PERSISTENT PAIN IN ELITE ATHLETES

ARE WE DOING OUR BEST?

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INTRODUCTION

Although research and knowledge surrounding pain has grown exponentially over the past decade, its clinical application still lags behind[1]. It is now understood that pain can exist without tissue damage[2] and accounts for the updated definition of pain from the International Association for the Study of Pain – 'An unpleasant sensory and emotional experience, associated with, or resembling that associated with, actual or potential tissue damage'. Persistent pain is thought to be pain persisting after the natural tissue healing times[3], with the timings varying between different tissue types, and is illustrated by Figure 1.

It is proposed, therefore, that there are other factors that contribute, influence and drive the experience seen in persistent pain[4]. The possible mechanisms by which these drivers affect pain can be explained by an understanding of pain biology which, when relayed to the individual affected, helps rationalise a holistic approach to their pain. A knowledge of the possible pain drivers (see Table 1) can facilitate the clinician's identification of those relevant to their patient, which, in turn, promotes the use of appropriate interventions and formulation of a coherent, relevant management plan. The combination of and relative contribution of these factors will be unique to the individual experiencing pain and so, too, must be the management plan proposed to address them.

This article will address pain biology and how to apply this knowledge to treat an elite athlete with persistent pain. The key principles of a pain management plan will be outlined and then a real example of a professional tennis player with shoulder pain will be used to illustrate the process of elucidating drivers, treating, re-evaluating and ultimately returning the athlete to competitive play.

PAIN BIOLOGY

It is easiest to explain pain by thinking about an acute injury such as cutting your finger with a knife. As the process by which the tissue damage is conveyed to the brain, via the spinal cord, as pain is explained, let's elaborate on processes that can become involved when pain becomes persistent;

The tissue damage caused by the cut stimulates neurones called nociceptors. The primary function of these neurones is to detect danger and keep us safe. They do this by sampling the environment using sensors. These sensors, however, do not detect pain, and instead they respond to mechanical (e.g. pinch), chemical (e.g. stomach acid/ external allergens) and temperature stimuli (hot and cold). When these sensors are stimulated they will open, allowing ions to flow into the neurone. If enough ions enter the neurone it will reach its all or nothing point (Figure 2) and an impulse will be sent to the spinal cord. It is important to note that 'pain information' is not being transmitted at this point, but only that there has been a mechanical, chemical or temperature change to that tissue or that there is possible 'danger' present.

This is the first place where pain can be influenced. Processes such as activation of the sympathetic nervous system and adrenocortical axis can raise the excitability of the nociceptor. The neurone is brought closer to its 'all or nothing' threshold, allowing smaller stimuli to trigger an impulse. A number of pain drivers may affect these systems; for example lifestyle factors such as sleep deficit seen in jet lag.

The peripheral neurone meets the second order neurone in the dorsal root ganglion (DRG) found in the spinal column. This neurone is responsible for transmitting the information from the periphery to the brain and can be up or down regulated. As well as meeting the second order neurone, it meets peripheral neurones from surrounding tissues and the neurone coming down from the brain (Figure 3). At the point where the action potential reaches the second order neurone it releases neurotransmitters into the gap. These neurotransmitters have a specific configuration, represented in the diagram as a shape, and will only fit into its specific, same shape sensor using the lock and key principle. In doing so gates on the neurone travelling to the brain are opened or shut. If opened, more ions flow into the neurone making it more excitatory and likely to fire, and if shut the opposite occurs, making the neurone less excitatory and less likely to fire. Neurotransmitters therefore can be labelled as excitatory or inhibitory.

This is the second place where pain can be influenced. The information from the periphery, for example mechanical change detected, can be up or downregulated. It can even be stopped if enough inhibitory neurotransmitters are released from the neurone coming down from the brain; for example think of the rugby player with a severe injury that continues playing because they are caught up in the match.

If the danger message does get through to the brain, its 'information' is taken in context of all the other information being received, for example visual stimuli from the eyes, noxious stimuli from the nose and previous memories from hippocampus. Its job is to construct a story based on the information it is receiving. This concept is supported by research using PET scans on individuals in pain[5]. It shows not one but many areas of the brain lighting up during the pain experience. Each area is called an ignition node and is a congregation of a huge number of neurones (many more than at the DRG) with common locations being seen in areas of the brain such as the motor cortex, amygdala, hippocampus and sensory cortex. These nodes are linked both electrically and chemically; the pattern created during pain being called a neurotag (see Figure 4) and unique to each individual. Any one node can be stimulated, triggering the neurotag[6] and thus pain.

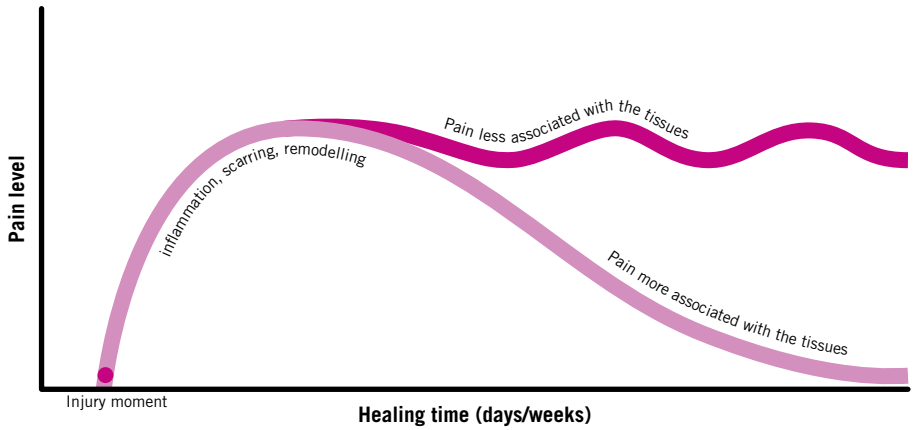

Figure 1: Reprinted with permission, Butler DS and Moseley GL: Explain Pain, 2nd Edition, Noigroup Publications, 2013.

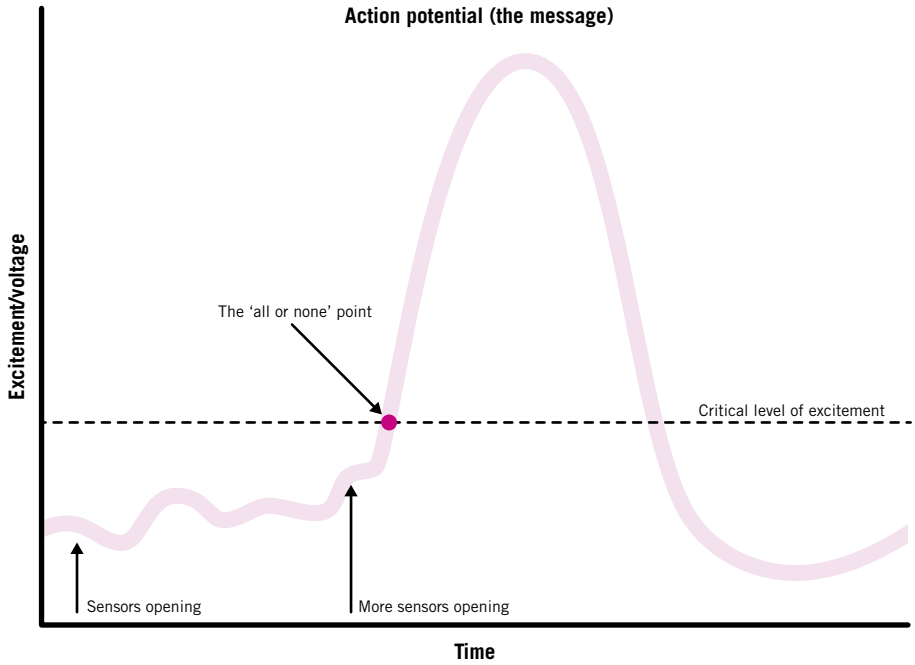

Figure 2: Reprinted with permission, Butler DS and Moseley GL: Explain Pain, 2nd Edition, Noigroup Publications, 2013.

This is where the information of change from the tissue is interpreted as pain and is the third, and final place where it can be influenced. It is where pain is most adapted, made unique and bespoke to the individual and is the target of many interventions. The multitude of ignition nodes in an individual's neurotag allows for different stimuli to activate pain and can explain the phenomenon of a soldier feeling pain in an old injury when revisiting a battleground. Here the stimuli maybe visual, auditory or even olfactory. The more the pain experience is felt, or neurotag triggered, the greater the reinforcement of the associated electrical and chemical pathways and the more easily the pain can be triggered again. This can be repeated again and again such that the patient's pain may be initiated by an ever decreasing stimuli. Think of a tennis player whose shoulder pain was initially felt towards the end of a match, who after developing chronicity feels it even when attempting a shadow serve off court.

MANAGEMENT

Recognition of persistent pain in the elite sport setting is fundamental. The culture within this environment promotes a strong belief that pain is related to injury and tissue damage. Clinicians need to be open to the concept that persistent pain can also exist in this population and address it accordingly. Management involves a thorough assessment and diagnostic work up to ensure the exclusion of 'red flag' pathology. In contrast to other settings, it is likely that the majority of persistent pain problems have started as an acute injury. Elements of the initial examination findings may still be present and are important to note so that aggravating movement/s can be reintroduced in a gradual manner. It maybe that there is still nociceptive input from the original injury, however other contributing factors (as described in Table 1) are likely to play a greater role and as a result this should be explored in detail in the assessment. The nuance to a pain management plan is to work out how much the other pain drivers are contributing and what interventions might be appropriate to address them. With this in mind, there are some key tools that can be universally applied:

1. Promotion of a strong therapeutic alliance

In simple terms getting the player to trust you. This has a number of effects; it allows them to feel safe, decreasing the sensitivity of danger system which has likely been upregulated since the onset of the injury including, but not limited to, the activation of the sympathetic nervous system and the adreno-cortical axis. In addition, if a player trusts you as a clinician, they are more likely to listen and take on board what you are recommending. This can include education around pain and injury but also suggested treatment. Management of persistent pain often asks the individual to step out of their comfort zone; for example, perform an activity they have been avoiding, and for that they need to trust the individual giving the advice.

How to build a strong therapeutic alliance is beyond the scope of this article but suffice to say communication is a vital component. This is not only important in delivering information to the patient, but also can help optimise the workings of an interdisciplinary team which are likely to be involved in managing an elite tennis player. Good communication within the team improves pain outcomes in two ways; firstly, the team understand the problem and how to manage it but also, and probably more importantly, each member of the team delivers the same message to the player. This in turn builds trust in their team and a positive feedback loop is set up where the player feels more safe, the danger system reduces further and the player is even more likely to continue down the recommended rehabilitation route.

One important component of managing persistent pain problems is the handing over of responsibility of management to the individual affected[7]. Better outcomes are seen when the individual learns to

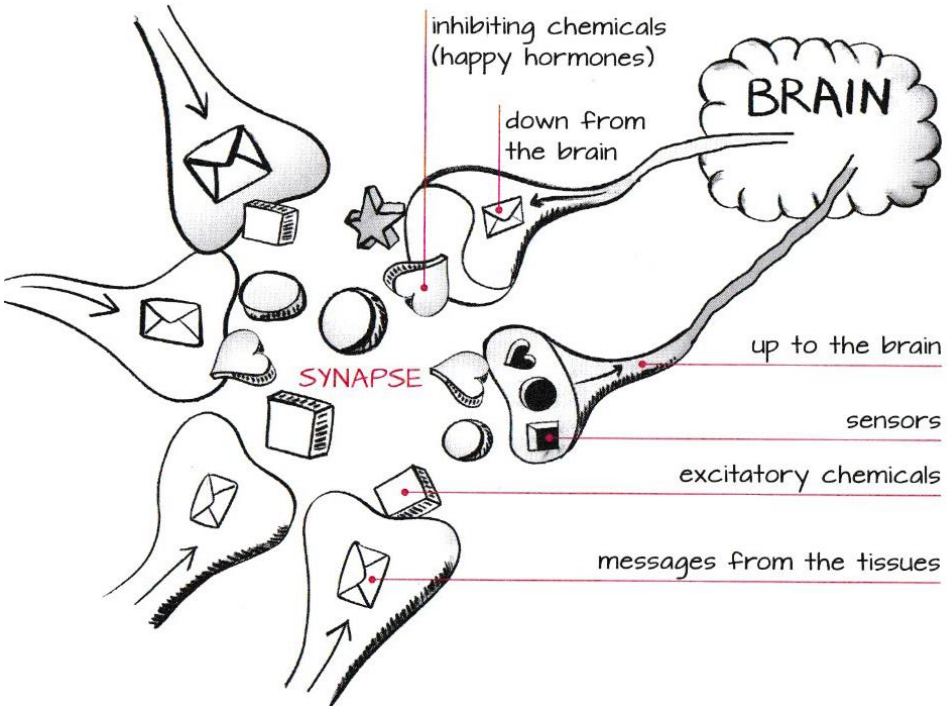

Figure 3: Reprinted with permission, Butler DS and Moseley GL: Explain Pain, 2nd Edition, Noigroup Publications, 2013.

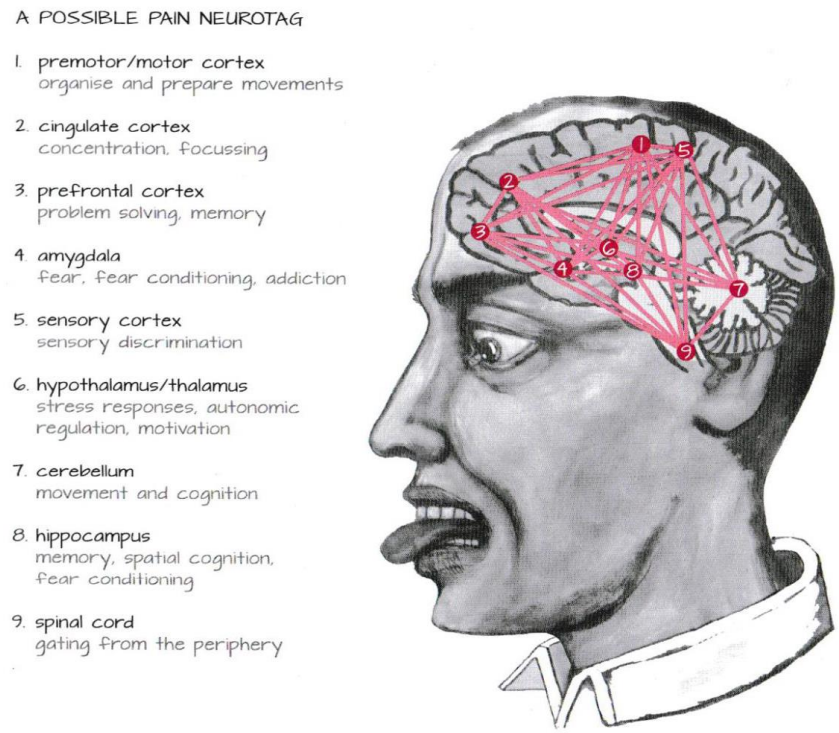

Figure 4: Reprinted with permission, Butler DS and Moseley GL: Explain Pain, 2nd Edition, Noigroup Publications, 2013.

Table 1

Contributing factors		Examples
Physical factors	*Extrinsic*	*Repetitive movements in sport e.g. tennis forehand*
	Intrinsic	*Changes to motor control e.g. holding back in extension in particular activities such as lifting*
Lifestyle factors		*Smoking, sedentary behaviour, sleep deficits*
Cognitive factors		*Fear of movement*
		Belief that pain is all structural e.g. 'discs are crumbling'
Emotional factors		*Stress, anger, anxiety and depression*
Social factors		*Work, family, coach expectations*
Genetic factors		*Pain is seen to be more prevalent in certain populations*
Individual factors		*Readiness to change, patient values, goals, acceptance.*

Table 1: Contributing factors to persistent pain[4] with examples.

self-manage, only using the clinician as a sounding board. It could be argued that this is more relevant to tennis players than other athletes, as they often spend time on the road without health care professionals and as a result have to self-manage niggles.

2. Education and knowledge acquisition

Education around pain and its management helps the patient in several ways. 'Deep learning', when information is retained and understood, allows incorporation into a person's attitudes and beliefs, which in turn promotes integration of behaviours into every-day life[8,9]. It helps the affected individual be independent and cope with similar situations in the future. The ability to self-manage improves outcomes, especially when they understand what they are doing and why.

Where education has been paired with movement approaches or an exercise programme it has been shown reduce pain further[13]. Additional benefits include an increase in strength and physical function and an improvement to quality of life [10–12]. Education is likely to reduce the threat of pain[13,14]. Understanding fundamental points, such as pain doesn't mean tissue damage, can allow patients to challenge unhelpful behaviours such as limping, or in the case of upper limb avoding simple tasks using the affected lilmb. This knowledge will reduce the activation of our protective systems such as sympathetic, immune, endocrine and motor which can directly feed back into the brain, reducing the sensitivity of the ignition nodes and potentially the perception of pain.

3. Movement and exercise

It is well recognised that exercise is an effective treatment to reduce pain and improve function in a number of pain conditions[16]. There are some uncertainties over the underlying mechanism, but it is thought to affect central processing by increasing the sensitivity of the descending inhibitory pathways and decreasing the sensitivity of the descending facilitatory pathways. It may be that this central processing change is initiated when the 'sweet spot' of exercise is achieved; just above the baseline.

The key with exercise prescription is that it stimulates and uses the affected tissues but at a level that does not cause a flare in pain. A small amount of pain is acceptable, but severe pain that takes days to settle is unhelpful as the individual will reduce their activity and decondition. In addition, it may prevent the individual participating in life, which may upregulate other inputs such as fear and anxiety. The first step, therefore, is to find the patient's baseline level. For example with persistent serve-related pain, shadowing a serve with no racket and no ball may be a good starting point (alongside the usual rehabilitation plan), or even in rare cases just going through the movement in their head.

The following case study aims to illustrate how to apply the knowledge of pain biology and its management to an injured elite tennis player with persistent shoulder pain. When formulating a treatment plan, aside from the usual criteria-based return to play plan, clinicians should consider what possible pain mechanisms could be at play and then tailor interventions to address them. As you can never be quite certain what mechanisms are involved or their relative contribution, it is important to reassess response to interventions regularly. The player knows their condition and themselves best, so involve them in the decision making as much as possible.

CASE STUDY

A 25-year old right hand dominant professional tennis player presented to the team doctor with right shoulder pain. She was upset and asked for a 'scan to see what was going on'. Due to a drop of her ranking she was no longer receiving funding from the tennis governing body, which resulted in her not receiving input from the wider multi-disciplinary team, including physiotherapy, nutrition and psychology. She had been experiencing shoulder pain for a year, which had been getting progressively worse. She reported pain in the deltoid region on increasing serving load. When this occurred she would stop competing and serving and present to her external physiotherapist to receive treatment. She would then undergo a mini training block and re-enter competition. She would find on restarting serving she would have a return of pain. As this pattern repeated over the year she found the pain started to affect her serve as well as her forehand. She had a past medical history of an eating disorder and generalised hypermobility.

A joint assessment with the doctor and physiotherapist was performed. History and clinical examination was consistent with a presentation of multidirectional instability. Imaging confirmed no coexistent tendon tears and a thickened subacromial bursa.

It was decided due to ongoing pain that her bursa would be injected, but it was clearly explained to the player that this was only the starting point, and that a criteria-based return to play (RTP) plan was needed to prepare the whole shoulder complex (and the rest of the body) for competitive tennis. She was granted permission to complete the RTP plan 'inhouse' with the multidisciplinary team.

Table 2 summarises a five-staged RTP plan. Specifically to the shoulder: stage one focused on optimising recovery from the injection while restoring pain free range of motion below shoulder level; stage two on resuming upper body strengthening below shoulder level while optimising muscle control and reintroducing backhands on court; stage three on overhead strengthening and serve preparation drills while reintroducing forehands on court; stage four on rate of force development while reintroducing and building up serving volume and intensity and finally the fifth stage reintroduced match play in a controlled environment.

The pain biology behind the RTP plan:

1. Ultrasound guided injection; The doctor injected a 'structural cause of pain' and, on retesting the provocative shoulder movement after the injection, the pain was no longer present. Possible underlying mechanisms include anaesthetising of peripheral nociceptors and reduction of the threat response secondary to increased trust in the clinician and plan.
2. Education. At each stage of the RTP plan the reasoning behind exercises and interventions was explained to the player. Key points such as 'pain does not equal tissue damage' were emphasized, and that strengthening will improve tissue tolerance to be able to meet the demands of repetitive serving.

Table 2

	Stage 1 (of 5) RECOVERY	*Stage 2 (of 5) RELOADING*	*Stage 3 (of 5) RECONDITIONING*	*Stage 4 (of 5) RETURN TO SERVING*	*Stage 5 (of 5) PRE-COMPETITION*
PURPOSE	*Post-injection recovery & low tendon reload*	*Below shoulder strengthening & forehand (FH) preparation*	*Overhead shoulder strengthening & serve preparation*	*Serve reloading Increase on court tennis time*	*Return to match practice*
PHYSIO	*Manual therapy Compex disuse atrophy Basic cuff activation & scapula setting*	*Game Ready/cryotherapy (if required) Manual therapy RC NM drills into higher ranges and different planes*	*Manual therapy for maintenance Serve preparation program RC NM drills for warm up*	*Manual therapy for maintenance Independent shoulder warm up*	*Manual therapy for maintenance*
STRENGTH & CONDITIONING	*Left -sided UBWs only Core -avoid WB via upper limb (UL) Cardio (excluding heavy running/versa) LBWs without loading right arm*	*Reintroduce Push/pull exercises (excluding OH) Core (avoid WB via UL) Cardio (excluding versa) LBWs as per normal*	*Overhead UL strengthening focus Maintenance of body conditioning*	*Shoulder rate of force development training Maintenance of body conditioning*	*Maintenance of body conditioning*
TENNIS		*Backhands (BHs) only - time on court limited*	*Build up volume of FHs*	*Build up on-court training volume to normal Return to serve protocol (build up to 520)*	*Practice matches Normal serve intensity*
END STAGE CRITERIA	*PROM Horizontal Flexion at 35deg/+ Painfree AROM Flexn & Abdn to 90deg Thoracic rotation AROM at >40deg*	*Full and painfree AROM Flexion, Abduction & ER Passive TROM shoulder rotation within 10deg different Equal isometric shoulder strength @ 0-60deg elevation Dynamic rotary stability test (DRST) normal at 0-60deg Pain free shadow FHs x10*	*No scapula dyskinesis DRST normal at 90degrees Negative empty can test Complete serve prep drills comfortably Isometric ER:IR ratio of >0.75 @90/90*	*Eccentric ER:concentric IR ratio of 0.9-1.1 Eccentric ER/BW peak force @>25%BW*	*Play 10 practice sets in 4 days*

Table 2: Return to Play plan.

Legend: UBW=upper body weights; LBW=lower body weights; AROM=active range of motion; TROM=total range of motion; RC=rotator cuff; NM=neuromuscular.

Illustration

3. Promotion of a therapeutic alliance; The player was involved in the decision-making process. She had regular input from the same doctor and physiotherapist and was seen almost daily. Her concerns were addressed at every stage. In addition regular input from the psychologist helped reinforce the same message about the pain, as well as working through other concerns in day to day life.
4. Exercise prescription; At all points she was exercising. Not always the shoulder but her body. As well as staying conditioned it is likely this exercise helped modulate central processes of reducing pain.
5. Pacing and graded exposure; Each stage was challenging the shoulder more and more through progressive strengthening work as well as tennis-specific progressions, with a goal of no flare in pain at the end of each stage. In healthy tennis players the ratio of eccentric external rotation: isometric internal rotation at 90° of abduction and 90° of external rotation ranges from 0.8-1.2, and the average eccentric peak force is at 23% relative to body weight (BW)[17,18]. Her baseline shoulder strength markers were initially much lower than those, and so higher exit criteria targets were set. And specific to serving, stage 4 looked at increasing serving load through intensity and number of serves per session. Professional female players serve an average of 96 serves per match, and so the goal was to build up to at least 520 serves in a six-day training week, to prepare her for up to five singles matches during tournaments while lowering the risk of significant spikes in serving load.
6. Considering the context and the brain in pain; Stage 5 of the rehabilitation plan incorporated two three-set matches in a non-tournament setting. This emulation of the competitive environment, as well as the promotion of a therapeutic alliance and education to reduce the threat response, addresses several central mechanisms.
7. The player was treated holistically; She saw a psychologist and nutritionist to address her eating disorder. This condition may have put her in a pro-inflammatory state, making her nerves closer to the all or nothing threshold. By addressing her nutritional intake and the drivers contributing to her restriction her threshold will have been reduced.

Outcome: The player returned to competition after four months with no recurrence of shoulder injury.

SUMMARY

As clinicians working in the sports medicine field and particularly in the elite setting, we don't often consider the mechanisms at play behind the persistent pain an athlete experiences when injured, which in turn can affect their outcome. By understanding the biology behind pain clinicians can incorporate key principles into their management thus enabling more holistic and whole person care.

When formulating a treatment plan, aside from the usual criteria-based return to play plan, clinicians should consider what possible pain mechanisms could be at play and then tailor interventions to address them.

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