quite sure that answers the

question. I'm sure it doesn't. There are, as I say, around a dozen different models that try to interpret the fascial pathways, connecting the plantar fascia with the Achilles tendon, with gastrocnemius, soleus and making these junctions and links all the way up to the top of the head. Now you can do a dissection where you can actually create one piece of fascia with all of those bits but not all of the pathways that is identified seem to be…that's not capable of being done in all of them, as far as I know.

APM: Shall we move on to Gary? Gary is a member of the Feeding the Fish troupe. So he is a performer, a dancer, particularly but also a singer. So breathing, I imagine, is quite important to Gary.

LC: Gary, I don't quite know what we're going to do with you. You've got various bits and pieces that aren't working as well as they might and we looked at your neck.

GARY: Neck, yeah.

LC: Let me just remind myself what I wanted to do here. I think what I'd like to do is just have you lying face downwards and there's a little face hole there and just lift up part of your t-shirt. I'll go the other side and try and demonstrate something. Are you going to lift it up or…yes, I think I'll just work that up. OK, so let's just start here. Are you reasonably comfortable, Gary?

GARY: Yes, I'm fine.

LC: If we try and do a little experiment and just see what happens, I'm going to just use finger pads to just ease the skin on the underlying fascia superiorly. So if you can see my fingers and I'm going to move these tissues and I'm trying to compare one side with the other to see if the same amount of movement is available and so far, they do seem to be pretty symmetrical. I can try coming in…because he's got some quite well developed…extremely well developed muscles here. OK, so that was a little bit more interesting. For me, that seems pretty symmetrical. If I come in from the side here, Gary, can you feel the difference left and right when I do this or not?

GARY: No.

LC: See, that feels to me very loose and pliable. That feels as though I'm hitting…can you feel my fingers? Let's do one at a time. You feel that? It goes on forever.

GARY: Yeah.

LC: Not quite. Same area, coming this way. I'm almost hitting a barrier as soon as I start.

GARY: Hmm-hmm.

LC: Can you feel the difference now?

GARY: Yeah.

LC: That suggests that the subdermal fascia, the fascia just below the skin is not as slidable as it might be. So if I put…no, don't worry. If I just put 2 or 3 finger pads on that. Now let's just try and get a sense of that resistance, restriction. I'm having to force that if I go any further. So what I'm going to do is move this in directions which seem comfortable to it. So I'm going to put these tissues into a functional ease position. I'm going to test the movement superiorly and inferior. It likes to travel superiorly. So I'm taking it in that direction. I'm trying to see if it…it won't travel laterally and it certainly won't travel medially. So I've gone superiorly and slightly laterally. From there, I'm going to check to see will it rotate? Not at all that way, slightly that way. I've gone superiorly, laterally and rotating and I'm just going to hold that for 10 or 15 seconds. What we're doing here is taking these tissues into their functional ease position, where they want to go, not forcing them against any barrier and taking them in the directions of free movement. And then I release it and they come back and I'll test it again. Well, it's a little bit more pliable, I would say. Is it normal? It's more normal than it was. As an experiment, you can try this on any tense or tight area. Look for skin features which are…where the skin doesn't slide. That little interfascial triangle I talked to you about is found here. So find your 12th rib, Gary and crest of your pelvis and we've only got that couple of centimeters of space to access deep in that little triangle which is somewhere around here. We've got a barrier of the ribs and we've got the pelvis. So here, we have just a very small aperture. If I want to touch that or touch the…get close to it, I have to feel laterally to this muscle mass, the paraspinal muscles and gently, gently, gently, with a soft contact, ease my way into those tissues and come a little bit obliquely superiorly and if I go deeply in there, do you feel a slight discomfort yet, Gary?

GARY: A little bit, yeah.

LC: Is that just a local feeling or is it spreading at all?

GARY: It's just local I think.

LC: Now if I was doing this in the serious way, I would be standing on that side and I'd have…if I was trying to treat these tissues where we have a junction of all the abdominal and the back muscles, everything meeting in this, what's called a lateral raphe in the…I would apply deep compression there. I'll show you what I'll be doing but I would be standing on the other side. Let's say I

had accessed the point we are going to try and use something called counterstrain. That's a bit uncomfortable, yes?

GARY: It's all right.

LC: I need you to just at least sense discomfort, not pain.

GARY: It's a little bit uncomfortable, yeah.

LC: Tell me if that pain changes or how it changes if I put a little bit of compression through the pelvis like that.

GARY: It seems to ease it.

LC: It's eased?

GARY: Yeah.

LC: By what, 50%, 100%? What?

GARY: It's almost 100% maybe.

LC: Almost and that would be treating using counterstrain, this lumbar interfascial triangle and that would have an effect on the muscles attaching to it. If I did that and it didn't work, I could put compression from here or maybe from the lower leg or maybe from here. One of those would modify the tension in the tissues and make it easier and unfortunately, we found out you can touch your toes. If you couldn't touch your toes, I would do something similar to that in these type paraspinal muscles and you'd be able to touch your toes. OK.

APM: Well, he may be able to bend further forward now —

LC: Well, yes.

APM: Leon, I was actually going to ask just as an aside here, is that triangle…is that readily identifiable in a dissection or —

LC: Yes.

APM: It is.

LC: Yes and it is a little…because it's not only here. It runs all the way up but you can only access that bit of it and the paper that I referenced in that slide by Willard and Vleeming, it's well worth reading. I mean it's a remarkable research. OK, so we've looked at the possibility of using just a superficial, functional technique, taking tissues where they want to go. I've also given

you a sense of…you can modify tissue behavior by using a counterstrain which means create a discomfort and then take it away by unloading it. This is what they were doing, unloading with that distressed fascia. OK. So we're going to play with your neck, won't we?

GARY: Hmm-hmm.

LC: So if we can have you turned over to your back. Now, I'm wondering where I'm going to place myself. If I go there, it'll collide with this thing. OK, does this table rise at all?

APM: It's quite difficult to make it —

LC: Quite difficult for it to rise. Quite difficult for me to lower you see. What we didn't do is…what the camera didn't see was your neck rotation. So I wonder if we could —

GARY: Yeah.

APM: Will it help if you got a chair? Could you do this sitting?

LC: I would prefer to do it sitting, yes, if I could have a chair there.

APM: So let's have a look at your neck —

LC: So we don't do anything exotic, Gary. I just want to have you sitting nice and tall. I want you to do two things. In your own time, try and take your right ear down to your right shoulder and just get a sense of how far that goes comfortably, not forcibly and then the other way. Does that feel more restricted? Does it —

GARY: This side. I think so, yeah.

LC: It doesn't quite…it looks as though you hit a point of…

GARY: Yeah.

LC: It doesn't want to go further. OK, let's just see what happens when you try and turn your head and the other way. Do they feel the same or…?

GARY: Pretty much the same actually.

LC: Pretty much the same? One more time, just the rotation. It's about there, OK. Yeah. So it was…that side flexion was the most restricted —

GARY: Hmm-hmm.

LC: Let's have you lying down and while I've got you there, can you just put your arms above your head? Just let me see how free and easy they are. OK, so there's a little bit of shortness in the pectorals. Your arms should be able to lay flat on the table but they can't. So we need to do something to these pectorals in a minute. We'll do that. First, we'll see if we can get your neck to move more freely. So my first evaluation will just be to see how Gary's cervical spine moves when I try and just take things gently from side to side. I'm just trying to see each segment whether it wants to freely translate and then I'll try and explain where fascia fits into this. So I want you to give me feedback, Gary. As I'm taking this, where I'm touching you here, I'm going to move that to the right and the same level, I'm going to try and move you to the left. Does one side feel more gummed up than the other to you? Going that way.

GARY: Going to the left.

LC: So if I'm going to the left, it's like hitting a brick wall there.

GARY: Hmm-hmm.

LC: Now, when you have…I don't want to get too biomechanically technical. When you have a restriction in side to side translation movement in the neck, it unmasks or reveals underlying restrictions in side flexion and rotation. So I'm going to take Gary's neck in the direction that it is most restricted. I'm just going to hold it at that barrier. We need to talk about this whole 4%, 8%, 12% in a minute and what is a barrier. So I'm holding it there, Gary and I want you to try…you remember we looked at the inside of a muscle when you contract it, what happens. The fascial component lengthens as you try and shorten the muscle. So we're going to ask you to do a couple of things. Very gently, try to side flex your neck to the left against my hand, just very gently. Just hold that 3 or 4 or 5 seconds and go loose. Now that isometric contraction would've loaded the sarcomeres and some of the fascial tissues and I can take the tissues…the segment that I'm holding slightly further in the direction it doesn't want to go. This time, I'm going to ask you to try, while I hold you fairly firmly, to take your head back against the pillow. Just try and extend your neck, just push back your head. So we're just getting a general isometric contraction, 3 or 4 seconds, stop and go very loose. And that moves more freely now. So let's try from the bottom again. I'm just going to take it from side to side. Again, do you feel a difference down here, Gary, if I go that way compared with coming this way? Is there a slight difference or does it feel the same to you?

GARY: Pretty close, to be honest.

LC: But I want…even slight is good. What about up here? So here, we have free movement. I think that feels —

GARY: Yeah.

LC: You don't like that, do you? So I'll hold that one in the other direction, towards the right and this time, I'll ask you to try and bend your neck, so if you put your ear down on to that shoulder very gently, very gently and stop now. OK, now what I want you to do is try to bring this ear down to your left shoulder. Gently, gently. Too much effort and go loose. OK, let's just reevaluate that segment. OK, we're nearly done. OK —

APM: Is this counterstrain that you're doing now, Leon?

LC: That wasn't counterstrain. That was a bit of muscle energy technique because I was taking tissues to a barrier, getting him to contract various muscles and seeing how that changed the movement. So this is muscle energy. Can we sit you up and have a look and see whether your rotation has improved? Now could you side flexion to the left. Let's see what happens if you go over to the left now. Are we getting that same pull here?

GARY: It's easier.

LC: Come this way again. OK, now this way. OK, sit further back, if you would. I want you to take it as far as it'll comfortably go that way. OK, so he will do this seated. What I want you to try and do…you can feel my forearm —

GARY: Hmm-hmm, yeah.

LC: So I want you to try and side flex into it but in this sort of little rhythmic pulsing movement. I'm just going to give you a lot of support here. Is my watch bothering you?

GARY: No, it's fine.

LC: I think it might well bother you. Could you take it off? You can't have it. OK, let's put it down.

APM: It's yours.

LC: Do remind me. So I'm right into his…the angle here of the neck and supporting him. Are you comfortable?

GARY: I am, yeah.

LC: Now, the action I want you to make is pulse into my arm. I'm going to give very firm resistance. So can you just…smaller than that and faster. You're a dancer so you can do that. Terrific. Well done. They learn quickly, these dancers.

APM: Anything with rhythm, they're fine.

LC: That's enough, about 10 or 15 of those. We're getting many isometric contractions here, very non-stressful and very educational proprioceptively for his muscle control over that area. OK, and just let's see if you can take your head further. That's better, OK. And just let me see your rotation. OK, we'll try and get that to go a little further. This time, firm…hold here, firm support for your head. Try and turn to the right in many pulsing movements. Smaller and pulse and pulse and pulse and pulse. That's it.

APM: Is there an optimal frequency for this?

LC: This is developed…I'll explain in a minute. There is. That's enough but no one can do it. Can you turn further to the left now, please? Oh, gosh, you're nearly coming off. OK, come this way. OK, we'll try and get it to move a little further this way this time. So I'm just holding you firm, flat hand. That's got to be really solid for you to work again. So pulse into my hand, turn and turn and turn and turn. A little bit more rhythmic, pulse and pulse and pulse. The developer of this was someone called Rodi, Thomas Jefferson Rodi and he trained with Andrew Taylor still in around 1900. And turn both ways now. Terrific. You feel the difference?

GARY: Hmm.

LC: Hey, look at that. You haven't been there for years. Rodi's work was remarkable. I think I talked about him in the last talk as well and he developed this idea of retraining proprioceptively not just the large muscles but the intra-articular spinal muscles, rotatory and so on through many contractions. What we now know from the fascial viewpoint, why is this important, many contraction's going to change the fascial behavior in the sarcomeres. OK, what was the next thing I said I was going to do apart from —

APM: Before you move on, if I may, we've had some questions come in, not surprisingly. First of all, relating to when you had Gary lying on his back, are you holding his neck in the position of ease or barrier of restriction?

LC: Answer is either. I was doing it to…into ease. If you take it into ease, it becomes a functional technique. If you take it to the barrier of restriction then I would either use this bouncing pulsing or sustained muscle energy. So you can either work direct or indirect. Next, I'd like to work indirect, if I can because he's young, strong, able. I can do some active work as well. And yeah, just one second. Let me finish the thought. But what I was doing down there was functional. It was indirect.

APM: Anna has joined the conversation and said, “Do you feel any specific isometric contractions in the neck help release cervical meniscoid entrapments?”

LC: I never know what’s being released. What I’m looking for is a change of function. No pain, reduction in pain and do actually know what’s going on inside? I go back to Andrew Taylor still, that image, the body is self-regulating. We are removing obstacles to self-regulation. So I’d say my answer to Anna is maybe but if she knows something I don’t know, tell me.

APM: Thank you and final one before you move on. Have you used seated thoracic rotation, isometric contractions to help relax acute lower back pain episodes?

LC: In a number of ways, we could do it seated. I actually prefer to do it side lying. So let’s have you seated with your back…which way are we going to do this? You’re going to have your legs over this side. I’m going to come there so I’m going to block the view of the camera. So you sit there. Move back a little bit. So we sit side by side. You fold your arms right onto your shoulders, if you would. You’re too tall. OK, I would normally have a firm cushion here so that I can really get Gary solid against me. So let’s just say…so what was the actual part of the…it was sort of thoracic —

APM: Seated thoracic rotation, isometric contractions.

LC: Get very loose, Gary. Let me take…. So let’s just say…because this stuff is very tight. I’m going to take you into slight flexion, side flexion and rotation. OK, we’re hitting up…I mean this is really rock solid. So we’re going to try and change that…the behavior of that a little bit. I would take Gary into a barrier. That means I’m not forcing him through the barrier. A barrier for me is the very first sign of resistance. So I’ve taken Gary into flexion, side flexion by putting a little bit of compression from my axilla and a little bit of rotation until I feel…that’s the barrier. How can we change that? Now, there are lots of different ways. I can put a hand on those lower ribs and say, “Gary, let’s try the pulsing again. Can you, where I’m holding you, very firmly there, pulse into my hand with that part of your chest?” Now, you did it about 500 times stronger than I wanted. I want a mini pulse.

GARY: OK.

LC: Even smaller. OK, that’s lovely. You won’t always find people who can learn those quickly but I know dancers have to learn routines extremely quick. So you want about 10 or 15 of those, maybe 20 in about 10 or 15 seconds and go very loose. I take out the slack into flexion, side flexion and rotation. I don’t force it. It just goes further. Let’s try something a little bit more obvious. I want to improve the flexion of this. So where I’m…my hand is here, exactly there but not below that, just here. Try to bend backwards over my hand and now stop. You’re going to do that as a pulsing again. Extend and

stop, extend and stop. So we have a rhythmic…now this is having a number of effects because Gary is…it's going to release and relax those tissues but it's also connecting his brain with bits of his anatomy that he probably didn't realize there was a connection. Go very loose now, OK. Back into more flexion, more side flexion, more rotation. If we wanted to do a sustained isometric contraction, I could simply say, "OK, as I'm holding you, Gary, try and just derotate and sit up but use only about 10% of your strength. Just twist back and sit up," and I don't let anything move and go loose and we flex inside, bend you a little bit further. This time, try to bend further and twist towards me but only 10% of your strength. Terrific, well done and enough. Just 3 or 4 seconds of that and it changes. If I wanted to work on individual segments, although it would take me a long time to explain that but you could actually work segment by segment by segment. The other way I would deal with this is to ask you, "Are you still OK?"

GARY: Yeah, good.

LC: If I could have you lying on your side, so your head is up there and your feet is down here. OK, you'll your little, tiny cushion. Can I have all of you a few inches further? So I want the weight of Gary's legs here. So I can play around with his flexion and extension. We find a point here that I want to…let's just say I want to change this a little bit. I can let his feet drop a little and I can say, "OK, putting a little bit of tension on to these tissues as it side flexes and rotates, try to take your feet further towards the floor, sideways." So we're getting a contraction of the antagonist and go loose. Relax completely and we can take it a little further or I could come the other way. You've got a back problem. So if anything I do causes you pain, you let me know. OK, so I'm just engaging a rotation barrier here by lifting Gary's legs. I want you, with your ankle, where I'm touching you, just the ankle, push down to the floor and stop and push and stop and push and stop and push and stop. So we're getting a rhythmic movement here. How much of this is fascia? Well, a percentage. You can't separate soft…keep doing. You're doing nicely and go loose. OK, so I don't know if that answers the question but that's the sort of thing I would do if that was what I was asked.

APM: You know, we've got 5 or 6 minutes left. It's gone so quickly —

LC: You're kidding.

APM: Really, we are very, very late into the broadcast already. There's some other questions that have come in. One of which was how do you transition between treating fascia and muscle? And I imagine you…because you —

LC: Let's just have you lying down again on your back. Firstly, I want to just, before I touch on that, talk about barriers. The first…if I take Gary's head and turn it, OK. Put your arm by your side. As I take…OK, Gary in full rotation. If I take it in slight side flexion, at a certain point, his shoulder will start to move.

I would've gone past the barrier. I don't know if you can see a little bit of his shirt there, a little dimple. So from neutral, as I go into side flexion, OK, that's the barrier. This should be about a 40° angle from.. it's not, it's about 20°. So there is shortness in upper trapezius. If I'm palpating those tissues as I rotate Gary, as he go into side flexion here, as that begins to tension and move, I would feel tightness in the muscle as we're side flexing. So that's the moment I have gone past the barrier. It's not the barrier. The barrier is short of that. So if we're going back to that 4%, 8%, 10%, if I wanted to treat the fascial component of this, let's say a form of myofascial release which I want to do a little bit on here in a moment…only 7 or 8 minutes, you say?

APM: I'm afraid so.

LC: I would work from that barrier…how much? If I was going to just put some load into that of 4%, I would simply hold the barrier and I would imagine 1° or 2° of extra range would give me the 4% or 8% that I want. Can we take this off for a second?

GARY: Yeah, of course.

LC: So I didn't want to leave out myofascial release aspect. When am I treating fascia? Every time I'm treating a muscle, I'm treating fascia. Are you OK? OK, so show me that movement that you…OK, that one's I think have improved already. OK, that should be able to touch the table. What I'm going to do is just going to put a soft hand here. Now, you're a little bit hairy, so I hope I don't pull the hairs.

GARY: It's all right.

LC: I'm going to use the soft side of hand here and I'm going to ask you, you can bend your arm, to do two things. I'm going to ask you to fold your arm down and take it up again until you feel it doesn't want to go further. While you're doing that, I'm just going to be putting light pressure on these tissues and slightly easing that way. Slower, slower, slower. When you feel that you're hitting a pull like when you get to there, go a little bit further, you feel this beginning to tighten there, just try and pull through that gently. You do the work. I just hold these tissues. We're working on the superficial fascia. Now back again and I keep my pressure going down on these tissues. Pull up, up, up, up, up, up. The way this is done is usually to use a loose fist but I prefer the soft side of hand, yeah, all the way down. All the way up until you feel the tightness on my hand, pull through that. Pull through that, pull past it. Keep going, keep going, keep going. Am I hurting you?

GARY: No.

LC: Are you sure?

GARY: A little bit sore.

LC: Brave boy, OK. Let's see what happens when your arm comes up here now. So you're nearly there. We've gained about so much. If we did that through these different fibers, all I would do is hold those tissues and you would work your way through them. The patient is doing the work, I'm compressing the tissues and this is working on the areolar tissue, connective tissue below the skin. We needed to do a bit up there as well. So the barrier, when I took his head into side flexion, was the moment the shoulder moved and gone past the barrier, therefore the barrier is just short of that and if I'm using the 4%, 8% idea from the research, I would simply hold it at that point with a minimal amount of force, what I judge to be 4% or 5%. OK, anything else?

APM: Yes, we've got quite a few questions. We won't be able to cover them all at all. Someone's asked it'd be rude to ask you to run over by 15 minutes. I think we can't do that but we can try and get as many questions to you as possible. You talked about psoas being very important earlier on.

LC: I didn't do that, did I?

APM: What would be your approach to psoas in —

LC: Firstly, let's turn you over on to your tummy because you may well have a psoas problem which could be important in terms of your back. Psoas, a number of ways. I check its shortness. So I'm supporting the leg. I compress the sacrum to stop extending. Go very loose now and I'm palpating with my fingertips, transverse processes, L3. So I'm locking the sacrum down. I should be able to take this leg into at least 10^{o} of extension. I can't even get 1^{o}. Very tight psoas on that side. Very tight on this side. I could work from here. I could simply say, "Gary, where my hand is touching your knee, push firmly into my hand for about 4 or 5 seconds. Go very loose, go very loose." That didn't help. Pulse down into my hand. Pulse with your knee. Pulse and pulse and faster and shorter. And go very, very loose. Yeah, very loose. So we've gained a few degrees, have we? I would work on —

APM: And by that approach, you would expect to get your full —

LC: And I would do a different eccentric stretch which would take too long to demonstrate.

APM: We've had a number of other questions. I don't think we have time to go through them. If I can ask you those questions afterwards, we'll post your answers on the website —

LC: Thank you, Gary.

APM: But Leon, first of all, thank you very much for coming in.

LC: Thank you. Thanks for asking me.

APM: Would you like to drive or shall I?

LC: Well, let's have a look at this, shall we?