Academy of PHYSICAL MEDICINE

Myogenic Headaches

with Bob Gerwin

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TRANSCRIPT

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Steven Bruce
I have a really renowned guest today, Dr. Bob Gerwin, who is the Associate Professor of Neurology at John Hopkins University in Baltimore. Dr. Gerwin, as you may recall, if you've watched any of the programs that we've done with Simeon Neil-Asher, has collaborated very closely with Simeon over his Trigger Points 3D app, something which you can see on all of those previous recordings, and something that's well worth looking at, because it is a novel, innovative and extremely useful app in terms of incorporating trigger points into your treatment. Now, he's worked very closely with Bob Gerwin on this and I think there are something like four or five hundred videos of Bob actually doing treatment on trigger points on that app. And that is one of the reasons why, of course, we've got Bob live on the show this afternoon. And we're going to be talking about myogenic headaches. So, first of all, Bob, welcome to the Academy of Physical Medicine, very kind of you to join us.

Bob Gerwin
Thank you for the invitation.

Steven Bruce
Just give me a little bit of background on your involvement with trigger points, because I think you've actually done some very new and innovative research into trigger points as well, haven't you?

Bob Gerwin
We had a recent publication on segmental sarcomere contraction, the first time that contracted sarcomeres were found in taut bands since the original publication by David Simon's based on some work he did back in 1976. So that was exciting, because it had never been reproduced before.

Steven Bruce
Does this kill off the controversy about trigger points? Because I knew there are some naysayers out there who say that they don't exist.

Bob Gerwin
I think the issue is whether trigger points or muscle pain is a phenomenon that results from nerve dysfunction, specifically radiculopathy, radiculitis, nerve compression, or whether myofascial trigger points really exist as an entity by themselves. There are a small group of people, particularly in Australia, that don't hold that trigger points are an entity by themselves, but I think most of the medical world accepts trigger points at this time.

Steven Bruce
Okay. And you're going to talk about the connection of trigger points and other elements of anatomy and physiology in connection with myogenic headaches?

Bob Gerwin

Yes.

Steven Bruce
So where do we start with that?

Bob Gerwin
All right, so I will be talking this morning, here this morning, the afternoon for you on myogenic headache, headache of muscular origin. Myogenic headache is a term that I thought I coined, but as you see in the next slide, actually Olesen and Tfelt-Hanson in 1981 use the term in a publication on muscle tenderness and common migraine. Janet Travell on the next slide, spoke of headache coming from trigger points in the sternocleidomastoid muscle, she published that in 1955 and in 1967, she talked about referred pain from trapezius and masticatory muscles causing headache related pain or headache-like pain. So, the concept is an old concept. But in fact, it has been rather controversial. And I will get to that in the course of the lecture.

Steven Bruce
I should just point out to our viewers, as I always do, but if you're new to our format, the slides will be available as handouts afterwards. So, don't worry about having to write down everything you see on the screen or anything like that. We will provide that information for you afterwards.

Bob Gerwin
Thank you. In any case, the issue was not so much as to whether or not there's muscle tenderness in people with headaches, the question has been what is the relationship of muscle tenderness and trigger points to headache, and I will talk about that a little bit later in the lecture. Right now, I'd just like to briefly review the topic of myofascial trigger points, just to remind you what they are. A myofascial trigger point is a tender zone and a taut band of muscle, it does not involve the entire muscle, it's a discrete entity within muscle. When stimulated, an active trigger point will refer pain to an area which is outside of the trigger point itself. So we call that pain at a distance and an active trigger point, as opposed to a latent trigger point, will reproduce the person's usual pain when the trigger point is stimulated. The next slide shows an electromyographic signature of a trigger point, this finding is unique to the site of the trigger point, it was identified first by David Hubbard and published in 1993. And shows two features, one of which is a constant background of low amplitude, very fast activity, looking like endplate activity, and in addition to that, there is a less frequent occurrence of high amplitude spike discharges, you can see that on the left hand of the screen. On the right hand of the screen is the interesting phenomenon that this electrical activity is inhibited by phentolamine which is an alpha-adrenergic inhibitor. So, it appears that the electrical activity that is unique to the trigger point is generated by alpha-adrenergic activity, which actually makes it look as if it is a presynaptic phenomenon. The next slide shows that the myofascial trigger point can be identified by ultrasound. As generally speaking, as the hypoechoic area, this is actually quite important because one of the longstanding complaints in acceptance of trigger points it was the fact that you could not see them, you could identify them on physical examination. Physical examination is a learned art and not everybody is capable of doing that well. But we can in fact visualise them, we can see changes in the trigger point on a diagnostic ultrasound when you treat the trigger point. And on the right-hand bottom image, you can see a

small arterial passing through the trigger point showing that there is a close association, at least on occasion, with small vessels and trigger points. And this is important because with muscle contraction, contracting the small vessels, the capillaries and small arterioles, there's retrograde flow of blood around the trigger point making the trigger point ischemic, so the trigger point is ischemic and it is also hypoxic, which is related to the mechanism which results in pain. The next slide shows the results of trigger point microdialysis work done by Jay Shah at the National Institutes of Health in Bethesda, Maryland. Jay Shah showed that at the heart of the trigger point, a point that is reached with very slow movement of the needle into the trigger point and that point is reached at the peak of the line that you see, the yellow line on the graph. And Jay Shah showed that there is an increase in a number of cytokines and neurotransmitters associated with the trigger point, cytokines and neurotransmitters such as CGRP, substance P. And there is also a decrease in pH so that the trigger point is acidic, which fits with it's being ischemic and hypoxic, so the trigger point may be at a pH of four or five. And the pink and the dark blue lines show the control areas, the latent trigger point and the non-trigger point areas, that shows that there is a considerable difference between the findings on microdialysis at the trigger point, compared to the control sites. David Simons made quite a point of the fact that in his opinion, there is segmental contraction of sarcomeres at the trigger point site. In fact, he based this on one micro photograph that he published along with Stolov in 1976. And he went back and reviewed this paper and found this micro photograph. And this was the basis for a great deal of his integrative theory on the nature of the trigger point. And in truth, this was found in a palpable band of muscle in a canine preparation. And we don't know whether it's related to a trigger point or not. But it was the only finding we had and then a castle, a huge palace, was built with this as the foundation, which I think was rightly criticised. It has never been reproduced until this past year when I published a paper in association with colleagues, Kayleigh De Meulemeester and Barbara Cagnie and their colleagues at the University of Ghent in Belgium. And we once again looked at taut bands and trigger point areas. We know that the biopsy was taken at least in a taut band, if not in a trigger point, but we could not identify more than that. And we also found examples such as this of segmental sarcomere contraction, which fits with the presynaptic alpha-adrenergic drive of the trigger point and an excess of acetylcoline at the neuromuscular junction, causing sub threshold contraction of muscle and you get localised segments of sarcomeres that contract in trigger points. We think that when this happens, you have a transient injury to the muscle, which results in the release of nociceptive substances to lead to pain and which also tend to activate the central nervous system in such a manner that you get referred pain elsewhere. So that is a very brief visiting of the concept of myofascial trigger points.

Steven Bruce
Bob, you talked a lot there about what they are, and I might have missed this, but do you have a theory on what provokes a trigger point? What makes it happen in the first place?

Bob Gerwin
We think that trigger points develop when a muscle is either acutely or chronically used beyond its ability to sustain activity. So it's an overuse phenomenon, it can occur acutely, as in an accident or sudden fall, or it can occur with repeated overuse such as in repetitive strain syndrome, for example, postural dysfunction, and it is more likely in people who are vulnerable, people have underlying conditions such as hypermobility,

for example, nutritional deficiencies, iron deficiency, hypometabolic disorders, hypothyroidism, for example. So, it's basically, when the muscle is asked to do more than it can, it can develop trigger points, we think that a disorder of the way muscle handles calcium is intrinsic to the development of trigger points. Calcium is needed for muscle contraction, of course. And we think that this persistent segmental contraction of muscle indicates that calcium reuptake into the sarcoplasmic reticulum is impaired. Calcium stays in the muscle cytoplasm when it should not, muscle stays contracted. Muscle contraction will lead to compression of capillaries, the development of hypoxia and ischemia, and then you have the whole cascade of release of nociceptive substances and development of pain and referred pain. Then to move on to headache, I'll be talking about three types of headache: migraine, tension-type headache, and to some extent cervicogenic headache. Migraine headache, is characterised by headache attacks that lasts from four to 72 hours, they tend to begin to unilaterally, they may become bilateral, they pulsate and they have moderate to severe intensity. They're aggravated by activity, so you'll see someone with an acute migraine lying down in a dark room for example. And they're often associated with autonomic phenomenon such as nausea, vomiting and photosensitivity. Tension-type headache on the other hand, tends to be infrequent episodic, but it can be chronic. And it is characterised by pericranial tenderness, which is usually present between headache. As opposed to pericranial tenderness in migraine, which is associated with a headache but not between headaches. And as in all of these definitions, headaches should not be explained by any other better cause. Cervicogenic headaches are headaches that occur in the neck they tend to be unilaterally in the neck at least in the beginning, they are accompanied by cervical muscle tenderness. In addition to that, there is usually reduced range of motion, there is decreased extension or flexion of the neck. Motor activation patterns are altered, as is the case in most situations with myofascial trigger points. And in cervicogenic headaches there has been found to be atrophy of several muscles at are the base of the skull and in the neck itself. So, we'll be talking about the role of trigger points in migraine, tension-type headaches and cervicogenic headaches. We have a number of slides showing the association of trigger points with a variety of headaches. Trigger points are extraordinarily common in migraine headaches. Calandre in 2006 published a study in which he showed that 93.9% of patients that he studied with migraine headache had trigger points, compared to only 29% in controls. In unpublished data from my own clinic, when we looked at 54 headache subjects, we found migraine symptoms in 36 of these, or two thirds of the headache patients that I looked at in this sequence of headache patients. And these 36 patients or two thirds of the headache patients had headaches associated with nausea, vomiting, photophobia and dizziness, all characteristic of the autonomic dysfunction associated with migraine. 40% of these migraineurs had headaches more than 15 days a month so they would qualify as chronic migraine headache patients. So of these, as shown on the next slide, 100% of the patients with migraine had active trigger points, compared to 30% of non-headache control patients. A significant difference indeed.

Steven Bruce
I guess the big question, and you I think you're coming to this at some point anyway, Bob, is that which is the chicken and which is the egg?

Bob Gerwin

Yes, we're gonna talk about that quite a bit. That is in fact, the major question. Are trigger points a comorbid phenomenon that's secondary to headache or are they related in some way to the initiation and presentation of headache. So, to continue with this on the next slide is work done by Cesar Fernandez-de-Las-Penas and his group in Madrid, looking at a group of migraineurs, and it showed that migraineurs showed more active trigger points than control patients. The trigger points were ipsilateral, by and large, except for the suboccipital muscles, which were bilateral. And he found as a provocative factor that forward head posture was greater in the migraine patients than in the control subjects. And he also found that movement of the neck was impaired in migraineurs. And all of these findings were to a significant degree.

Steven Bruce
Can I just ask, Bob, when you say neck mobility is decreased, is that generally or is that at the time of the headache?

Bob Gerwin
Certainly, at the time of the headache, I don't think you can make the claim generally because I don't think these patients were studied in between the migraine attacks. Although I must say that when trigger points, in my own clinical experience, when trigger points are present between headache attacks, trigger points in the cervical muscles generally result in some restriction of rotation or flexion and extension. And when you release the trigger points, you can see an increase in neck movement. So Tali in 2014, showed that there's a greater number of both active and latent trigger points, active trigger points are trigger points that reproduce someone's pain and are painful spontaneously, latent trigger points are painful only when stimulated and do not reproduce someone's spontaneous pain. But in patients with migraine, both active and latent trigger points occur more frequently than in control groups and they're seen in the trapezius and sternocleidomastoid muscles most commonly. Tali also showed that there was cervical facet joint stiffness with reduced motion at the occiput, atlas, at the C1, C2 levels in the upper cervical spine. So that the role of trigger points is while we can't say that they are causative or secondary to it, there is certainly an association of the presence of trigger points in migraine headache patients. That is also true with, as we see in the next slide, patients with tension-type headaches. And the supposition was that the trigger points which we know can sensitise, result in central sensitization, they can sensitise the central nervous system so that Do and his colleagues, conjectured that sensitising the central nervous system could lower the threshold to headache, making headache more likely to occur but in reality, the relationship to the development of headache remains questionable. Although I will have a definite comment and position on this question, based on data which I will be showing as we get further into the talk.

Steven Bruce
Bob, just to interrupt you once more if I may, Tracey's asked about cluster headaches and you haven't mentioned those.

Bob Gerwin
Cluster headaches are a form of autonomic cephalalgia, transient autonomic cephalalgia and while there are some intriguing relationships between trigger points and activation of cluster headaches, by and large, we

would not look at cluster headaches as related to myofascial trigger points except in certain unusual circumstances. To go on with tension-type headaches, we find again work done by Fernandez-de-Las-Penas, from Madrid and associates in Denmark, show that referred pain from trapezius trigger points, looks very much like the pain that people complain of who have chronic tension-type headaches. Trigger points are found in large numbers in patients with tension-type headaches far more than in controls, just as they are in migraine headaches. So, we find in one study that 75% of patients with episodic tension-type headache have trigger points in the upper trapezius, 74% in temporalis, and 60% in the sternocleidomastoid muscles. There's some very interesting findings in tension-ype headache, and that is that if you put muscles in the shoulder under chronic tension, in this case in the trapezius muscle, that pericranial tenderness is increased in patients with tension-type headache, but not in control patients. Moreover, if you go farther away from the head and go down to the leg and you have the patient perform static contraction of the anterior tibialis muscle, you find again, that pericranial tenderness is much greater in patients with tension-type headache than in control patients. Patients who do static exercise are more likely to develop headache neck and shoulder pain, if they have tension type-headache than if they have no headache whatsoever. So, we think that the association of trigger points and tension-type headaches is a very active association, a very real association. Here again is work published in 2017 by Palacios-Cefia showing that active trigger points are found far more in chronic tension-type headaches, so you find 4.3 plus or minus two active trigger points per subject in patients with chronic tension-type headache, compared to none at all in controls. While latent trigger points and trigger point tenderness, there's no difference between the two. Of course, with latent trigger points, you don't expect any pain whatsoever because if there was pain associated with a trigger point, it would be termed as an active trigger point. So that was found by Chatchawan and his group published in 2019. And the Palacios-Cefia group found the number of active trigger points always directly related to headache burden and anxiety. So, the more trigger points, the greater the headache disability and the anxiety associated with the headache. But interestingly enough, there is no relationship to depression, which we think may be related to the onset of tension-type headache altogether. So now we're going to move on to looking at the distribution of the trigger points in chronic tension-type headache. And this is a slide taken from the work from Chatchawan at all published in 2019, which showed that the most common muscles with trigger points in patients with chronic tension-type headache are the temporalis muscles, the suboccipital muscles, the sternocleidomastoid muscles and the trapezius muscles. And there's a huge overlap in this, in my experience, with migraine headache patients, as well, where you find the trigger points that refer pain to migraine areas in the head commonly come from the upper trapezius, the sternocleidomastoid muscles, the temporalis and the suboccipital muscles as well. Romero-Morales found that there are significant differences in the pain threshold, or the tenderness, between patients with tension-type headaches and with controls so that the threshold of pain is much lower. Or in other words, the muscles are more tender for the trapezius and the temporalis muscles in particular. In cervicogenic headaches, this is likewise true in the trapezius muscles. Now then the question comes up, are there medical factors that predispose to the development of trigger points and the development of myogenic headache? And there are a number of them, I've shown in this slide just four of them. On the left-hand side iron deficiency, particularly in women related to menstrual cycle and heavy menstrual blood flow. So, you'll almost never see iron deficiency in men, in the absence of carcinoma or some strange dietary situations where there's a low intake of iron. But in women, this is certainly a major concern and a factor and the other point about iron

deficiency is that iron deficiency itself is associated with headache. So, there is a double whammy factor here with relationship of iron deficiency. Forward head posture puts a strain on the trapezius and sternocleidomastoid muscles, as well as other intrinsic posterior cervical muscles, and leads the development of trigger points and then to headache of one sort or another. Because of the genetic nature of migraine headache, there's that genetic factor that may move people from a chronic tension-type headache situation to a migraine headache situation. Hypermobility is interesting because patients who have hypermobile or say loose joints wind up using their muscles to control their joints, the muscles are under chronic activity and they're more likely to develop trigger points and pain. And temporomandibular joint dysfunction. But as we mentioned in the beginning, association is not causation and the controversy, as we see in the next slide, is whether trigger points act as a causative factor that trigger headache, are they the central nervous system sensitizer the lowers headache threshold, or, as was long thought, do they occur as a consequence of the headache. In other words, headache causes central nervous system sensitization which makes trigger point development more likely. I think that the fact that the nociceptive input or the input of pain from trigger points in the neck, for example, activate the descending nucleus caudalis of the fifth or trigeminal cranial nerve, which descends in the neck down to the C5 level, that this is a mechanism whereby the trigeminal vascular cascade is associated with migraine headache can be activated, so I think that's the muscles that feed into the nucleus caudalis may have a role in initiating headache. Now in the remaining portion of the talk, I'm going to be talking about the results of treatment. And I think that plays a major role into my thinking as to the causative nature of trigger points as one of many factors that trigger migraine and tension-type headache. The other point to keep in mind is that trigger points have referred pain patterns and, as you can see in this slide, many of them taken from Trigger Point 3D, that the referred pain pattern reproduces the kind of headache complaint that patients with both migraine and tension-type headache and to a lesser degree, as shown here, but certainly also seen from referred pain patterns of neck muscles, for cervicogenic headache as well. So, I'd like to talk about the effective treatment of trigger points through a variety of mechanisms. So, in 1981, Tfelt-Hansen, Lous and Olesen published the first paper showing the effect of a treatment of what we will call the trigger points in the neck in patients with migraine headache, and they injected lidocaine, or saline as their control, and they found that 50% of their migraine patients were symptom free 70 minutes later. That's an acute effect of injection on migraine and the neck and this lasted for 24 hours. So, it's important to realise then that as you inactivate trigger points, the migraine symptoms can diminish or vanish.

Steven Bruce
Can you just clarify that Bob, because it says after the injection, patients were symptom free, that's injection of lidocaine? Not the saline?

Bob Gerwin
Lidocaine, actually saline. But I think the point is that, in fact, it's not so much the substance as the needle. Because you find the same effect with dry needling. So, the next major paper that spoke about this was in 2007, by Marie Giamberardino and her group in Ghedi, Italy, and followed by another publication 2018 by Affaitati from the same group. And they showed that lidocaine injection of cervical trigger points, trapezius cervical muscles, the trigger points that refer pain to the head and reproduce the kind of pain that patients

had with their migraine headache. And that resulted in tenderness, lowered pain threshold to electrical stimulation, which is the model that they used, that referred pain or tenderness was normalised when you inactivated the trigger points. And with repeated trigger points over six months, there was no difference between the controls and the migraineurs patients, whereas at the beginning there was a great deal of difference. So, you got rid of the tenderness in the referred pain areas. But you also decreased the intensity and frequency of migraine headache. And they showed that even if you do not inject patients, this is the 2018 paper, that if you put anaesthetic gel topically over the trigger point area, you have the same effect and in both cases you reduce the intensity and frequency of headache and reduce medication intake in the migraine patient. So, the same thing has been shown if you do dry needling of cervicogenic headache and tension-type headache and migraine headache, this study published in 2020 was a systematic review and looked at two studies of cervicogenic headache, two studies of tension-type headache and one study of migraine headaches and three of mixed tension-type and migraine headache, all using dry needling. So, no anaesthetic at all. And all showed the same kind of similar improvement in function, decrease in pain and decrease in intensity and frequency of headache. This is also shown in another paper published in 2020 by a group in Tehran, which showed a significant decrease in migraine frequency, intensity, duration, drug consumption and return of normal muscle thickness and a decrease in muscle tenderness and an improvement in cervical range of motion, both acutely and at one month following treatment, following dry needling of trigger points in patients with migraine headache. There's a group who actually do surgical removal of trigger points with a small scalpel, I don't necessarily recommend this, but the intriguing thing is that they showed a similar kind of improvement. There's much less work in terms of manual therapy of migraine and the studies are not as good. But nonetheless, improvement in headache parameters and decrease in drug consumption is shown in several papers that have been published recently. Both using a variety of different means of manual therapy, including trigger point ischemia or compression of trigger points, as well as stretching, osteopathic manipulation and so on. Lidocaine injection does the same thing. So, I have a number of studies that show this. And the real question then goes back to the controversy that we spoke about in migraine headache and in tension-type headache, are trigger points a peri phenomenon, an epiphenomenon if you will, that are present primarily because in headache patients there should be central nervous system sensitization making trigger points more likely to develop, or in fact, as trigger points result in pain because they act as one of the factors that can trigger the trigeminal facet or cascade relay and result in migraine? And the thinking over the years has changed in this and my own thinking, based on the results of the studies that show that if you inactivate the trigger points by dry needling, even manually, in an acute migraine patient, you can eliminate the headache. So, the headache in 10-15 minutes the headache is gone. And by treating the trigger points, you can reduce the intensity and frequency of headache. So, I think that the results of treatment strongly suggest to me that trigger points act as an initiating factor or as a trigger to migraine, tension-type headache and even cervicogenic headaches. So, I think it is well worthwhile treating patients with the intention, not only relieving a headache that they may have, but in a prophylactic manner as well. And I think that's the take home point from this lecture.

Steven Bruce

Bob, thank you. I'm conscious that a lot of our audience may have to leave at two o'clock in order to treat patients. So, can I put a few questions to you that have come in? Sarah has asked, how much do genetic factors do you think predispose to long term chronic migraine?

Bob Gerwin
My recollection is that 50% of patients with migraine headache have a genetic basis, they have a strong family history of migraine.

Steven Bruce
Okay. Somebody who's calling themselves PC says, do hypermobile patients with migraine respond well or not to lidocaine? Because their understanding was that lidocaine wasn't very effective on hyper mobile patients in general.

Bob Gerwin
Because you're treating the muscle trigger points, lidocaine remains effective, but one then has to address the underlying problems of hypermobility, which is another issue altogether.

Steven Bruce
And Constance has asked a question, it's a slightly different take on a slide you showed earlier on, you said that there's a connection between the menstrual cycle and headaches. Constance says is there any connection between trigger points and the menstrual cycle?

Bob Gerwin
Interesting question because there is menstrual migraine as well. So, in women, menstruation both can result in iron deficiency, which can result in headache, but menstruation is also a trigger to a migraine. And I do not have any data at all on treatment of trigger points in women who have primarily menstrual migraines. Interesting question.

Steven Bruce
Christine has asked whether the flow of CSF has a relevance in migraines, perhaps for the buildup of toxins?

Bob Gerwin
We have no data on that whatsoever.

Steven Bruce
And Vispie says, does what you're saying mean that to relieve a trigger point headache one needs to address the trigger point as opposed to the headache site? I think Vispie means tension-type headache, I'm not sure.

Bob Gerwin
I think it's in both because I think that the headache is an expression of referred pain, so that in an acute migraine, you have trigger points in the trapezius muscle that will refer pain directly to the headache site in

the head, and you treat the trigger point site in the trapezius, you treat it in the sternocleidomastoid, in the posterior cervical muscles, and you will relieve the referred pain in the head and you will relieve the migraine headache.

Steven Bruce

Bob, thank you for that. I think my audience will be very pleased because first of all, you conveyed a lot of information there, but also it must be one of the presentations where I've kept most quiet and that probably is a great relief to my audience. We've talked quite a lot about trigger points. We've talked about Trigger Points 3D and this is the app which you've developed, Bob, in conjunction with Simeon Niel-Asher. Simeon has made an offer to our audience that if you want to try out the app Trigger Points 3D, you can do that, you can get it free for a certain period of time and you get a discount if you put in the code which is on the slide. And I'll be sharing that with you after this. It looks as though we don't have it on the slide deck because it came in slightly late in the day. But I'll share that with you. So, it's well worth looking at, we've seen it on some of our previous broadcasts and not only is it go four or five hundred videos of Dr. Gerwin himself treating trigger points but also there's a wonderful interactive display of where the trigger points are, how they refer, and just how they affect various conditions in our patients.