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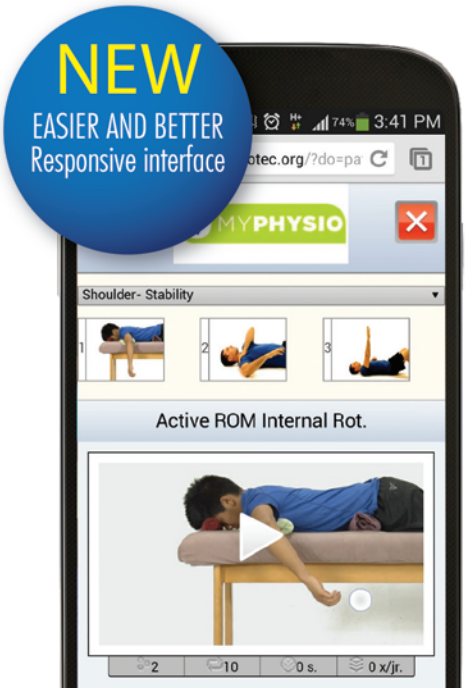

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Editorial

The why, what and how of our future…

There is a quote from Shakespeare that goes something like this: "There is a tide in the affairs of men which, taken at the flood, leads on to fortune". I'm sure that you will understand the meaning. Physio First certainly does; we know that we have a shot at a better future, a chance that if we take it, will change OUR world. This explains WHY we are.

The WHAT is going to enable us to own our marketplace in a way we have never done before, and if you have been part of the Data for Impact project, you will have earned the right to a part of this. You are the people who said: "I can do that." You are the "WHY not-ters."

When I am with a group of people, I look around and willl see the "yes-but-ters" and the "not sure-ers", but it is the "why not-ters" who will change the world. Remember, it is neither the strongest, nor the most intelligent of the species that survives; the survivors are those who are adaptable to change.

So HOW will we use this data? We will link the stats that we have collected to then allow us to respond to the questions that the marketplace needs answering. We want to unstack the data that we have collected to enable us to provide the best possible service for our patients in an informed way, using cost effective, evidence based practice. We want to provide a structure that will empower us, the members.

Physio First continues to inform and educate, and this year's Conference was another great example of us doing so. Thank you to all those generous authors for their contributions. Remember they do this for free.

So our time is coming. With our new-found forward thinking and our analysed data as evidence, I really believe we are on the edge of greatness.

Paul Johnson

Paul Johnson MSc BSc MMACP MCSP Editor

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Editorial panel

Funky treatments for tendons

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Funky is a great word, especially for someone who grew up in the '60s where it implied that you were cool. As with many words, it also has an opposite meaning, of being smelly, strange, stupid or weird. The meaning can vary in different countries and when applied to different things and situations. This article is based on the Kenneth Balfour Memorial lecture given at the Physio First 2015 Conference and investigates what funky treatments are, why they are used and what is the evidence for their use.

Learning outcomes

1. Understand how interventions should be placed in tendon rehabilitation.
2. Understand evidence that underpins interventions for tendons.
3. Identify how loading and interventions interact.
4. Identify what interventions are appropriate for individuals with tendinopathy.

Introduction

Funky treatment in sports medicine might be defined as one that is outside mainstream, innovative or cutting edge (Cook 2010). It may also take on some of the '60s meaning, of being perceived as cool because it is done by the elite, is expensive and / or is promoted by the media. From a scientific perspective, it could be perceived as one that lacks prior art, or is not accepted as a standard treatment and has little evidence to support it. Often it is where the clinical use runs ahead of the science.

Treatments these days can be heavily influenced by media exposure and online testimonials (promoted or spontaneous). Unfortunately, these (desirable) treatments often come with a big price tag despite their lack of evidence. Sometimes the price even seems to be attractive: if it is expensive it must be good. Some treatments are clearly funky. You may recall, several years ago, it was considered essential for England's Premier League football players with problematic injuries to visit a health practitioner where a horse placenta was used to elicit recovery from injury. Despite the ridiculous premise that this was based on and the expense associated with it, clubs and players were prepared to swear by its effectiveness.

Conditions for which established treatments are effective suffer less from funky approaches; there are few treatments for ACL rupture other than conservative management or reconstruction. It is often the conditions that are more difficult to manage that are susceptible to questionable treatments from those within the medical profession and outside it. Tendinopathy is one such condition where there is no one successful treatment, exposing it to the development of funky treatments.

In tendinopathy, I have taken funky to mean treatments that really seem out of place from a pathophysiological, pathoaetiological or pain perspective, where it is a struggle to see how it might be an effective treatment. Many of the more recent treatments are injection-based, some are directed at the tendon cell, either stimulating it or replacing it with active cells, and some at the matrix, especially the blood vessels. However, the underlying pathology of tendinopathy is one of cell proliferation and disrupted matrix, and there appears to be little that will easily change this. To complicate this, there is a disconnect between pain and tendon pathology, where profound tendon pathology may not be painful. Many of these therapies have a primary underlying premise of altering structure, yet the patient may only want to improve pain. It is also well-documented that there are clinical improvements in pain and function without structure changing at all (Drew *et al* 2014). So, this suggests that treatments should aim to change pain, not structure.

A further complication is that tendon pain is linked to poor function. This is especially the case if the pain is severe or if it has been present for a long time. It is not uncommon to see people with tendinopathy who have substantial muscle wasting, poor power and no endurance in the affected musculotendinous unit, or through the rest of the kinetic chain. It is clear that a uni-modal treatment directed either at pain or at structure, or that is supposed to be improving both, cannot affect function, except indirectly, after resolving the pain. As clinicians, we know that function does not restore itself naturally. It needs directed intervention and time to change.

So, if there were a treatment available that improved tendon structure, reduced pain, and regained function, would that not be

ideal for tendinopathy? Surprisingly, there is such a treatment and it is exercise. Unfortunately, it suffers from the perception that it is boring, takes too long and is not expensive; in summary, it is not funky enough. It also suffers from limited research and the fact that good quality studies have single interventions. As clinicians, we know that progressing a patient through exercise requires alterations to the programme as the person's capacity improves. Although many researched exercise interventions increase load, they rarely change exercise or add new ones, as that goes outside the study parameters. Therefore, although study results show improvement, exercise programmes can often fail when applied to real world populations (efficacy vs effectiveness). In tendinopathy, we know that athletes require many more exercise interventions than the simple, slow, eccentric ones that may be effective for the recreational runner who does only a few kilometres a couple of times a week.

It is unlikely that research will provide clinicians with answers as to the best exercise loads for pain, structure and function. It may be that the best exercise for each of these is different; with isometric for pain, and energy storage for higher level function and, perhaps, for structure. Of course, all these must also have an endurance perspective, depending on the ultimate goals of the person being treated. This again emphasises how exercise interventions have to be multi-faceted to be effective.

What are the funky treatments for tendinopathy?

Many funky treatments purport to have effect on both pain and structure. This is an interesting claim as the local source of pain in tendons is unknown. It seems that the nerve supply is only superficial in the tendon and that the deep parts of the tendon are not innervated (Danielson *et al* 2006). This makes sense as the forces deep in the tendon may be quite high and a nerve may become compressed and irritated.

It seems that any injection therapy will have an effect on either the cell or the matrix – even placebo injections can have a similar outcome (de Vos *et al* 2010). This indicates that the active substance in the injection may not be having an effect, either on the pain or the structure. There is also evidence that clinical improvement is not linked to change in structure (de Vos *et al* 2011).

Treatments to improve structure

It is worth looking at the history of this area of research and clinical practice to demonstrate just how fraught it is. As there was thought to be a connection between pain and vascularity, injections to improve vascularity were the treatment mainstay of the 2000s. Several studies and some good randomised trials showed the benefits (Alfredson & Ohberg 2005). However, further studies showed that there was little connection between vascularity and pain and that prognosis was not impeded by having vascularity. This may be because the increased vascularity is just a marker of matrix discontinuity (Ingber 2002), and that as the matrix disintegrates, the vessels increase in number as they are not impeded by an intact collagen matrix. The fact that the curve of enthusiasm for this treatment has waned suggests that the treatment is not as effective as shown in the studies when applied in the population. Effective treatments have longevity, those that come with a burst and then fade are nearly always not effective.

Presently, there are a number of treatments that are directed at "healing" or repairing the tendon. At the forefront of these are blood-derived treatments such as platelet-rich plasma that are reported to have growth factors suitable for tendon pathology, despite there being little knowledge on growth factors in either a healthy tendon or a pathological one. There are many studies reporting the effectiveness of these treatments, and certainly the early ones were case series and uncontrolled interventions. More recently there have been several randomised controlled trials that show no additional benefit of blood-derived products over placebo (de Vos *et al* 2010). As with many interventions that are shown to be ineffective, the supporters of these treatments cry foul, saying that the right treatment parameters were not used, or the right intervention product was not used. What is never reported is that the underlying premise that circulating blood, however treated, will have exactly the right growth factors for every tendon regardless of its pathology, pain and length of symptoms, is untenable.

As a direct comparison, substances that increase cellularity, such as stem cells and autologous tenocyte implantation, are now at the forefront of funky treatments. The premise on which their use is built is that tendinopathy is an hypocellular pathology and, as a result, the tendon loses its capacity for repair. Increasing the cell population will enable the tendon to manufacture the matrix proteins required, the structure will improve and pain reduction will logically follow. Again, there is very little evidence to support these suppositions. Tendon metabolism is 25 times higher in a pathological tendon than a normal one, mainly due to an increase in resident cells that manufacture and degrade extracellular matrix proteins at a high rate (Parkinson *et al* 2010). This results in protein debris in the tendon, especially proteoglycans, which interferes with extracellular matrix structure and restructuring after injury. There is some evidence that stem cells may take a more directorial role, working to control the resident cell population rather than becoming integrated into the matrix; however, that does not mean that healthy matrix will result. There are some treatments that are used in other conditions that are rebranded as treatments for tendons.

Hyaluronan-based products that are used in osteoarthrosis are now marketed as treatment for tendon pathology (Abate *et al* 2014). However, although these products may be relevant for peritendinous conditions, especially in the wrist and hand, and perhaps after tendon surgery, there is little evidence to support their use in overuse tendinopathy.

Treatments to relieve pain

The cause of pain in tendinopathy is not known (Rio *et al* 2013), therefore it is hard to direct treatments at the tendon tissue source. Many treatments have an effect on the nerves as they enter the tendon, inducing either a total or partial disruption of efferent signals to the central nervous system. Direct severance of the nerves will likely occur when the fat pad is separated from

the tendon (Ruergård & Alfredson 2014), and in treatments that use methods to ablate the area of pathology (Abat *et al* 2014). Temporary nerve disruption may be the reason for the effectiveness of extra-corporeal shock wave (van Leeuwen *et al* 2009) and vascular sclerosing agents as these are neurotoxic (Wilde *et al* 2011). Corticosteroid is also effective for pain relief, although it is likely that its effect is on the tendon cells, as it damps down their activation and proliferation. However, it is possible that it directly affects the nerves as well.

Evaluating funkiness

There are several questions we should ask when evaluating a treatment and the answers should guide the clinician on the possible benefits and associated risks.

- Is the premise underlying the treatment defensible? The known pathoaetiology must fit the likely mechanism of action.
- How much comprehensive, clinical data is there? This is the most important question the clinician should ask, together with: What is the quality of the data and its place in the clinical reasoning process?
- What are the health and economic risks to the patient? These are critical. In today's connected world, the media can promote treatments directly to patients that are not based on science.

It is important to realise that there is a time, and a tendon, for every intervention and that good clinical reasoning will guide you. Uni-modal treatments for a complex condition such as tendinopathy will nearly always fail. Good clinicians with well-honed clinical reasoning skills are the answer for treating tendons.

About the author

Jill Cook is a Professor in Musculoskeletal Health in the School of Primary Health Care and NHMRC practitioner fellow, Monash University in Australia. Jill's research areas include sports medicine and tendon injury. After completing her PhD in 2000, she has investigated tendon pathology, treatment options and risk factors for tendon injury. Jill currently supplements her research by conducting a specialist tendon practice and by lecturing and presenting workshops in Australia and overseas.

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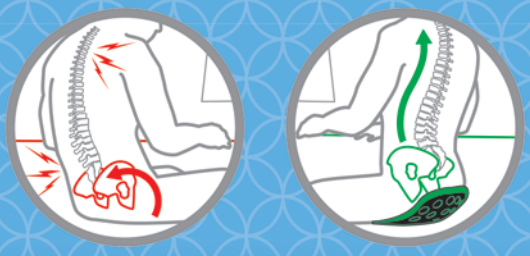

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Electro physical agents and tissue repair: can we really make a difference?

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This article is based on the Olive Sands Memorial lecture presented at the Physio First 2015 Conference and aims to provide an overview of the complexities of the tissue repair events, together with the use of electro physical agents (EPAs) in practice, considering the evidence for their effectiveness.

Learning outcomes

1. Appreciate the key elements of the normal and the disturbed tissue repair sequence.
2. Identify the EPAs which are most strongly evidenced as being able to influence the tissue repair process.
3. Appreciate the context in which each of these EPA modalities are most effective.
4. Identify the potential (evidenced) value of modalities which the individual may not currently employ.
5. Be equipped to make clinical decisions with regards the use of EPAs in their practice, together with how they might be most effectively integrated with other interventions, such as manual therapy, exercise etc.

Introduction

While electro physical agents (EPAs) have been a component of physiotherapy practice since the early days of the profession (the first edition of *Clayton's Electrotherapy and Actinotherapy* was published in the UK in 1948 and an interesting historical review can be found in Kahn 1994), the modalities, the rationale for their use and their delivery methods have changed considerably since the early days, and continue to do so.

The most popular modalities used in current practice are, in many respects, quite dissimilar to those of 60 or more years ago though they are, of course, based on the same physical principles. Even in the last 10 years there has been a noticeable change in emphasis and delivery methods. The change in terminology from "electrotherapy" to the broader, and more accurate EPAs is not just a trend, but reflects a reality that the range and scope of interventions employed extends way beyond "medical electricity". Ultrasound, shockwave and body vibration, for example, are accurately included under the EPA banner but clearly are not types of electrotherapy in the strict definition of the term.

Modern practice needs to be evidence based in order to retain its currency and justify its inclusion in clinical practice. It is sometimes said by critics of EPAs that there is a lack of evidence to support their use. There is, in fact, a wealth of published evidence and it is possibly more an issue of awareness of this evidence rather than there being no evidence. There certainly appears to be an evidence / practice mismatch; those modalities most widely employed do not necessarily match those with the strongest evidence, and vice versa (Watson 2014a). Some of the research can appear daunting and a substantial proportion is not published in the therapy journals per se, which can make access more problematic, but the evidence is there. Searching for and evaluating the evidence is an integral part of an individual's evidence based practice, and reviews can support the busy clinician by providing a concise summary of the evidence pertaining to a particular field or therapy area.

When EPAs are used in line with the evidence, they can be phenomenally effective. Used unwisely or inappropriately, they will either do no good at all or possibly make matters worse. This is not a principle that is only applicable to EPAs. Drug therapy used inappropriately would not be expected to be effective, neither would manual therapy, exercise therapy, acupuncture or any other intervention when used at the wrong time, or for the wrong reason, or at the wrong dose. In this, the application of EPAs is no different to any other intervention.

In the physical therapy domain, EPAs are sometimes perceived as being different in that;
a) a machine is employed as part of the delivery system, and
b) the practitioner must make an overt decision with regard to dosage.

Dosage decisions are, in fact, an everyday component of all therapies, but perhaps it is less obvious when determining the number of exercises that are needed to achieve a particular strengthening effect, or how long a static stretch needs to be held in order to achieve an increased range of motion. The skill of the practitioner using EPAs is to make the appropriate clinical decision on which modality to employ, when, and at what dose, using the best available evidence (Watson 2010).

One of the commonly levelled criticisms of the evidence based practice philosophy is that treatment packages and care plans become generic, but even on a limited consideration, this view can be rapidly dismissed (e.g. Olive & Solomonides 2009). While a universal or generic treatment plan or care pathway for every patient with whiplash, OA knee or a median nerve lesion can be completed, they are unlikely to actually match the demands and needs of any one individual patient and thus, without flexibility and modification, they tend to fail. Genuinely evidence based practice enables the best evidence to be considered relative to a specific patient. There will be commonality between the presentations of any three patients with, say, OA knee, but there will be differences too. It is the differences in presentation and context that make one treatment package more effective than another, and differentiates the more skilled from the less skilled practitioner.

This article is not a "cookbook" approach, and quite deliberately so, as the aim following the philosophy outlined above is to consider **which** of the EPAs have an **evidenced role** in the management of tissue damage and tissue repair, outlining their strengths and areas for optimal use. It does not, and makes no attempt to, provide a recipe for the use of ultrasound for a grade 2 tear of the medial collateral ligament of the knee, nor a prescription for the use of shockwave as a treatment option for chronic Achilles tendinopathy. This information is available from multiple sources, including my own web pages (www.electrotherapy.org). Instead, the overall aim is to consider whether EPAs can have a positive effect on tissue repair and, if so, which modalities are most strongly supported by the evidence, and in which **clinical circumstances** are they most usefully employed.

Key issues for tissue repair

Although the general model of tissue repair currently employed is not radically different from those presented 15 or 20 years ago, the understanding of the complexity of the repair process has moved on phenomenally in that time and the number of papers published annually, which inform our knowledge in this field, has increased exponentially. More is now known about the complexity of the messenger systems which control the repair sequence, and about the interactions, interdependency and co-dependence of the various elements, together with a deeper appreciation of both what is normal and how normality can drift or be pushed, or dragged into abnormality, resulting in delayed healing, stalled repair and adverse clinical outcome. A commonly employed general repair model is shown in Figure 1 and discussed in numerous publications including Watson (2006a).

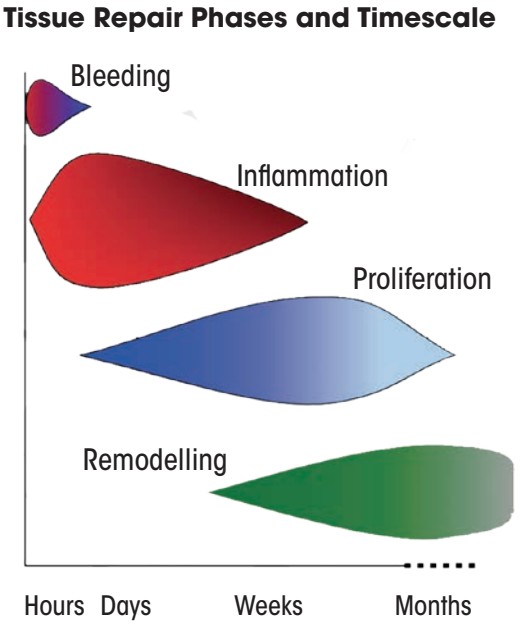

Figure 1: ***General model of the tissue repair sequence***

The essential division of the repair process into key, overlapping and interlinked phases; inflammation, proliferation and remodelling being those classically listed, is the commonly adopted approach, though it is important to state that this division into discrete phases is a matter of convenience rather than a reflection of reality. In the real world, all phases are fully integrated and, from the body's perspective, it is more like one continuous process (Figure 2) than a series of identifiable events.

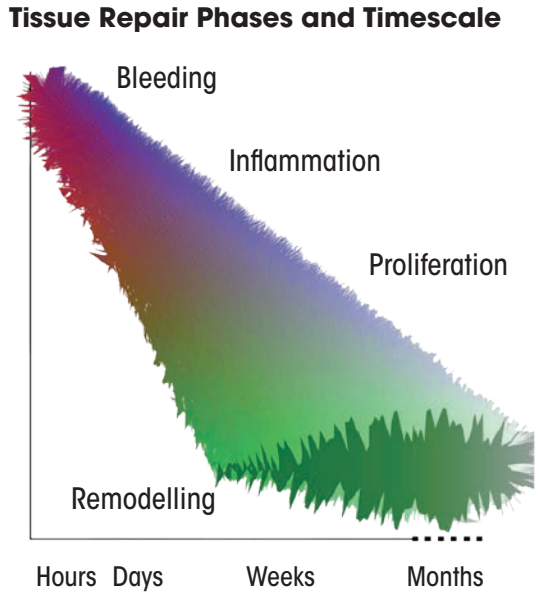

Figure 2: ***Realistic 'integrated' tissue repair sequence without discrete phases***

Within each of these key phases, a deliberately and carefully controlled series of events take place which culminate in a tissue which is repaired and has both structural and functional capacity as close as possible to the original tissue. Until stem cell research and development reaches everyday musculoskeletal therapy, we will not be replacing the damaged tissue with new ligament, or tendon. Most tissues are repaired

rather than replaced, or replicated. The repair is essentially made from scar tissue, which is the best that the body can come up with. The quality and behaviour of this scar tissue can be overtly influenced by the environment in which the repair takes place, the behaviour of the surrounding tissue, i.e. things that the patient does to themself, and is "influenceable" by therapy intervention including exercise, manual therapy and EPAs.

There is no evidenced argument that changing the process of repair is an appropriate or sensible option. The process of repair is well organised, comprehensive, and overall a very effective sequence. In the therapy environment, we tend to see a disproportionate number of patients in whom the process has gone wrong one way or another, skewing our perception of how effective the process actually is. The primary, logical and evidenced role of therapy is to support, facilitate, stimulate or enable (whichever is the preferred term) the process rather than trying to change it. The **inflammatory** events are essential to a successful repair and managing them is sensible and evidenced; turning them off is not. **Proliferation**, during which the essential scar tissue is laid down, is critical and again, therapy can positively influence these events, encouraging their progression into the final stage of **remodelling**, during which the basic or generic repair scar tissue is refined, making it capable, as far as possible, of replicating the functional capacity of the tissue that is being repaired.

Our current understanding of the essential nature of chemical messenger systems, as the drivers of the repair sequence, means that it is now possible to identify HOW the repair sequence is influenced by therapy. Therapeutic interventions, including manual, exercise and EPA based therapies, are capable of influencing the chemistry of repair, thereby exerting their influence (Wang & Li 2010). It is not argued here that therapy ONLY works through a biochemical pathway, but that there is a growing body of evidence to support the argument that this is ONE of the mechanisms of effect. For example, the capacity of low intensity pulsed ultrasound (LIPUS) to positively influence fracture repair, is essentially one of a chemical pathway; the mechanical energy stimulates the expression of mediators and it is this enhanced expression that results in a stimulating effect on the bony repair (Padilla *et al* 2014). This does not change how the modalities are actually employed in practice, but does affect our understanding of the mechanism / how the effect(s) are achieved.

There is an additional strand to this concept. Endogenous bioelectric activity, i.e. electrical activity which is internal to the body, as opposed to exogenous that is electrical energy provided from a source such as electrotherapy, is understood also to have an influence on repair. While chemical changes alone do not explain all elements of the process of repair, nor the total influence of therapy, the internal electrics of the body do have a positive and supporting influence (Watson 2008a). Therapy can positively influence these events and thereby provide an additional pathway through which intervention makes a difference to tissue repair.

Microcurrent based therapies specifically target these pathways (Poltawski & Watson 2009), employing very small (less than a thousandth of an Amp) currents as a means to promote and support the small internal currents which are an essential element of a normal repair sequence. We now appreciate that exercise and manual therapy also exert influences on these endogenous currents, providing a potential mechanism by which different therapies can have a common target and a mutual mechanism of action. It is not suggested that neither manual therapy, nor exercise therapy only exerts a therapeutic influence by means of endogenous bioelectrics, but that this is a component of their mode of action.

Repair is enhanced through the gross physiological, biochemical, bioelectric and neural mechanisms exerted by EPAs (Watson, 2011). In this regard, the effects of EPAs are not exclusive and their use alongside manual and exercise therapy will, in almost all circumstances, provide an optimal environment in which the damaged tissue can progress through the essential sequence, resulting in an optimal outcome: a quality repair with maximal functional capacity, something that would be a reasonable aim or intent from any therapy treatment package. Selecting the optimal modality to achieve this result is a key element of the clinical decision making process, just as it is essential to identify the optimal manual therapy and "optimal exercise" programme to achieve the same end. There are times when EPA use is

Electrical Stimulation Agents / Modalities	Thermal Agents / Modalities	Non Thermal Agents / Modalities
Transcutaneous Electrical Nerve Stimulation (TENS)	Infra Red Irradiation (IRR)	[Pulsed] Ultrasound
Interferential Therapy (IFT)	Shortwave Diathermy (SWD)	Low Intensity Pulsed Ultrasound (LIPUS)
Neuromuscular Electrical Stimulation (NMES)	Microwave Diathermy (MWD)	[Pulsed] Shortwave Therapy (PSWT)
Functional Electrical Stimulation (FES)	Other RF Therapies	[Pulsed] Laser Therapy (LLLT / LILT)
Faradic Stimulation	Hydrocollator Packs	[Pulsed] Microwave Therapy
Iontophoresis	Wax Therapy	Low Intensity RF Applications
High Voltage Pulsed Galvanic Stimulation (HVPGS)	Balneotherapy (inc spa/whirlpool)	Pulsed Electromagnetic Fields (PEMF's)
Low Intensity Direct Current (LIDC) and Pulsed LIDC	Fluidotherapy	Microcurrent Therapies
Twin Peak Monophasic Stimulation	Therapeutic Ultrasound	MAGNETIC THERAPIES
Diadynamic Therapy	Laser Therapy	Pulsed Magnetic Therapy
H Wave Therapy ; Action Potential System (APS)		Static Magnetic Therapy
Russian Stimulation : Medium Frequency Stimulation	Cryotherapy / Cold Therapy / Ice / Immersion Therapy	Microcurrent Therapy (MCT)
Rebox Therapy; Scenar Therapy, NRN (InterX) based therapy		(Radial) Shockwave Therapy
Microcurrent Therapy (MCT)		

***Figure 3:** Listing of the more commonly encountered EPAs under electric, thermal and non-thermal groups*

not indicated, just as there are times when manual therapy, acupuncture, stretching, etc. is not required. This does not negate the value of EPAs in therapy, it just means that they are not essential in all circumstances, that no one therapy is essential in all circumstances. Knowing when EPAs have an evidenced role to play, and when best left to one side, is key to therapy success.

Influencing repair

There are a wide range of EPAs, a selection of which are incorporated in Figure 3. This is not claimed to be a complete list of all modalities available, it is a representation of the range and attempts to categorise them under three broad headings.

It isn't possible within an article of this length to run through all modalities. Those which are most able to influence tissue repair directly are in the non-thermal group; of these, the most commonly employed in current practice are ultrasound, laser and pulsed shortwave. There is no doubt from the evidence base that LIPUS (an ultrasound variant), other radiofrequency applications such as frequencies other than shortwave, various magnetic field based interventions and micro-current therapy, are all capable of having a positive influence on repair. Whether they are currently used in therapy practice does not negate the fact that there is evidence to support their employment in practice. This is the evidence-practice mismatch.

While **ultrasound, laser** and **pulsed shortwave** therapies all have a stimulatory effect on the repair sequence, the existing evidence would suggest that they are effective because they stimulate the normality of the repair sequence, not by changing it. The energy delivered with a clinical ultrasound unit is technically very different from the radio energy, but their effect on the inflammatory, proliferative and remodelling stages is remarkably similar. The primary difference between these interventions is WHERE that effect is achieved rather than WHAT the effect is (Watson 2006b).

Ultrasound

Energy is preferentially absorbed in tissue of high protein content and, as collagen is the most prolific structural protein, tissue such as ligament, tendon, joint capsule, fascia and scar will be high on the response list. This is central to the clinical decision-making argument. In order to have an effect, energy needs to be absorbed. Tissues which preferentially absorb energy XxX will be those in which the strongest response is seen. Other tissues will of course respond, but to a lesser extent (Watson 2008b, 2014b).

Laser

Delivered to the tissues at appropriate levels or doses, laser energy will be primarily absorbed in the superficial tissues, especially those with strong vascularity such as skin, muscle, tendon sheath and joint synovium. It is effective at stimulating repair in these tissues and it is no great surprise, therefore, that its use for open wounds is strongly supported. It is also effective for inflammatory presentations in synovial joints and lesions in musculoskeletal tissue as long as they are sufficiently superficial for the delivered light energy to achieve sufficient penetration, probably to a maximum of 15mm or so. Laser can be, and commonly is, employed in the treatment of a wide range of clinical presentations, but it is at its most efficacious when directed at the tissues which absorb light energy at the wavelengths employed (e.g. Alves *et al* 2014; Newman & Homan 2014).

Pulsed shortwave

Also facilitating repair and recovery from injury, the energy delivered by pulsed shortwave, at 27.12MHz radio frequency, will be preferentially absorbed in the tissues of low impedance and high ionic content. These are essentially the wet tissues such as muscle nerve, bursae and areas where oedema, effusion or haematoma are present (Al Mandeel & Watson 2008).

As these three common modalities are evidenced as being effective at stimulating repair in the tissues which absorb the energy being delivered, the choice between them, based on the best available evidence, pertains primarily to the nature of the target tissue, over and above any other clinical decision. If a patient presents with a ligamentous lesion, whether in the hand, knee, ankle or the acromio-clavicular joint, ultrasound is more likely to be effective than any of the other options, based on the nature of the target tissue; whereas, if the patient presents with a joint effusion or soft tissue swelling around an injured structure, pulsed shortwave is more likely to achieve maximal benefit.

Once the particular modality of maximal potential benefit has been identified, the selection of the most strongly evidenced dose is essential, but is certainly beyond the remit of this article. If both the optimal modality and the optimal dose are employed, the EPAs can have a highly significant effect on the repair sequence. If the optimal modality is delivered at a sub-optimal dose, it would be illogical to expect maximal effect. Worse still, if a sub-optimal modality delivered at a sub-optimal dose is employed, there should be no surprise when no significant effect at all is the result. Clinical decision making with the EPAs focuses around this core concept (Watson 2010).

Other modalities that might be less familiar to therapists but which, based on the evidence, have tissue repair capabilities include:

Low Intensity Pulsed Ultrasound (LIPUS)

This relatively recent development in the use of ultrasound involves the application of the same modality, but at considerably lower power as a means to explicitly influence bone repair. It is an evidenced intervention, effective in the management of fresh fractures, stress fractures, delayed and non-unions. Ultrasound has long been considered to be contraindicated near fracture sites as, at higher doses, it can be a painful intervention which makes it a useful technique to find stress fractures. At very low doses however, it has been shown to have a positive effect on the rate of fracture healing; sufficient evidence, in fact, for NICE to support its use (NICE 2010, 2013).

The applied daily dose of 1.5MHz; 0.03W cm^{-2}; pulsed 20% at 1000Hz; 20 minutes is not possible to achieve with current clinical machines, even when turned down as low as possible. This level of power is some three times lower than most machines deliver

on their lowest setting. Therefore, currently, specialised machines need to be employed, most commonly on a loan / rental home-use basis.

While employing LIPUS for bone repair is already established, research into its use across a range of other musculoskeletal presentations is currently being carried out.

The modality and the evidence are summarised at www.electrotherapy.org web pages, and the mechanisms by which the effects are achieved are reviewed in Padilla *et al* (2014) and a useful clinical review can be found in Bashardoust *et al* (2012), Griffin *et al* (2008), Watanabe *et al* (2010) and in the NICE guidelines and review (NICE 2010, 2013).

Shockwave

As the name would suggest, shockwave based therapies are not subtle but are evidenced as being effective, most strongly in the treatment of chronic tendinopathies. While there is an increasing range of clinical presentations for which this treatment is being investigated, it is in the field of chronic tendinopathy where the existing evidence is most compelling.

There are multiple methods to deliver this energy, but in therapy the RADIAL shockwave applications predominate. These are non-destructive in nature, but serve to stimulate a stalled or slowed tissue repair sequence by providing a strong pro-inflammatory stimulus. Shockwave treatment is most effective when combined with a complementary tendinopathy management programme, including eccentric loading. There is a summary of the current evidence at www.electrotherapy.org and reviews in the literature include Romeo *et al* (2014) and Speed (2014).

Microcurrent therapy

This is almost the antithesis of shockwave in that it is a very low powered and truly subtle approach to the facilitation of tissue repair. It involves the delivery of a particularly small (magnitude) electric current; by definition, less than 1mA. This will be sub-sensory in nature and its mechanism of action, unlike other forms of electrical therapies, is not to stimulate action potentials in peripheral nerves, but instead to support the endogenous currents which are endemic to tissue repair in the musculoskeletal environment.

Microcurrent therapy has a long and established use in bone injury and the management of open wounds where it has been employed, albeit with a variety of different names, for decades. More recently, its use in soft tissue and other musculoskeletal presentations has been researched, with a growing body of supportive evidence.

It appears to be at its most effective when employed for long treatment times, i.e. hours rather than minutes, and due to its sub-sensory nature, application by the use of small battery powered treatment units and ease of application, home based treatment – potentially on an overnight basis – is gaining ground.

The evidence of its clinical efficacy and scope was reviewed in Poltawski & Watson (2009) and again a review of the evidence and mechanism of action is provided at www.electrotherapy.org.

Conclusion

The topic of EPAs is one that would easily fill an entire book. Therefore, this paper provides a very limited review of their current position as a means to positively influence tissue repair and healing. The process of tissue repair, in both normal and disturbed modes, is considerably better understood with the emergence of research evidence. The capacity of therapeutic intervention(s) to influence these processes is not in doubt, and EPAs are just one of the methods by which tissue healing can be affected. While the evidence supports the use of various EPAs as a component of a treatment and management package, it does not suggest that the EPAs are better than other forms of intervention, rather that their integration or amalgamation into a comprehensive treatment programme will make a difference to the outcome.

It is important that the most appropriate, evidenced modality is employed in each clinical circumstance. It is also important that the dose, or machine parameters, is carefully selected in order to achieve optimum effect. Used in such a fashion, the EPAs have evidenced support. Used unwisely, inappropriately or indeed, incorrectly, they provide no useful effect whatsoever, but exactly the same would rightly be said if manual therapy or exercise therapy were used in the same way.

About the author

Tim qualified as a physiotherapist in London in 1979. After working in the NHS, various sports clubs and national teams, he became a lecturer at West Middlesex Hospital. He then attained a degree in Biomedical Sciences followed by a PhD in Bioelectronics from the University of Surrey in 1994. He researches in the areas of electro physical agents / electrotherapy and tissue repair and is currently Professor of Physiotherapy at the University of Hertfordshire. He has published over 50 journal papers, is editor of a core text on electrotherapy and has contributed numerous chapters in other edited texts. He is on the editorial board of two physical therapy journals and reviews for more than 25 journals and grant authorities. He's presented more than 870 lectures, short courses and conference papers. He is responsible for the www.electrotherapy.org website, was awarded a Fellowship of the Chartered Society of Physiotherapy in the UK in 2013 and is on the Executive for the Electro Physical Agents and Diagnostic Ultrasound group in the UK and on the ISEAPT sub group of the WCPT.

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Multidirectional instability of the shoulder: what is it and is physiotherapy effective?

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This article is based on the PPEF lecture, originally delivered at the Physio First 2015 Conference, and reviews multidirectional instability (MDI) of the shoulder and the physiotherapy interventions that might be used in the rehabilitation of this condition.

Learning outcomes

1. Understand the different forms of glenohumeral joint instability.
2. Understand the aetiological factors that contribute to development of multidirectional instability (MDI).
3. Discuss the current levels of research evidence for effectiveness of conservative management of MDI.
4. Recognise the research evidence from current clinical outcome trials that demonstrates effectiveness of rehabilitation for MDI.
5. Apply knowledge of how to treat patients with MDI effectively.

Introduction

Glenohumeral joint instability is defined as a pathological increase in translational motion that interferes with joint function and / or produces pain (Lewis *et al* 2004; Jobe & Iannotti 1995). This differs from laxity, which is an asymptomatic increase in translational movement (Lewis *et al* 2004; Shea 2013; VandenBerghe *et al* 2005; Guerrero *et al* 2009). Rowe (1956) first delineated two distinct categories of instability: traumatic and atraumatic (Rowe 1956). Traumatic instability is associated with a traumatic onset, such as a fall or contact sports and usually results in higher grades of instability, such as a dislocation or subluxation (Liavaag *et al* 2011). There is a high association with structural lesions such as ligament avulsions, labral tears and bony lesions in traumatic instability (Habermeyer *et al* 1999). The general consensus in the literature is that traumatic instability is associated with a high risk of recurrence, especially in younger patient populations (Hovelius *et al* 1996; Simonet & Cofield 1984; Robinson *et al* 2011), hence surgical treatment is seen to be more likely to be required in this group and often is considered to be the most predictable solution (Chahal *et al* 2012; Godin & Sekiya 2010; Handoll *et al* 2004).

Atraumatic instability presents as a result of repetitive, overuse, or non-traumatic movements such as those involved in overhead sports (Garth *et al* 1987; Salomonsson *et al* 1998; VandenBerghe *et al* 2005; Guerrero *et al* 2009). However, the line between what is traumatic and what is atraumatic is a difficult one to define and perhaps the term micro-traumatic instability is a better one. Certainly, in reality, overuse injuries are clearly not truly atraumatic but involve repetitive excessive stresses, strains and micro-trauma being applied to glenohumeral joint structures. These eventually lead to damage or overload of the capsuloligamentous structures and produce a lower incidence of specific structural lesions (Miniaci *et al* 2002; VandenBerghe *et al* 2005). Rehabilitation is the treatment of choice for atraumatic instability, especially in the initial phases of management, and it is considered to be most effective (Yamaguchi & Flatow 1995; Kiss *et al* 2001).

Multidirectional instability (MDI) was first described in 1980 by Neer and Foster, as an extreme form of atraumatic instability. Multidirectional instability (MDI) may be defined as symptomatic glenohumeral subluxation, or dislocation in more than one direction, as a result of repetitive micro trauma, lack of muscle co-ordination and congenital differences in the joint (An & Friedman 2000; Bahu *et al* 2008; Mallon & Speer 1995).

The quintessential finding of this clinical condition is the presence of symptomatic inferior instability, sulcus sign, and anterior and posterior dislocations or subluxations of the shoulder (Ide *et al* 2003; McFarland *et al* 2003; Fischer 2004). The literature presents varying opinions on whether two or three directions of instability are required to be classified as MDI, whether the onset is always atraumatic (Shea 2013; Liavaag *et al* 2011; Chahal *et al* 2012; McFarland *et al* 2003; Fischer 2004; Gerber & Nyffeler 2002; Joseph *et al* 2003; Throckmorton *et al* 2009), or whether MDI exists at all (Kuhn 2010). The clinical presentation is often missed due to the fact that clinicians frequently focus on the patient's symptoms of secondary rotator cuff overload, and miss their underlying cause.

Definition of MDI

For the purposes of the clinical trial presented in this article, MDI was defined as atraumatic instability with symptomatic subluxation / dislocation in all three directions. It should also be noted that MDI is different to joint hypermobility syndrome (JHS): a common condition with a mixed phenotype of heritable disorders of connective tissue (HDCT) that shares overlapping features with other heritable connective tissue disorders such as Ehlers-Danlos Syndrome (Tinkle *et al* 2009; Hakim & Grahame 2003).

While these patients may also present with MDI type symptoms of the shoulder, they have generalised whole body system hypermobility issues and should be considered as a separate sub-group, often requiring a slower and more global type of rehabilitation approach (Keer & Simmonds 2011). It is important when reviewing articles presented in the literature regarding management of MDI, that the clinician clearly identifies how the study has defined MDI so that they are certain exactly which patient population is being treated.

The aetiology of MDI is multifactorial and, for it to occur, several factors must be present. First, there is an underlying joint anatomical variation or anomaly, such as a ligament, labrum or glenoid dysplasia, or a large, redundant capsule that results in hypermobility (Salomonsson *et al* 1998). Superimposed on this is, usually, an episode (or episodes) of overuse, fatigue or quick unguarded motion that results in altered muscle function. Deficiencies of the rotator cuff or deltoid have been implicated in the literature, but there is little concrete evidence of either in the research currently published. Altered humeral head centring is commonly associated with MDI (von Eisenhart-Rothe *et al* 2002; Inui *et al* 2002). One of the primary reasons for this is thought to be insufficient upward rotation of the scapula, allowing excessive inferior translation of the humeral head (Ozaki 1989; Itoi *et al* 1992). The final component that is frequently present is an element of abnormal motor patterning where muscles are recruited using an aberrant co-ordination pattern during normal movement (Nyiri *et al* 2010).

The general consensus is that rehabilitation is the first treatment of choice for MDI and, due to the reported variable outcomes, surgery should be avoided if possible (Kiss *et al* 2001).

Conservative treatment is largely reported to achieve 80-90% success (Matsen *et al* 1991; Tibone *et al* 1993; Warner & Caborn 1992), but there is a paucity of evidence in the literature to justify this figure which is largely based on anecdotal evidence. A systematic review conducted by these authors (Warby *et al* 2014) revealed only one study (Ide *et al* 2003) that utilised pre- / post-prospective intervention, incorporating functional outcome or clinical measurement tools, designed to assess the effectiveness of physiotherapy. The fact that evidence for efficacy of conservative rehabilitation programmes is lacking, and there is a paucity of clear published clinical rehabilitation parameters for optimal symptom resolution, indicates that there is a need to conduct a clinical outcome trial to determine if physiotherapy is effective in the management of MDI.

Current trial

The aim was to investigate the functional, clinical and EMG changes in a group of MDI patients following conservative rehabilitation. One orthopaedic surgeon assessed 46 participants in order to confirm the diagnosis of MDI. All underwent an MRI to exclude any structural lesion and all were given a barrage of baseline measures performed by one independent tester. Strength was measured by a PowerTrak II hand-held digital dynamometer from JTech and scapula position was measured at rest and during abduction with an inclinometer. Both devices have been demonstrated to be reliable and valid in shoulder patient populations (Riemann *et al* 2010; Watson *et al* 2005). All participants also completed three functional instability specific questionnaires: Western Ontario stability index (WOSI), Melbourne instability shoulder scale (MISS) and Oxford instability shoulder scale (OISS), all of which have been shown to be reliable, valid and sensitive to change in instability patient populations. Eleven of the patients also underwent evaluation with fine wire EMG prior to rehabilitation and this was compared to a previously collected database of the normal population (n=24).

All participants underwent a 12-week rehabilitation programme with one therapist. This was made up of four primary phases:

- Phase 1: The primary emphasis here was on scapula correction and stabilisation. Each patient was examined at rest, during motion and during loaded muscle testing. Manual correction of the scapula was performed to determine the best position for elimination, or improvement of the patient's instability symptoms and to best facilitate improved movement patterns. The patient was then taught to correct their scapula into this position with scapula setting drills; the most common of which was upward rotation with a little elevation. The majority of patients were educated on how to perform a modified shrug drill in 20° - 30° abduction. This has been demonstrated to facilitate significant increases in activation of upper, middle and lower trapezius in normative and MDI patient populations (Pizzari *et al* 2014). Endurance and recruitment repetitions were the primary emphasis of this phase, with patients only exercising short of fatigue or pain reproduction and performing drills with graduated light weights and Theraband™ resistance (Figure 1).

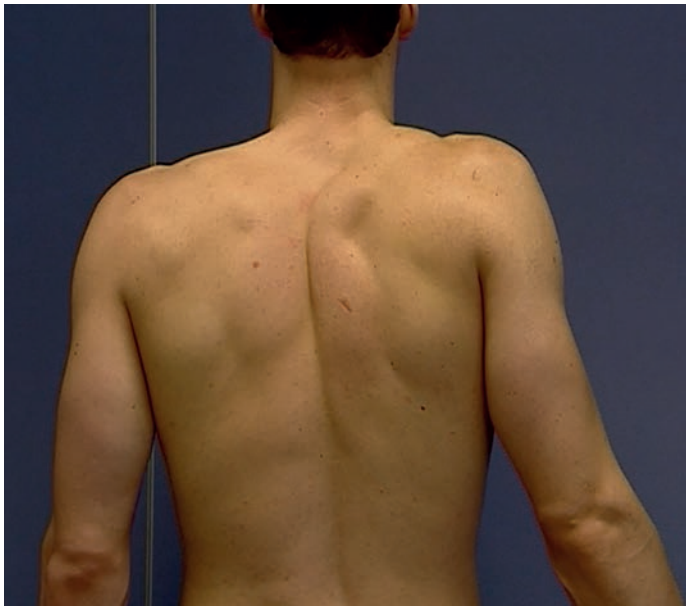

Figure 1: ***Modified shrug with arm in 30° abduction to achieve effective upward rotation of the scapula***

- Phase 2: Here, the emphasis was on humeral head control and centring. Some patients benefitted from facilitation of humeral head control by co-activation drills with Theraband™ resistance around the proximal humeral head. This has been found to produce a significant increase in subscapularis activation (Pizzari *et al* 2013; Magarey & Jones 2003). Once again, recruitment and endurance dosages were utilised in exercise selection, such as two or three sets of 20 repetitions in any one session, performed two to three times a day (Figure 2).

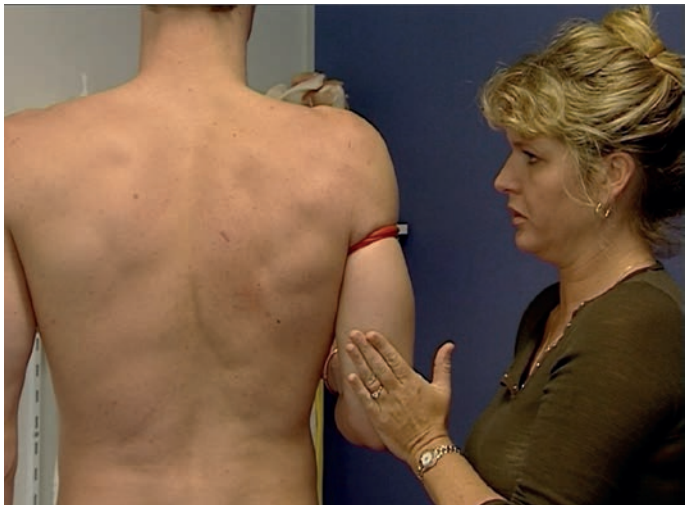

Figure 2: ***Humeral head centring / control with a Theraband™ positioned around the proximal humeral head***

- Phase 3: Once scapula and humeral head were controlled, motion was introduced. The patient performed a range of motion drills against a light Theraband™, making sure that both scapula and humeral head position were controlled. Movement control was re-trained in lower ranges of abduction / external rotation with the patient performing only the arc of motion they could control. Once control was achieved within a range of motion, resistance was added either through incremental increased grades of Theraband™ resistance or incremental weights, usually ½kg increments (Figure 3).

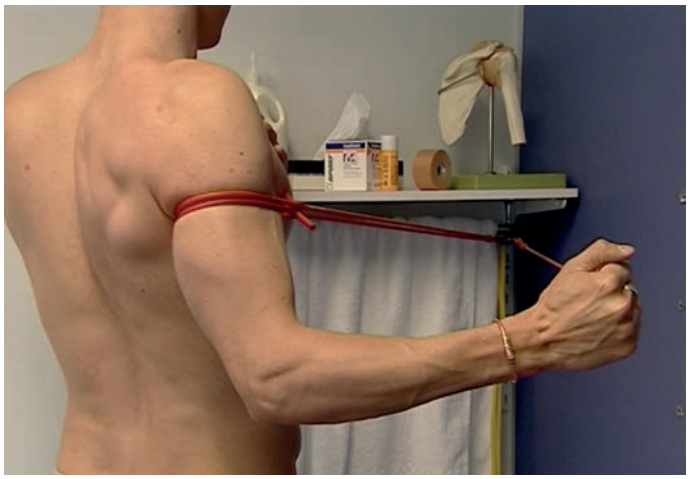

Figure 3: ***External rotation strengthening with a proximal Theraband™ for facilitation of humeral head control***

Specific muscle groups that tested as weak were selected in lower ranges to strengthen with exercises such as side lying external rotation for posterior cuff, and bent over rows for posterior deltoid (Figures 4a & 4b).

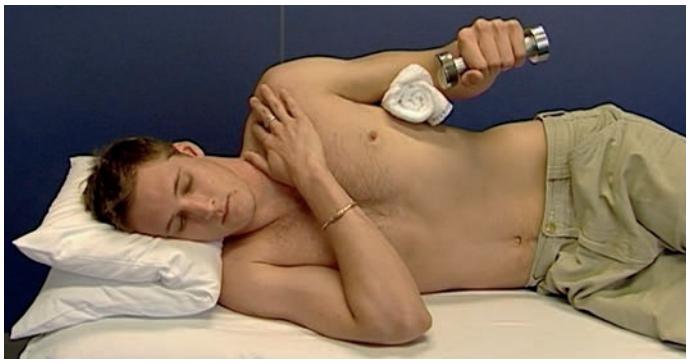

Figure 4a: ***External rotator cuff strengthening; scapula or humeral head control Therabands™ could be incorporated***

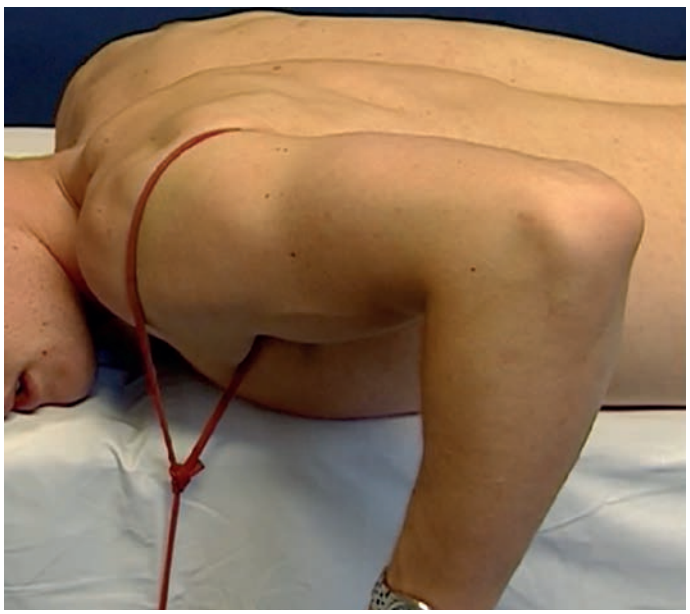

Figure 4b: ***Posterior deltoid strengthening with a scapula facilitation Theraband™***

Once control was established in lower ranges, drills were moved up to 45°-60° abduction / coronal plane. As higher ranges of coronal plane were established, weights were introduced into

higher ranges as required. Once higher ranges of strength and control were established, movement control drills were moved around in the plane of the scapula at 0º - 45º and then the flexion / sagittal plane 0º - 45º. Weights / resistance training could be performed within this movement arc. The same principles were then applied to higher ranges of abduction and flexion control (Figures 5a to 5d).

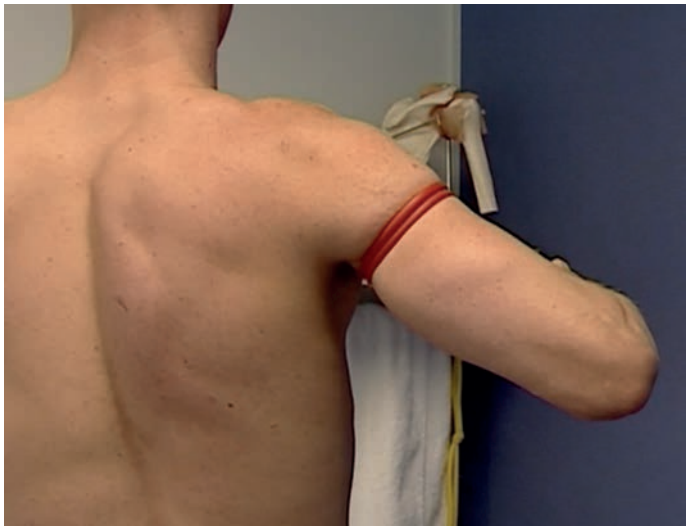

***Figure 5a:** Extension strengthening at 45° abduction*

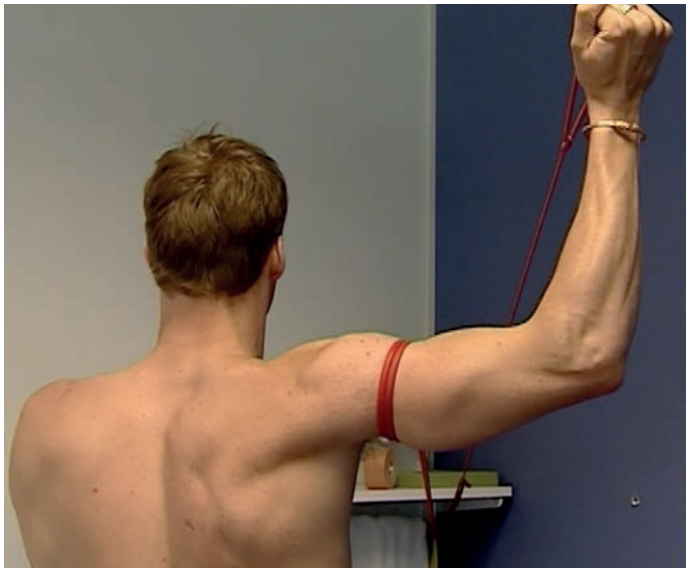

***Figure 5b:** External rotation strengthening at 90° abduction*

***Figure 5c:** Flexion strengthening into elevation*

***Figure 5d:** External rotation strengthening at 90º abduction in horizontal flexion with scapula upward rotation Theraband™*

- Phase 4: The final phase introduced functional specific training drills and strength work as determined by the patient's sporting and occupational requirements, and as they were able to demonstrate control of the scapula and humeral head within that movement strategy.

All outcomes were re-measured at 12 weeks post commencement of rehabilitation, although not all patients had completed their rehabilitation programme at the 12 week stage. Once the participants were happy with their own functional level, including sporting activities, they were discharged. The average discharge time was five and a half months.

The results of this clinical trial demonstrated significant functional improvements in all three functional outcome questionnaires ($p<0.001$). There was also a significant increase in rotator cuff, deltoid and scapula stabiliser muscle strength ($p<0.001$). Upward rotation of the scapula was also significantly increased at 0º, 30º, 45º and 60º of abduction ($p<0.01$). Intra-muscular EMG demonstrated the rotator cuff switches on significantly earlier in patients with MDI than it does in individuals with normal stability ($p<0.05$); this is possibly an attempt by the rotator cuff to stabilise the humeral head. EMG also demonstrated a significant decrease in the percentage of MVC of the upper and middle trapezius ($p < 0.008$, $p < 0.015$). This would contribute to the decrease in upward rotation of the scapula seen in lower ranges of abduction.

The MDI group showed much less variability in all EMG characteristics observed pre-intervention, compared to normal, across all muscles tested. Post rehabilitation, there was a significantly earlier onset of upper trapezius and subscapularis in the MDI population, the significant decrease in upper and middle trapezius was normalised, and there was increased variability demonstrated across all EMG characteristics across all muscle groups tested.

Conclusion

The results of this clinical trial demonstrate that physiotherapy, in a group of MDI patients who completed a conservative rehabilitation programme, could alter scapula resting position and motion through range abduction, restore scapular upward rotation in lower ranges of abduction and increase shoulder muscle strength in rotator cuff, deltoid and scapula stabilisers. It could also improve pain, instability and function as determined by instability specific outcome questionnaires. According to the dynamic systems theory, variability in motion is normal and ideal and it is what enables us to cope with adaptation to a dynamic environment. There is a suggestion that the broader spectrum of EMG variability achieved is associated with the improved functional output (Davids *et al* 2004; Bartlett *et al* 2007). Potentially, by allowing patients with MDI to move with more than one motor strategy, we are dispersing soft tissue loads and diminishing the stress of repetitive microtrauma on the capsuloligamentous complex. One could reflect that, theoretically, the rehabilitation programme employed in this study altered motor patterning, but further research is required to know this conclusively.

About the authors

Lyn Watson is a clinical manipulative and sports physiotherapist who has been working exclusively in the diagnosis, assessment and rehabilitation of shoulder injuries for the past 25 years. She is the Clinical Shoulder Physiotherapy Specialist at LifeCare Prahran Sports Medicine Centre and Melbourne Orthopaedic Group.

Lyn graduated in 1986 with a Bachelor of Applied Science in Physiotherapy (Hons) from La Trobe University. She completed her postgraduate training in Manipulative Physiotherapy in 1990 and was granted her Sports Physiotherapy title in 2000. Lyn is currently finalising a Clinical Doctorate through La Trobe University. She has been conducting clinically relevant, published research since 1996. Her research has been published in the *Journal of Shoulder and Elbow Surgery, Manual Therapy* and *British Journal of Sports Medicine*.

Lyn is currently working with the shoulder surgeons at Melbourne Orthopaedic Group collecting data and researching the long-term clinical outcomes of many common shoulder surgeries and their physiotherapy rehabilitation programmes. She is also the Specialist Shoulder Physiotherapy Consultant to a variety of national and international teams and individual athletes. She has been consulting to the Australian Cricket Team since 1994, and working with Australian Olympic-level swimmers, water polo, beach volleyball, sprint and slalom paddlers, rowers and professional tennis players for over 20 years. Her clinical practice has a balance of elite athletes, weekend warriors, office workers and manual workers of all age groups. As such, she treats and specialises in the everyday typical shoulder conditions such as rotator cuff pathologies, instability, impingement and the stiff shoulder.

Lyn is an internationally renowned lecturer, course presenter and clinician. Over the last 25 years she has developed a logical and progressive approach to the assessment, diagnosis, rehabilitation and management of the shoulder and shoulder girdle. She has been teaching this approach in her shoulder physiotherapy courses throughout Australia, Canada, United Kingdom, France, Italy, USA, South Africa and Asia since 1992. She also lectures on the Masters of Manipulative Therapy and Sports Physiotherapy programmes for both Melbourne and La Trobe universities.

Both of her Level 1 and Level 2 courses are fully accredited by the Australian Physiotherapy Association.

Simon Balster worked in Hydrotherapy and Sports Medicine practices in Brisbane for two years before taking up a position at LifeCare Prahran Sports Medicine Centre in 1997 to work alongside Lyn Watson.

Today, Simon specialises in the assessment, diagnosis and treatment of the full spectrum of musculoskeletal shoulder and shoulder girdle problems. He treats a wide variety of post-operative sporting and occupational orthopaedic conditions, and often attends surgery at the Melbourne Orthopaedic Group.

Simon has conducted clinical research and has published papers in *Manual Therapy* and the *British Journal of Sports Medicine*. In addition to his clinical practice, Simon is part of a research group that includes both Lyn Watson and Tania Pizzari and their results are published in peer-reviewed journals, *Manual Therapy* and the *British Journal of Sports Medicine*.

In 2005, Simon began working with Lyn on an educational resource to assist physiotherapists with the many assessments of the shoulder. This culminated in the General Assessment of the Shoulder, a CD-Rom that details every shoulder assessment tool used in a clinic. The second resource in this series is the Exercise and Rehabilitation of the Shoulder.

Simon is also involved in lecturing, teaching and tutoring on a variety of shoulder topics.

Tania Pizzari graduated from La Trobe University with honours in 1997 and with a PhD in 2002. She lectures and conducts research part-time in the La Trobe University Department of Physiotherapy. She regularly conducts lectures and practical sessions for the Australian Physiotherapy Association on shoulder, knee and hamstring injuries and is a member of the Shoulder and Elbow Physiotherapists of Australasia. She is a regular speaker at national and international sports medicine conferences. Tania is able to provide particular expertise in the diagnosis, management and rehabilitation of shoulder conditions as well as the treatment of musculoskeletal conditions in children.

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Reorganised sensorimotor control in neck pain and the benefits of training

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This article is reproduced from the lecture, originally delivered at the Physio First 2015 Conference, and briefly reviews evidence which describes alterations in neuromuscular function of the cervical spine in patients with neck pain and outlines the benefits and limitations of therapeutic exercise for cervical spine disorders.

Learning outcomes

1. Review the contemporary evidence base for sensorimotor adaptations in people with neck pain disorders.
2. Review contemporary theory of motor adaptation to pain which considers the diversity and intricacy of individual change in sensorimotor control.
3. Recognise the principles of practice for motor control interventions in patients with neck pain.
4. Identify patients that are likely to respond to motor control interventions.
5. Appreciate the scope and limitations of exercise for the rehabilitation of patients with neck pain.

Introduction

Neck pain is often a disabling and recurrent disorder characterised by periods of remission and exacerbation (Cote *et al* 2004). Disturbingly, a study has suggested that only 6.3% of individuals who suffered from neck pain in the previous year were free of recurrence (Picavet & Schouten 2003). This tendency for chronicity of many neck pain disorders may, at least in part, be attributed to maladaptive changes in neuromuscular control of the neck, especially considering the heavy dependency the cervical vertebral column has on muscles for physical support (Panjabi *et al* 1998).

There are an ever growing number of studies identifying neuromuscular dysfunction in individuals with neck pain. Although deficits in motor control may lead to poor control of joint movement, repeated micro-trauma and thus, eventually, to pain, there is convincing evidence from experimental pain studies that pain can provide an initial trigger for changes in neuromuscular control (Falla *et al* 2007a, 2007b; Cagnie *et al* 2011). In many cases, the immediate reorganisation of the motor strategy which occurs during experimental neck pain mimics the altered motor strategies identified in people with clinical neck pain disorders. Furthermore, there is evidence to suggest that even with a reduction in symptoms, the altered muscle behaviour does not necessarily automatically reverse with time (Sterling *et al* 2003). This enforces the need for high-quality and specific rehabilitation, especially if the altered patterns of muscle activity involve less efficient combinations of muscle synergies which may increase the vulnerability of the cervical region to strain and further pain.

Features of sensorimotor adaptation in neck pain

In general, sensorimotor adaptations to pain and / or injury presents across a spectrum, from subtle changes in sharing of load between synergist muscles (Hodges *et al* 2013; Muceli *et al* 2014) or the distribution of activity within a muscle (Tucker *et al* 2009; Falla *et al* 2009), to a complete avoidance of movement or function (Vlaeyen & Linton 2000).

Specifically for the neck, biomechanical disturbances include reduced range of motion (Antonaci *et al* 2002; Sjolander *et al* 2008; Dvir *et al* 2006) and reduced concurrent motions in the associated planes (Woodhouse & Vasseljen 2008), increased range of motion variability (Sjölander *et al* 2008; Vogt *et al* 2007), decreased movement speed (Grip *et al* 2008; Ohberg *et al* 2003) and reduced smoothness of movement (Sjölander *et al* 2008; Grip *et al* 2008). Greater error in repositioning the head following voluntary movement has been observed in people with neck pain of both insidious (Revel *et al* 1991) and traumatic onset (Heikkila

& Astrom 1996; Kristjansson *et al* 2003; Treleaven *et al* 2006), although proprioceptive acuity is most affected in people with chronic whiplash associated disorders (Kristjansson *et al* 2003) especially in those reporting higher pain and disability (Feipel *et al* 2006) and dizziness (Treleaven *et al* 2003). In addition, people with whiplash-induced neck pain show reduced shoulder and elbow proprioception (Knox *et al* 2006; Sandlund *et al* 2006) which likely affects co-ordination and movement of the upper limb.

Disturbed postural stability may be a feature in some people with neck pain and includes larger postural sway during quiet standing and dynamic tasks such as walking up and down stairs (Karlberg *et al* 1995; Michaelson *et al* 2003; Sjöström *et al* 2003) and reduced stability in response to predictable and unpredictable perturbations (Michaelson *et al* 2003).

Changes in the behaviour of neck muscles are well documented in people with neck pain. For instance, superficial neck muscle activity has been shown to be augmented during isometric contractions (Falla *et al* 2004a; Jull *et al* 2004; Chiu *et al* 2005; Descarreaux *et al* 2007) and functional upper limb activities (Szeto *et al* 2005; Nederhand *et al* 2000; Falla *et al* 2004b) in several neck pain patient populations, including cervicogenic headache (Jull *et al* 2007a, 1999), whiplash-induced neck pain (Sterling *et al* 2003; Jull *et al* 2007b), occupationally induced neck pain (Johnston *et al* 2008), as well as non-specific neck pain groups (Falla *et al* 2004c). Patients with neck pain also show reduced specificity of superficial neck muscle activity which includes increased activation when acting as an antagonist (Falla *et al* 2010; Lindstrøm *et al* 2011). Such observations may reflect an attempt to stiffen the spine to compensate for poor passive or active segmental support (Cholewicki *et al* 1997).

Activity of the deep cervical flexors longus colli, longus capitis (Falla *et al* 2004c), and deep extensors semispinalis cervicis, and multifidus (O'Leary *et al* 2011; Schomacher *et al* 2013; Schomacher *et al* 2012) may be reduced in the presence of neck pain and during postural perturbations, the onset of the deep cervical flexors is delayed (Falla *et al* 2004d), reinforcing observations of deep spinal muscle dysfunction.

Variability of sensorimotor adaptations

Neuromuscular adaptations have been documented in people with neck pain with varying degrees of pain severity, varying duration of pain and different pain aetiologies suggesting that neuromuscular dysfunction is a generic finding. However, individuals do not present with the same neuromuscular impairments to the same extent. On the contrary, neck pain is heterogeneous both in terms of the associated pain mechanisms, and physical and psychological features. In relation to the extent of neuromuscular impairments, there is substantial variability between individual patients. This variability is partially related to the magnitude of pain and thus the individual variability of the patient's presentation (Falla *et al* 2004b, 2011).

Studies have shown that the degree of neuromuscular impairment present before training may be an important determinant of symptomatic response to exercise. For example, specific training of the deep cervical flexor muscles in patients with neck pain reduces pain and increases the activation of these muscles, especially in patients with the least activation of their deep cervical flexors prior to training (Falla *et al* 2012). These findings suggest that the selection of exercise, based on a precise assessment of the patients' neuromuscular control, and tailored exercise interventions are the most beneficial for patients with spinal pain.

Training to address sensorimotor adaptations

Neuromuscular and functional changes in response to training are specific to the mode of exercise performed (Adkins *et al* 2006; Coffey & Hawley 2007; Fluck 2006; Gabriel *et al* 2006). A contemporary training approach for neck pain (Jull *et al* 2008) involves initially facilitating activation of the deeper spinal muscles with repeated isolated voluntary contractions to induce neurophysiological adaptations. Exercise is then progressed to resistance training, with the aim of inducing morphological adaptations in order to ameliorate endurance and strength of selected muscles and movements, and reversing chronic changes in muscle structural properties such as atrophy and fatty infiltration of muscle tissue (Elliott *et al* 2006, 2008, 2009).

Specific motor control training can change control of the deep and superficial muscles in neck pain. Targeted training of the deep cervical flexors increases their activation during an isometric task (Jull *et al* 2009), improves the speed of their activation when challenged by postural perturbations (Falla *et al* 2012; Jull *et al* 2009) and enhances the degree of directional specificity of neck muscle activity during multidirectional isometric contractions of the neck (Falla *et al* 2013). Activity of superficial neck muscles can also be reduced with specific motor control training (Jull *et al* 2009), even after a single session (Lluch *et al* 2014). Deep muscle control does not appear to be changed by generic forms of exercise (Jull *et al* 2009).

Studies also show that the degree of improvement in motor control is associated with the extent of symptomatic improvement. For example, enhanced capacity to recruit the deep cervical flexor muscles after training is associated with the degree of pain reduction in patients with chronic neck pain (Falla *et al* 2012).

Although programmes of supervised exercise may provide a clinical benefit both in the short and long term, the response to exercise is highly variable with responses ranging from excellent outcome to no relevant benefit (Falla *et al* 2013, 2006; Jull *et al* 2002; Michaleff *et al* 2014). People who respond well are likely to be individuals whose peripheral nociceptive input is continuing to drive their experience of pain. In contrast, it is important to recognise that people who do not respond to exercise interventions may have other causes driving their pain experience, e.g. central sensitisation. Thus, although it is unquestionable that sensorimotor control is affected, the key challenge facing clinical intervention is

to decide how sensorimotor changes relate to an individual patient's presentation, which aspects of sensorimotor control require management, and how this might be best achieved for the patient.

About the author

Deborah received her PhD in Physiotherapy from the University of Queensland, Australia in 2003. In 2005, she was awarded Fellowships from the IASP and the NHMRC to undertake postdoctoral research at the Centre for Sensory-Motor Interaction, Aalborg University, Denmark, where she was Associate Professor from 2007 to 2010. In 2012, she was appointed to Professor at the University Hospital Göttingen, Germany. Her research focus involves the integration of neurophysiological and clinical research in neuromuscular control of the spine in people with chronic pain. She has published over 100 journal papers and over 100 conference papers / abstracts. She is an international lecturer and educator. She is co-author of the book *Whiplash, Headache and Neck Pain: Research Based Directions for Physical Therapies* and co-editor of the 4th Edition of *Grieve's Modern Musculoskeletal Physiotherapy* and is Associate Editor of the journal *Manual Therapy*.

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Managing ongoing pain post ankle sprain: a practical approach

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The initial management of ankle injuries is something all physiotherapists are familiar with. What we are focusing on in this article are possible causes and treatment options for those patients who are still having problems despite the intervention of the physiotherapist.

Learning outcomes

1. Understand the multiple causes of ongoing pain post sprain.
2. Undertake clinical assessment and know what to look for when seeing these patients.
3. Develop clinical reasoning skills to determine the most appropriate management for ongoing pain post sprain.
4. Gain a greater understanding of what the surgeon can offer these patients.

When do I worry?

I am often asked when a clinician should worry about an ankle if the pain is not settling down. If it's not improving in the first two weeks, then a plain x-ray is a good idea. At that stage a missed fracture can still be identified and surgery is almost the same as if it had occurred on day one. Indeed, the trend in managing these fractures has now changed and the majority of Weber A and B fibula fractures which are stable and minimally displaced are treated without surgery.

Avulsion fractures of the fibula, when identified on x-ray, often cause the patient great anxiety, but in reality rarely need treatment. It just suggests it is a more serious injury that may take longer to settle down.

There are three other injuries that require vigilance: 5th metatarsal base fractures are caused by a similar mechanism to an ankle sprain and are sometimes missed on x-ray; a fracture of the anterior process of the calcaneus is another common injury which can be missed on the initial x-rays and is often diagnosed in retrospect; and a high fibula fracture with subtle disruption of the syndesmosis is sometimes not picked up on standard x-rays.

Anterior ankle impingement

Assuming there is no missed fracture, our next milestone, by which time some recovery should have been noted, is the six-week mark. At this stage most ankles will have settled down. Some don't, and this is the focus of this article. There are many causes for ongoing pain post ankle sprain. At this stage it is important to remember the common causes and consider and assess these first.

One of the most common causes of ongoing pain post sprain is anterior ankle impingement. The anterior talo-fibular ligament (ATFL) is a broad, but thin structure. When this is damaged, sprained, torn or avulsed, it tends to heal thickened. Scar tissue also builds up around the ligament and within the joint a pattern of reactive synovitis. The ankle is a tight hinge and this inflamed tissue then gets pinched in the lateral corner or gutter of the ankle, in either dorsi- or plantar-flexion.

The treatment of ankle impingement starts with the therapist. Local treatment including massage, ultrasound and ice are all beneficial. This condition is not easily diagnosed on MRI, as the tissue is too small to be seen, but MRI is helpful in excluding other sources of pain.

The diagnosis is made on clinical examination by applying gentle pressure to the anterolateral corner of the ankle, on top of the ATFL, and then dorsi-flexing the ankle. This should recreate the pain. If I am concerned that there is an impingement, then, if the patient is still sore eight weeks post injury, I may advise a cortisone injection to reduce the inflammation and probably by

"shrinking" the inflammatory tissue, and stopping it from pinching in the "hinge" of the ankle, ease the pain. Where pain persists, I sometimes remove the inflamed tissue arthroscopically using a small powered shaver. As the tissue is away from the articular weight-bearing surfaces of the joint, these patients often recover quickly from this type of surgery (Figure 1).

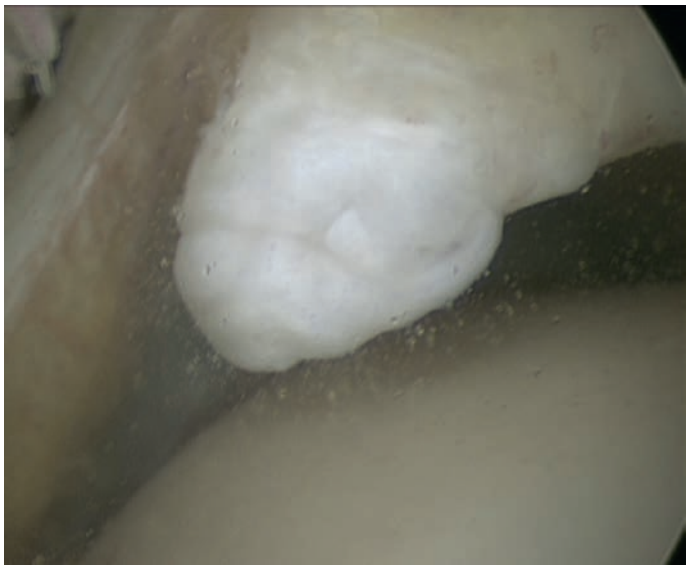

***Figure 1:** Intraoperative picture showing anterior impingement in the anterolateral corner of the ankle*

Osteochondral lesions of the talus

Osteochondral lesions of the talus cause a deep-seated ankle pain that persists. Many are traumatic in origin, often related to a twisting injury of the ankle. In younger patients it may be due to osteochondritis dissecans (OCD). Medial talar dome lesions tend to be more posterior so cannot be palpated; the presence of a residual effusion in the joint with deep ankle pain is what usually guides me to this as a differential diagnosis.

Osteochondral lesions of the lateral talar dome tend to occur more anterior and therefore may be palpable or tender when the talus is palpated with the ankle in full plantar flexion. Plain radiographs can sometimes show the lesion which is a slightly ominous sign as it means the OCD is cystic and large. Smaller lesions may only be visible on MRI or CT. Each method of detection has its own benefits and drawbacks; MRI is very sensitive but can overestimate the extent of the lesion while CT can miss the smaller ones but gives a clearer guide as to the extent of any cystic change in the underlying talus.

A lesion that is not painful can be left alone. If it hurts, then the lesion can be treated with arthroscopy and microfracture. The loose cartilage is debrided to a stable position and the underlying bone's subchondral plate is disturbed with a pick or a burr to stimulate some bleeding. Fibro cartilage then forms over the bare bone base. It is not as good as normal cartilage, but is a reasonable second best! We know that the size of the lesion is important. Studies suggest that lesions smaller than 10mm in diameter do better than those that are larger. Alternative treatments such as osteochondral grafting and autologus chondrocyte grafting do exist, but the results are not superior to microfracture and the surgery is more involved. It tends to be reserved for those cases where pain persists despite repeat microfracture.

Mechanical instability and ligament reconstruction

Where a patient has repeated sprains because of the ligaments being permanently lax or mechanical instability, they are at risk of permanently damaging the ankle joint. Once the diagnosis of the extent and severity of the ankle instability (the ankle giving way) is made then a treatment plan can be initiated. A strengthening programme is helpful; this is true even with patients who have had recurring sprains and chronic recurrent instability. About 50% of patients will respond to a regime of intensive peroneal strengthening and balance exercises.

Where the ankle remains unstable, surgical repair of the chronically loose ankle ligaments can be performed. There are many different techniques used to stabilise the ankle. The one I most commonly use is the Brostrom-Gould lateral ligament reconstruction. In this operation the lax ligaments are first taken off the bone, they are then secured with a special stitch and advanced and reattached using anchors into a bony channel made on the fibula bone (Figure 2).

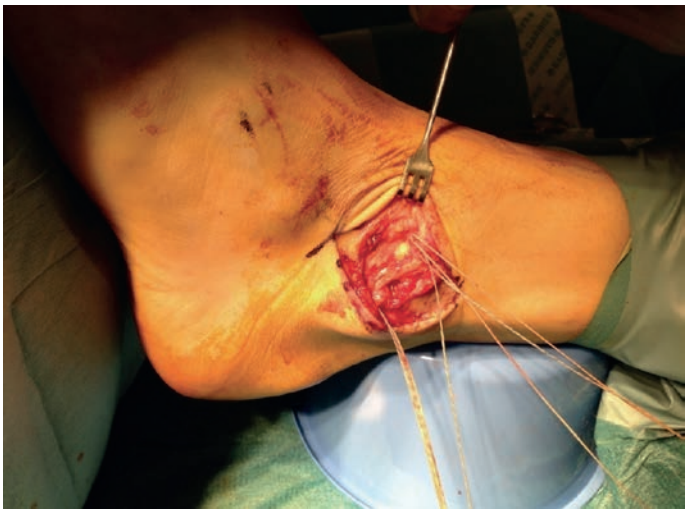

***Figure 2:** Sutures have been placed in the ATFL, Calcaneofibural Ligament and Anterolateral Capsule*

At the same time I use another local structure, the extensor retinaculum, to reinforce the repair. This operation usually works very well, allowing individuals to return to all forms of athletic activity without risk of recurrent injury to the ankle. Occasionally, a tendon behind the ankle (the peroneal tendon) or a tendon graft may need to be used.

Posterior ankle impingement

Posterior ankle impingement is caused by traumatic injury, or overuse, and particularly affects dancers, football players, runners and other athletes. Sometimes dancing or running on a hard surface contributes to the problem although, in some cases, there is a slight difference in the normal foot and ankle anatomy that

eventually leads to posterior ankle impingement. Whatever the cause, the end result is the same: chronic ankle pain, at rest and with palpation, along the back of the ankle, pain with movement, and loss of ankle plantar flexion.

One common cause of posterior impingement syndrome is the os trigonum – an extra piece of bone that develops behind the ankle bone and is present in affected individuals. Pointing the toes downward catches the os trigonum between the ankle and heel and repetitive force downward on the os trigonum every time the foot is pointed causes the bone fragment to pull loose. As the os trigonum pulls away, the tissue connecting it to the talus is stretched or torn. The area becomes inflamed causing pain and loss of ankle motion (Figure 3). Conservative care with physiotherapy and injections is the first line of treatment. If this is unsuccessful, surgery can remove the offending tissue, e.g. bone fragments, scar tissue, thickened joint capsule, etc. In recent years, this surgery has developed into a keyhole operation with a much quicker recovery.

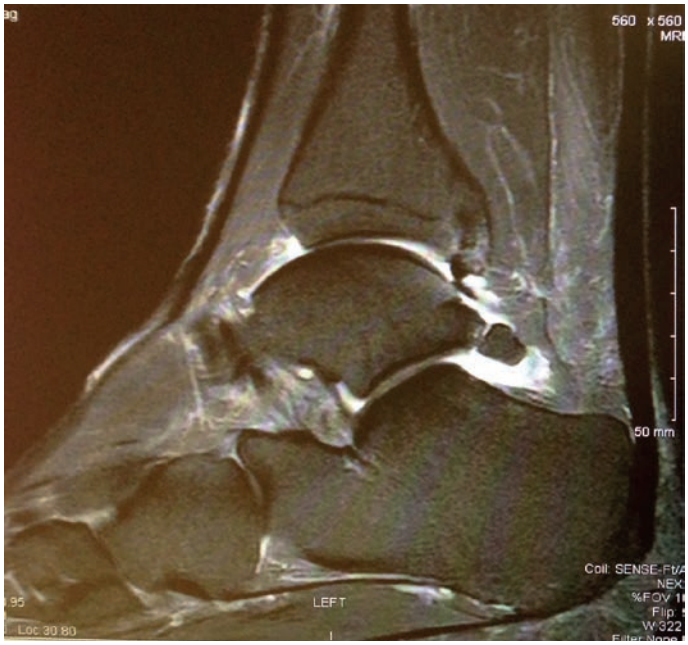

Figure 3: ***Sagittal MRI shows an os trigonum with fluid around it***

Peroneal tendon injury

The two peroneal tendons lie immediately behind the fibula, the bone on the outside of the ankle. They move the foot outwards. They are important in balance as they counteract the inward motion that usually causes sprains or instability. They are slightly weaker than the muscles and tendons on the inside of the ankle and are prone to injury as the ankle turns, rolls or becomes sprained.

Tears of these tendons do occur. One or both of the tendons can be torn. This leads to swelling, pain and a sense of instability behind the outside of the ankle.

Occasionally, the tendons can be injured in either a fall or an athletic injury. They pop out of the supporting ligaments that hold them in place and dislocate. Once this occurs, recurrent dislocation and tearing of the tendons is inevitable. If the tendons dislocate acutely in an injury, they need to be repaired to prevent future tearing of the tendons. Once dislocated, the tendons can only be stabilised through surgery.

The diagnosis of peroneal tendon injury is made through careful examination and palpation. An MRI or ultrasound may be required to document the extent of the tear.

If the tendons continue to be sore, the diseased tissue needs to be excised. After this the tendons can be repaired. Sometimes both tendons need to be attached together or sutured to a bone through tenodesis.

While the above list is by no means complete, it does give us an idea as to how to embark on assessing ongoing pain post ankle sprain.

About the author

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From transactional to transformational

James Butler
Director, Painless Practice

This article is reproduced from the lecture, originally delivered at the Physio First 2015 Conference, during which it was discussed whether clinicians found that the realities of running a private practice matched with their dreams of doing so, and how Physio First members could thrive and survive in the current marketplace.

Learning outcomes

1. Understand how to analyse the patient base of a private practice from a business perspective.
2. Understand how to determine the nature and type of practice most appropriate for the owner.
3. Understand how to create a plan to make that vision a reality.
4. Understand the key steps in getting started on implementing that plan.

Introduction

Physiotherapists who have followed their dream to go into private practice face numerous pressures and a constantly changing environment and the reality doesn't often match the dream. This could be because, in the view of Painless Practice, the profession has become too transactional and is shying away from engaging fully in the challenge of transforming the wellbeing of patients. There may be understandable reasons, so this is not a criticism, but there are indications that a significant part of the physiotherapy process is conducted at a surface or transactional level. Greater impact, greater value and greater commercial resilience, however, comes from deeper interactions that seek to transform wellbeing, and it is only with this deeper connection that the physiotherapy profession will fight off the trend towards commodity treatment: provided at lowest cost, for the shortest time.

It can be argued that the true measure of success for a course of physiotherapy treatment is not in the adherence to a private medical insurer's (PMI) guidelines on treatments per episode, or in it coming within a contracted NHS service's budget. It is, surely, whether the health and wellbeing of the patient has been transformed for the better.

The aim, therefore, is to deepen the level of interactions in the pursuit of transforming wellbeing, rather than just to transact in the field of healthcare. To illustrate this view, we consider this premise in the context of practice purpose, in the relationship with patients, with regard to private medical insurance and in relation to the people working in clinical teams.

What is the point of being a physio?

Cast your mind back to that day when you first realised that physiotherapy was the career for you. Perhaps then, or a little time later, you realised that the NHS wasn't for you and you wanted to step out into private practice. On those two occasions, what was it that excited you about the profession? What was the point of you choosing this path and not becoming an astronaut or a firefighter?

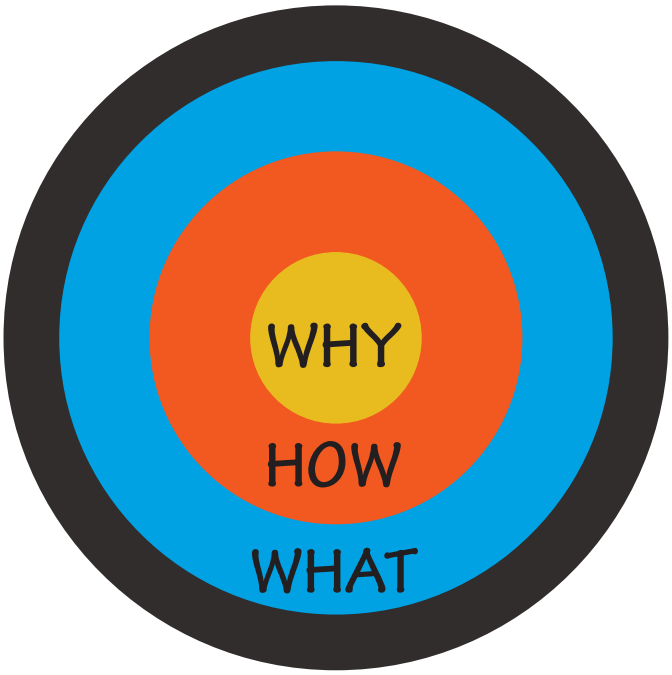
Once in practice, after years of study and the daily grind of treating patients, it is easy to become detached from that deeper motivation. As a group of mainly small businesses, it can be easy for the profession as a whole to lose its own collective focus until there comes a point when everyone is working incredibly hard but can't quite remember what for.

When we talk to physios about their clinics and how to make them more successful, we find most are well aware of **WHAT** they do; they know they are physios, they know what specialisms they have and, while they will seek continuing development, they know their craft and can normally articulate it to others.

A smaller group of physios also know **HOW** the way they treat differs from others and from other professions; something that develops after a number of years in practice. Some can also explain how their techniques, tools and experience help patients in certain ways, which is knowledge that is often the hardest to replicate or disseminate to others. When such an experienced

physio is working in a team, particularly where it consists of junior members, this can lead to the cult of the principal – where patients only want to be seen by that most knowledgeable practitioner, who then has a waiting list, while their associates have "gappy" diaries.

However, when we talk to clinicians about **WHY** they do what they do and what they see is the point of their practice, it is amazing how many struggle to answer the question because it is something they have rarely stopped to think about. Simon Sinek's excellent book *Start With Why* tells us that people don't buy what you do or how you do it, they buy why you do it. If you don't know your "why", how can you attract patients who believe in it?



If you are looking for inspiration, motivation and better ways to connect with your potential patients, spend some time to really consider what your point is: why you are a physiotherapist in private practice at all. I would hazard a guess that your "why" will be, in some way, about transforming not transacting. The personality types who choose physiotherapy as a profession are generally empathic, people-centred, rather than process or task-oriented. Transacting is about a task, transforming is about people.

If, indeed your answer to "why" is to improve your patients' wellbeing, have an impact on your community, and see people getting better, then you may want to review your transformational interactions by:

- taking a few moments to watch Simon Sinek's excellent TED talk online at www.ted.com/talks/simon_sinek_how_great_leaders_inspire_action
- taking more time to consider your own "why"
- considering the implications for your own clinical practice, your clinic and your profession.

If finding your own "why" proves inspirational, why stop there? Ask the same of your practice team.

Transforming patients

In the busy world of running a clinic, it is easy to find yourself on the hamster wheel of day-to-day business: running faster to turn the wheel, but never seeming to get anywhere. For the individual practice and for the profession, a wide range of assumptions about the service build up and become the orthodoxy. In particular, it is very easy to make assumptions about what patients want and never challenge them.

Have you ever asked your customers what they need or want? As the fundamental model for any business is to meet their customers' needs, profitably, then it makes sense that the first task of any business owner is to properly understand what customers need or want. However, patients sometimes don't know, or don't know how to articulate what they want. Here, we can consider Steve Jobs and his genius. He could see the vision for iTunes and iPods before any of us knew we wanted them. So, taking that model, in light of your practice purpose, what do you want for your patients?

You know that your patients want to get better, to restore function, reduce pain, increase sustainable recovery, but even each of these needs can be multi-dimensional. On a deeper level, most people will want the increased wellbeing for personal reasons, such as return to sport, enjoy a skiing holiday, or to be able to bear children. The more emotive the reason, the more you can transform their lives.

In the face of overwhelming demand, physiotherapy in the NHS is increasingly transactional. The waiting time for an "urgent" appointment with a physio can be two weeks, 11 weeks for non-urgent. Under this pressure, responsibility for recovery is increasingly transferred to patients by means of home-based exercise plans rather than with sessions of manual therapy, thereby reducing the time the patient will spend in being treated in the clinic. It could be argued that this approach empowers patients to manage their own care, but it can also been seen as an abdication of responsibility by the therapist due to the incredible pressure of patient load.

Patients who are able to access the private sector want something else for their money. They expect shorter waiting times, longer appointment times, more manual therapy and greater engagement from their practitioner in the treatment process. They do not expect to be seen as just a number or merely a transaction.

This is a huge opportunity for the private physio to offer transformative healthcare and see the patient through to a proper recovery, not just "recovered enough". Through health, prevention and lifestyle advice, they can possibly ensure that their patient feels better than they have felt in years. That is transformational and for many people is a service worth paying for.

Clinics that struggle often do so because they are trying to apply an NHS model in the private sector, charging as little as possible, for as few treatments as possible and just transacting with patients. This is often because physios worry (unnecessarily) about a person's ability to pay, and shy away from being bold and engaging patients in a full and proper programme of recovery. These physios are not doing any harm, but are they doing enough good, when the private sector has the opportunity to do so much more?

The inadvertent drive to transactional healthcare

At first glance it would appear we have a two-tier healthcare system in the UK:

- The publicly funded NHS
- The PMI funded sector

I would argue, in fact, that a third tier exists:

- The self-funded private sector.

These tiers are defined not by where they take place, as all three can be operational in the same clinic, but rather by the expectations of the level of transaction. In my experience, clinics that are PMI funded follow a similar trend to that of the NHS, as described earlier. The insistence of some major insurers to drive down costs and engender a race to the bottom is well known. Playing devil's advocate, one might be able to understand their position and have some sympathy; if I ran a PMI and saw my spend on physiotherapy spiralling out of control, I would probably want to take action.

Whatever the motivation, the effect on private physiotherapy is clear. Market forces have led to reduced fees, limits on length of treatment episode and increased administrative requirements. This all points to a more transactional interaction with patients, perhaps with shorter waiting times, better facilities and a bit more hands-on interaction than the NHS model, but not the in-depth transformational interaction a self-paying patient might receive. Anecdotally, there is also a trend towards the clinic having less experienced (cheaper) physiotherapists provide PMI funded treatments in an attempt to make the numbers work.

Two questions that practice owners should ask themselves are:

Is the PMI market one that I actually want to play in?

If one considers the purpose discussed earlier, and that purpose includes some aspirations to build meaningful interactions with patients that truly transforms their wellbeing, that doesn't just get them partly better but truly partners with them on their healthcare journey, then, arguably, the clinician must decide that the constraints of providing PMI funded treatments are not practicable.

Another reason for saying no might be that the economics of the PMI model mean that the payments for treatment can be less than the cost of production; why would a business do that? In such circumstances the private physiotherapist has a choice to decline to work with insurance funded patients.

How do I adjust my model to make it economically sustainable both for me and the PMI?

For those who do decide that they want or need to accept insurance funded patients, then a different way of thinking must be seriously considered. When the average number of treatments per episode is limited, the fee per treatment is often below market rate, and the administrative burden of evidencing outcome, chasing payment and managing imperilled cash flow is increased, the practice must find ways to adapt in order to survive. Many physiotherapists who have their heads down, spending every hour they work in treating patients, do not see, plan for and react to this need for change until it is too late.

For those who treat or are considering contracting to treat insurance funded patients, it is worth reviewing:

- the pressures of treating these patients
- whether your purpose can be fulfilled while keeping within the terms of PMI contracts
- alterations you are willing to make to your clinical practice to meet the competing pressures
- whether you and your business have a long-term future with PMI funded patients.

While PMIs can dominate the market and dictate pricing to small players such as physiotherapy clinics, it is not legal for those clinics, as a group of suppliers, to agree a floor treatment price as this would be considered a cartel, so there may be room for a co-operative approach, perhaps driven by a professional body which negotiates better contracts with PMIs and then farms treatments to members, in the way an intermediary would, but perhaps with

different principles. It could work, and it could produce greater parity in the market, but it is not without its own pitfalls.

Transforming people

Many physiotherapists over the past year seem to have experienced a challenge with recruitment. A long-term decline in graduate numbers and an increased willingness for physiotherapists to start out on their own, rather than become associates, seems to have made it harder to recruit across all of the musculoskeletal (MSK) professions, i.e. physiotherapy, chiropractic and osteopathy. Physiotherapists face a further pressure in the large competitor (the NHS) offering employment opportunities, which has less of an impact on the other two MSK groups.

One possible theory for this situation is that a large part of this type of employment environment is shallow and transactional. In my experience, deeper, transformational interactions are the exception rather than the norm because:

- self-employment can lead to a more transactional principal-associate relationship. Loyalty from both parties can be less: where a self-employed associate has allegiances to multiple practices in multiple locations, their engagement is often fragmented
- principals and associates often pass like ships in the night, with limited training, appraisal or other opportunities for deeper interactions. Sometimes this is deliberate, consciously or sub-consciously, on the part of the principal, and sometimes it is just a product of the reality of hectic diaries in a busy practice.

The consequences of this transactional management style is, therefore, likely to result in self-employed associates who perceive that their main interaction with the practice principal is limited to whatever financial transaction they have agreed to, deciding to set up on their own and have full control over their own earnings.

Proactive principals are realising that, to recruit, retain and transform their teams, a deeper involvement is required.

Employment contracts can provide greater stability, assured income, and show deeper commitment. Training programmes, appraisals, occasional one-to-ones and generally showing a bit of attention can improve skills and motivation, create greater loyalty and increase patient retention. If you are a practice principal, you may want to consider the following questions:

- What level of relationship do you have with the people in your team?
- What would transform that relationship, build loyalty and motivation and increase retention?
- What would transform your team members as individuals?
- What do you need to do to change the dynamics in your team?

A transformational agenda for 2015 and beyond

In order to not only face, but to survive the long-term trends and short-term pressures in the marketplace, private physiotherapists will need to transform the way they run their practices. Private physiotherapists run a people business – treating people and managing people and, as a higher-fee alternative to the NHS, must provide a deeper level of connection, not just a shorter waiting time.

Reconnecting with the whole point of what you do and living that purpose with passion and drive means always knowing:

- why you are doing this and why your practice exists
- who you want to do it for, including whether you want to work with PMIs
- what makes your clinic profitable and therefore sustainable
- what model you want to operate in your clinic.

All the answers must be towards transforming the wellbeing of your patients, not just transacting with them. After all, there are easier ways to make money, so it can't be all about the money!

About the author

James Butler is Director of Painless Practice, the leading firm of business coaches for therapists in the UK, providing assistance to physiotherapists who want to develop their practice to meet current challenges through one-to-one support, CPD training and published materials. James co-authored the *Seven Pillars of a Painless Practice* and two other books on running clinics. He and his team deliver practice-building CPD and speak widely on practice building techniques.

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Thursday 3 September 2015

Tutor .....Margaret Rees MSc MCSP FSOM
Duration ..One day
Content ...Theory & Practical
Cost.....£145 member / £165 non-member
.....online discount price: £135 member / £155 non-member
Venue:.... (Lombard Physiotherapy Clinic), West Moor Clinic,
..... 126 Great Lime Road, Newcastle upon Tyne NE12 6RU

The course is designed to refresh and develop functional and surface anatomy skills to enhance assessment, clinical reasoning, diagnosis, treatment and rehabilitation. By improving functional anatomy skills, participants will enjoy increased assessment, diagnostic and clinical reasoning skills, leading to more accurate and specific diagnosis.

"Excellent course which exceeded my course expectations. Inspiring Tutor."

Introduction to Assessing Fitness for Return to Work

Thursday 10 September 2015

Tutor .....Glyn Smyth MSc (Ergs) MCSP
Duration .. One day
Content ...Theory / Procedural
Cost.....£145 member/ £165 non-member
.....online discount price: £135 member / £155 non-member
Venue.....Haverhill Physiotherapy Clinic, Anne of Cleves House,
.....Hamlet Road, Haverhill Suffolk CB9 8EE

Glyn will be returning to our Central Education Programme for another year of valuable one day lectures. Glyn qualified as a Chartered Physiotherapist in 1987 and is a Registered Ergonomist having gained a Masters degree in ergonomics in 1994. With substantial clinical experience in managing work-related injuries and occupational health issues, Glyn has a wealth of practical knowledge, ensuring you will receive the best training in this field.

"An interesting, well presented course."

Avoid disappointment and book straight away to secure your place! www.physiofirst.org.uk or call 01604 684968

Physio First

2015 Conference report

Weekend showcases the latest in research and techniques

The East Midlands Conference Centre was once again the venue for our Physio First Conference. This year our theme was Soft Tissue. The Hard Truths!

Lecturers, both international and home-grown, met to provide "evidence" of the advances in clinical reasoning and optimal techniques for assessment and treatment to our delegates. The content for the weekend showcased the amazing research developments in our chosen profession and highlighted what makes physiotherapy such a great career choice.

Our Education Day kicked off the weekend's activities, followed by our Friday night supper – an informal and relaxing event aimed at launching Conference and offering the chance for those attending to meet the trade exhibitors who, this year, included both familiar faces and some new ones who were presenting innovative and exciting new products.

On Saturday morning, Amanda Marsh, our new Conference Chair, welcomed nearly 400 delegates to our Conference and our lectures programme began with Professor Jill Cook presenting the Kenneth Balfour lecture in which she educated delegates on how tendons become pathological, how they respond to load and, therefore, how to identify correct loading strategies accordingly.

The Olive Sands Memorial lecture was then delivered by Professor Tim Watson, whose entertaining presentation on electro physical agents and tissue repair, that aimed to make electrotherapy "sexy again!" was enlightening and warmly received by our delegates with regard to their clinical thinking in the use of EPAs alongside other manual therapies.

A Conference first this year was the decision to invite James Butler to deliver a business lecture. His talk allowed business owners and their staff to analyse the practice they work in / own and how to determine where and how they want to develop it. He encouraged delegates to plan

a vision of their practice and explained how to implement steps in getting started.

Lyn Watson, our second speaker from Australia, provided our first PPEF lecture. Her content was on multidirectional instability with discussion about how to understand the different types and factors that contribute to this complex condition, and up-to-date evidence that provided delegates with the knowledge of how to treat patients effectively.

Our final lecture on Saturday was given by Mr Sam Singh, a well-published orthopaedic foot and ankle surgeon from London. His easy style entertained a tiring crowd after such a long day and guided us on post ankle sprain diagnosis, with clear video footage of assessment tools and clinical reasoning skills.

Sandy Lewis presided over her last Annual General Meeting and was rewarded with a standing ovation from the attendees, which she so richly deserved, having served tirelessly for four years as Chairman, progressing Physio First along its path of

"championing evidence based cost effective private physiotherapy with Physio First members in the changing healthcare marketplace." Our new Chairman, Pam Simpson, was warmly welcomed alongside all the other new executive and non-executive members to the team.

After a long, but factually enlightening day we retired to the bar at the Orchard Hotel, enjoyed an excellent buffet supper and some amazing live music from the Bad Girls Groove Band. There were even some hard-core party animals who made it to the early hours of the morning.

Sunday morning was an early start (for some!) and began with Lyn Watson presenting the second PPEF lecture in which she presented on evidence and exercise selection for shoulder rehabilitation. We learned about scapula and humeral head position to help embark on appropriate shoulder exercise programmes.

Tim Beames then followed with his fascinating talk on altered body perception and pain, which included understanding the multisensory nature of pain and how it manifests clinically through peoples' differing perspectives – ideas we were allowed to explore further during his lecture.

Professor Deborah Falla flew in from Germany to present her lecture on the reorganised sensorimotor control in neck pain and the benefits of training. She reviewed evidence of the adaptations in people with neck pain disorders and how to identify patients who would benefit and respond to motor control intervention.

James Earls, writer, lecturer and bodyworker specialising in myofascial release and structural integration, presented a talk that educated delegates on how to apply myofascial chains, and their implications for normal movement. James helped us to understand the different roles of the stretch shortening cycle and of Anatomy Trains within the field of physiotherapy.

Our final presentation of the weekend was the second Kenneth Balfour sponsored lecture, in which Professor Jill Cook reviewed the most up-to-date evidence for funky treatment of tendons, enabling delegates to understand interventions in tendon rehabilitation, and identify how loading affects it.

This completed our line-up of such relevant, evidence based lectures that enabled all our delegates to return to their clinics with new nuggets of information and techniques to use with their patients.

As a member of the Conference sub-committee I was pleased to receive so many positive comments about the weekend. Thank you to all involved in the huge task of preparing and running another successful Conference and, of course, to all who attended. We look forward to seeing you again, and hopefully more of our members, for our 2016 Conference when we will be proud to be "Championing Sport in Private Practice".

Sarah Beldon
Conference sub-committee member

Conference competition winners...

Congratulations to those delegates who won prizes generously offered in the various competitions run by our trade exhibitors

Algeos Ltd
Laura Gardner-Wedge
A place at the 2016 Physio First Conference
Clare Spafford
An Easter egg and a bottle of wine

Blue Zinc/TM2
Robert Bailey
One free TM2 Licence worth £1200

Barrier Healthcare
Suzy Cramb
£50 M&S voucher

C&P Medical
Judith Pitt-Brooke
A bottle of champagne

Canonbury
Lin Connor
£250 to spend with Canonbury

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Andrea Blackshaw
An Empi Phoenix device

Online Ergonomics
Alison Hull
Hag Puls Chair
Karen Raine, Katie Knapton
Roller Mouse / Handshoe Mouse

Patterson Medical
Sarah Cox
A Red Letter Day

Phoenix Healthcare
Susan Godfrey
A bottle of Champagne

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Jo Gibson
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Physiotec
Alison Bray
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Helen O'Neill
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UK 3B Scientific
Kathryn Stephenson
Stan the full size (A10) skeleton

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Claire Morrell, Gayle Oakes
Liz Palmer, Sarah Brady
Each receive a free copy of the TakeBackControl™ System workbook

Trimbio
Pam Bruce
£50 to spend with Trimbio

Salaso Health Solutions
Michaela Forbes
One year subscription, including training and support, to Salaso exercise prescription software

Physio First Conference 2016

Championing sport in private practice

East Midlands Conference Centre, Nottingham, NG7 2RJ
16-17 April 2016

Confirmed conference speakers:

Lynn Booth MSc Sports Injury and Sports Therapy; Honorary Fellowship from the University of Central Lancashire.

Lynn led the Physical Therapy Services for the 2012 Olympics.

She has extensive experience working with Team GB and has been Head of Physiotherapy at a number of Games and was Chairman of the British Olympic Association's Physiotherapy Committee from 1992-2004.

Professor Karim Khan, MD, PhD, FASCM, is a Canadian sports physician and academic who is an advocate of physical activity for its public health benefit. Karim was also a major contributor to the paradigm shift that 'tendinopathies' are not inflammatory conditions and this led to physicians appreciating the need for active exercise as treatment – the concept of 'mechanotherapy'. He is co-author of 'Clinical Sports Medicine' Brukner & Khan.

Professor Bill Vicenzino, School of Health and Rehabilitation Sciences, University of Queensland. Bill is also Chair in Sport Physiotherapy and Director of Sport Injuries Rehabilitation and Prevention of Health (SIRPH) research unit.

And many more amazing speakers to be confirmed soon…

- For more details, contact Alice on 01604 684968 / alice.kettle@physiofirst.org.uk / www.physiofirst.org.uk

Book online and get a £10 discount!.....Bookings open 1 September 2015

Conference thank you . . .

to all those contributing to the success of our 2015 Conference either as an exhibitor or sponsor

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Thank you, Sandy

I am sure everyone involved in Physio First will join me in thanking Sandy Lewis for all her work, over the past four years, as our Chairman.

An inspirational leader and role model, Sandy has led our Organisation through a massive period of reflection, change and empowerment.

In the early part of her tenure, Sandy introduced to us our Organisational culture; the way we behave with each other as members and with our external partners, based on respect, transparency and empowerment. It also asks us all to proactively listen to one another and has led to improvements in important relationships with external organisations, as well as with all of us as members.

In 2013, Sandy led our Executive through our strategic facilitation process culminating in us unanimously agreeing our Vision and business goals for the next five years and starting us on a journey that we are now half way through. This was a very brave move and one that Sandy led with her typical calm, thoughtful and insightful style.

Thankfully, Sandy is not leaving us just yet. She has agreed to use all her knowledge and depth of understanding to assist us in the set up of our new Research Sub Committee which will examine the definition of evidence as this applies to Physio First and our marketplace. This Sub Committee will also help us, as members, to continue our data collection which is becoming an increasingly important tool in our conversations with all players in the marketplace, including the general public. She will find a way of using this data to our best advantage.

As our Chairman, Sandy has also been an important figure on the international stage and represented us at IPPTA meetings; the private practice subgroup of WCPT. Here she has shared the experiences of UK private practitioners with delegates from similar organisations worldwide and, in turn, learned much that has helped us to shape the future of private practice in the UK.

So, you will all understand that these are large shoes to fill. However, as I only have small size feet, I will take my own steps forward, but rest assured that they will be in the same direction of travel.

I have been involved with Physio First since 2005 when I first delivered the Manual Handling, Risk Assessment course. This gave me the opportunity to meet many of our members around the country. I then joined our PR Sub Committee, and then became its Chairman, a role that included a position on the Executive.

In 2011, I took on the role of Vice Chairman and have, for the past four years, worked closely with Sandy, which has been a delight and an inspiration.

Being Chairman of this wonderful group of committed people is an honour and a privilege, and with it comes a great commitment of volunteer time and energy. The post holders and all of you, our engaged members, are what make taking on this role both enjoyable and humbling. I am very excited to see where this journey will take us and encourage you all to join in and participate where you can. My inbox is always open and communication from all members will always be welcome.

Here's to the rest of the journey!

Pam Simpson
Chairman

Honorary life members

At our 2015 Conference, outgoing Chairman, Sandy Lewis, presented Karen Winrow and Margaret Rees with Honorary Life Memberships to Physio First for their extensive contribution to our Organisation at regional and national levels.

Margaret Rees

Margaret served two terms as Midlands Regional Officer from 1984 to 1990. During that period, OCPPP held a conference in spring and autumn and the location moved around the country. In 1989, there was a last minute crisis with the venue of the spring conference and Margaret played a significant part in relocating and reorganising the event. So much so that, when she finished her second term as Regional Officer, she became involved with the Conference Sub Committee and then its Chairman. In 1993, Margaret secured Lance Twomey, an Australian lecturer, as our first International speaker.

Following her term of office on our Conference Sub Committee, Margaret concentrated on her teaching commitments with the Society of Orthopaedic Medicine (SOM), later taking up the Chairmanship of SOM and teaching extensively in the UK, Spain and Italy.

In 2008, Margaret returned to her role as Regional Officer, then became Regional Officer Representative to our Executive Committee where she was instrumental in forming the

close team relationship between our Regional Officers and Executive that we share today. In that role, she led the full review of the Regional Officer's Handbook, making it electronic, thus facilitating updates more easily.

Margaret was also an influential member of our Public Relations Sub Committee with responsibility for dealing with our commercial relationships. This expert management led to her taking the Executive role of Commercial Officer, which she has worked very hard to define and develop.

Margaret's input at Executive level is thoughtful and insightful, with tremendous attention to detail. We all admire her ability to assimilate figures and statistical trends and then to explain them to the rest of us.

Karen Winrow

Karen has been a member of Physio First since 1996. In 2003, she took on the post of Regional Officer for Mercia and her next step in 2007 was to the post of Honorary Education and Research Officer. Under her expert stewardship, our Education Sub Committee has gone from strength to strength, forging a reputation for hosting courses for Physio First members that are exclusive, evidence based and uniquely tailored to the needs of the private practitioner.

Karen was directly elected to the CSP's Regulatory Board in 2005 and, as part of that work, served on the Professional Conduct Committee. She was also part of the Allied Health Professionals Research Network, contributing to the profession's development

and this helped to fuel her own second MSc through the University of Brighton where she pioneered the Private Practice module.

Her enthusiasm and generous nature are evident in everything she does, and are qualities she has brought to our Physio First Executive committee in abundance. Karen's positive nature infects us all.

Physio First working for us

Physio First for the next five years is:

"Championing evidence based cost effective private physiotherapy with Physio First members in the changing healthcare marketplace"

Physio First Data for Impact (PF-DFI) Study

In 2005 the Standardised Data Collection System was developed for private practitioners by the University of Brighton (UoB) and funded by the Private Physiotherapy Educational Foundation (PPEF).

Between 2009 and 2013, Physio First members used this system to collect data which Physio First, in partnership with the UoB and again funded by the PPEF, analysed and then published in eight snapshot reports which are now available to members on our website.

In 2013, the UoB and Physio First evolved a new, shortened Data Collection Tool (DCT) that is easier to complete than the previous versions, and will be permanently available to our members. So, having learned over the past five years "how to collect and analyse data", not only can we continue to do so, but now we can begin the process of learning how to use it.

This short form tool was piloted between March and mid-April last year and our preliminary results have been analysed. Again, the results of the pilot can be found in the members' area of our website www.physiofirst.org.uk. Following the success of this pilot, the full collection project was launched in November 2014.

The short DCT is now a permanent, online, developing bank of data that both Physio First, and in time, individual practitioners will be able to draw upon to fight our marketplace battles, "champion" private practice and back up what "we know we know"; without this data, who would listen?

So… what are you waiting for? Join the Data For Impact Study and start "Championing" NOW!

Visit www.physiofirst.org and click Data For Impact to find out more!

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Tips from the team

www.physiofirst.org.uk

Membership certificates

Thank you for renewing your membership with Physio First.

Your Physio First certificate of Membership for 1 April 2015 to 31 March 2016 was sent in the May edition of the Update Newsletter. If you did not receive this, please contact our office on 01604 684960.

If you want to find out what Physio First members are talking about then join Physio First's own private LinkedIn forum. You can do this by:

- Logging on to www.physiofirst.org.uk using your membership number and password
- Click on FAQ and then Membership and Benefits
- Find the FAQ 'How do I join the Physio First LinkedIn On-Line Community?' and follow the instructions.

Password reminder

Have you forgotten your password to log into our Physio First Website? If so click on 'forgotten your password?' on the bottom right hand corner of the log-in page.

Enter your Physio First membership number, CSP number and your surname and a request will be sent through to your preferred email address.

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