Back to the spine

InTouch

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Editorial

Critical thinking is at the core of our development, both as a profession and as professionals. Ideas and concepts need to be critically evaluated to ascertain their validity and relevance.

Closely linked to this process is an improvement in the understanding of a topic or a broadening of knowledge, with the usual result of an improvement in real-world application or an adjustment of clinical practice accordingly. This demonstrates why the concept and implementation of critical thinking is so central to us as physiotherapists, and to our practices.

Critical thinking is also the linchpin to improving the quality of our physiotherapy service, something that is certainly one of my drivers as a clinician. Collecting data and understanding our patient goals and outcomes is central to our Physio First ethos, and our Data for Impact (DfI) is an exclusive Physio First benefit that we can all access to help us achieve this.

The main focus of the high-quality articles in this issue is to illuminate different angles on the topic of the spine. In addition, we continue with our aim to underpin our articles with reviews from members who have achieved Physio First Quality Assured Practitioner (QAP) status, and hope this adds an insight into how *In Touch* can help inform your practice and improve your outcomes. Learning from our peers is a well-documented method of enhancing and improving our skills, so I am very excited by the inclusion of the QAP reviews.

As the UK starts to open up from the Covid-19 restrictions, I hope we can reflect on accomplishments of the past 18 months and move forward positively both personally and professionally.

My sincerest thanks are extended to the authors and reviewers of this edition of *In Touch* who have given up their valuable time to share their expertise with us.

Until next time.

TOBIAS BREMER | EDITOR

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Editorial Panel

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Is it a bird, is it a plane and does it matter? STOPS, STarTs and the case for individualising treatment

HOWARD M TURNER BSc BAppSc (PHTY) MCSP

Practice Principal and Clinical Lead, Wilmslow Physiotherapy

The STarT Back and Specific Treatment of Problems of the Spine (STOPS) back pain trials both evaluated the effect of individualising treatment to the specific features of each patient's presentation. The STarT Back trial did this based on psychosocial prognostic factors thought to predict long-term back pain, and the screening tool derived from that has been incorporated into the current NICE guidelines on the management of back pain. The STOPS trial is less well known and unique in that treatment was tailored to both specific psychosocial factors and clinically familiar pathoanatomical factors. Both studies had good results in terms of outcome.

The STOPS trial deserves closer attention and is the focus of this article because it provides, for the first time, evidence that a comprehensive and pragmatic application of pathoanatomical specific treatment is of value to back pain patients. Perhaps, more importantly, its results also challenge some long-standing assumptions about the importance of psychosocial factors in predicting chronic low back pain.

LEARNING OUTCOMES
TO SUPPORT PHYSIO FIRST QAP

1 Support your interventions with a more substantial literature base.
2 Improve clinical reasoning and interventional pragmatism in keeping with research findings.
3 Improve the accuracy of dialogue with patients about the likely course of their back pain.
4 Improve understanding of the more controversial approaches to back pain treatment.

More than ever before in my experience, the current guidelines for the management of low back pain have had a dramatic impact on the way physiotherapists conceptualise and treat patients with non-specific low back pain. The increased understanding of psychosocial factors in pain, and particularly in chronic pain, that has developed over the past few decades, formalised with the adoption of the STarT Back protocol (**startback.hfac.keele.ac.uk**) within the current NICE guidelines (2017), has placed consideration of psychosocial factors at the heart of treatment of back pain. This is undoubtedly a good thing. Indicators such as fear / pain avoidance, depression, job dissatisfaction and ongoing litigation appear to have a negative effect on progress and prognosis, so programmes that address these factors should help (Besen *et al* 2015; Deyo *et al* 1998). As a relatively recent target for research, there is as yet sparse evidence of effect, but a Cochrane review of the behavioural treatments concluded that there may be some short-term benefit (Henschke *et al* 2010).

The question is whether enthusiasm for this approach has outpaced its validation at the expense of "traditional" approaches. As Buchbinder *et al* (2020) comment that "…intensive pain and neuroscience education is popular, particularly among physiotherapists," but a recent high-quality trial showed that it was no more effective than sham education in patients already receiving standard first-line care (advice to stay active, avoid bed rest, option of spinal manipulation, and / or simple analgesics) (Traeger *et al* 2019). An increased awareness of psychosocial factors should give us an enhanced understanding of the complex multifactorial nature of back pain which should improve our ability to tailor treatment specifically and pragmatically to an individual's specific barriers to improvement. However, it is a concerning tendency of many researchers, clinicians

> *"AN INCREASED AWARENESS OF PSYCHOSOCIAL FACTORS IN THE COMPLEX MULTIFACTORIAL NATURE OF BACK PAIN SHOULD IMPROVE OUR ABILITY TO TAILOR TREATMENT TO THE INDIVIDUAL"*

and commentators to reject the traditional skill and knowledge base of our profession in the promotion of a purely psychosocial model of back pain (Monie *et al* 2016; Hancock *et al* 2011). This is despite a systematic review of chronic low back pain research evaluating the influence of psychosocial factors on outcome to be less than 25% (Wessels *et al* 2006).

One of the casualties in this rejection of established and recognised practice is the consideration, differentiation and treatment of anatomical sources of pain (Monie *et al* 2016; Moore & Jull 2000). For various reasons, we are told that the consideration of pathoanatomical sources of pain is unnecessary or unhelpful. Clinical guidelines do not recommend classification or specific management based on pathoanatomical principles apart from the exclusion of red flags (Ford & Hahne 2013). Guidelines from the CSP make no mention of pathoanatomical differentiation, and those from the American Physical Therapy Association (2001) explicitly state that it is neither appropriate nor useful. Some commentators go further, hypothesising that providing a low back pain patient with a pathoanatomical diagnosis may be counterproductive as it reinforces an excessive "somatic focus" (Deyo *et al* 2009; Fourney *et al* 2011; Nicholas & George 2011).

In the same way treatment in clinical practice associated with such specific diagnoses, in particular manual therapy, is criticised as being incompatible with a biopsychosocial framework to practice. There are fears of the same excessive "somatic focus", as well as concerns that such treatments encourage dependence upon the treating therapist. Studies that have empirically tested this found no evidence of such adverse outcomes (Ash *et al* 2008; Kleinstuck *et al* 2006), but it seems a common view among physiotherapists.

One quite reasonable argument for rejecting a pathoanatomical basis for treatment is that research has failed to show that it improves outcome (van Dillen

SUBGROUP NAME	SUBGROUPING CRITERIA
Disc herniation with associated radiculopathy (n=54)	Referred leg symptoms, at least one clinical examination sign suggestive of radiculopathy (positive straight leg raise or reduced lower limb reflexes, sensation or strength), and CT or MRI demonstrating a comparable disc herniation
Reducible discogenic pain (n=78)	At least four out of nine clinical features indicative of discogenic pain and a directional preference in response to repeated or sustained movements/ postures (mechanical loading strategies)
Non-reducible discogenic pain (n=96)	At least four out of nine clinical features indicative of discogenic pain and an absence of a directional preference in response to mechanical loading strategies
Zygapophyseal joint pain (n=64)	At least three of the following features: presence of unilateral low back pain, pain reproduction with extension and ipsilateral lateral flexion, localised pain on ipsilateral passive posteroanterior pressure to the transverse process or zgapophyseal joint, and improvement in pain and/ or movement following a one-min trial of manual therapy directed at the zgapophyseal joint
Multifactorial persistent pain (n=8)	Absence of membership in one of the above pathoanatomical subgroups and an Örebro Musculoskeletal Pain Questionnaire score of greater than 105/210

TABLE 1: Subgroup definitions in the STOPS trial

et al 2003; Chou *et al* 2007), although this might be a problem with the research rather than with the concept. The frustrating thing about back pain research is that while many treatments have been shown to be better than placebo, comparisons between treatments rarely demonstrate clinically meaningful differences (van Tulder *et al* 2006; Chou *et al* 2009; Keller *et al* 2007), implying that it doesn't matter what is done to the patient. One possible explanation for this is that studies have not adequately accounted for participant heterogeneity. Anyone working with back pain knows that patients present and respond very individually, even within the parameters of our ability to assess such things in the clinic. Given the multidimensional heterogeneity of back pain, it is an extraordinarily complex task to research individualised treatment packages, which may go some way to explain the less-than-stunning results of low back pain research. The STarT Back trial (Hill *et al* 2011) and the more recent Sciatica Outcomes in Primary Care (SCOPiC) research into sciatica management (Konstantinou *et al* 2020) are recent attempts to study subgrouped treatment, in these cases subgrouped on predictors of prognosis. To date, the STOPS trial is the only study that has attempted to do this on the basis of a pathoanatomical classification of low back pain (Hahne *et al* 2011; Ford *et al* 2016).

The STOPS trial

This study was conducted at 16 primary care physiotherapy practices in Melbourne, Australia and involved 300 participants with low back and side / or referred leg pain with symptom duration of between six weeks and six months. In order to be included in the trial, participants had to report a level of pain greater than two on a 0-10 numerical rating scale.

The aim of the STOPS trial was to investigate the effectiveness of clinically familiar and commonly used methods to subgroup and treat low back pain patients. Criteria for subgroup identification were developed and treatment protocols created that aimed to be consistent with commonly used methods and models. Where possible, these were based on evidence that was able to be accurately reproduced and generalised to a broad patient population. The five subgroups were:
- Disc herniation with associated radiculopathy
- Reducible discogenic pain
- Non-reducible discogenic pain
- Zygapophyseal joint pain
- Multifactorial persistent pain (Ford *et al* 2016).

The defining diagnostic criteria for each group is shown in table 1. ▶

The prescribed intervention was specific to the pathoanatomical or other classification of the group. However, all groups were also given two 30-minute sessions of advice and information as promoted by clinical guidelines for low back pain, e.g. NICE guidelines. The content was found to be of benefit in earlier research (Indahl *et al* 1995) and included *"a pathological explanation of the participant's pain, reassurance regarding the generally favourable prognosis of their condition, advice to remain active and instruction regarding correct lifting technique"*. The control (advice) group received only these sessions of information, therefore any difference in the outcomes of the groups can be attributed to the effect of the specific intervention.

It is worth remembering that two previous UK studies undertook a similar comparison to the one performed in the STOPS trial, i.e. comparing a physiotherapy treatment package with a simple package of education, and found no benefit in adding physiotherapy treatment to education. Hay *et al* (2005) compared psychosocial pain education with the same education plus physiotherapy treatment that included manual therapy with stabilisation and strengthening exercises at the discretion of the treating physiotherapists. The study involved 402 patients and no difference was found between groups on any measure at three and 12 months follow-up. The low-risk group in the STarT Back trial compared similar interventions and resulted in the same lack of difference at follow-up (Hill *et al* 2011).

The active treatment group in STOPS received 10 sessions of physiotherapy that included manual therapy, directional preference management, postural re-education, motor control training and graded functional exercise. The intervention was tailored to the patient presentation. Techniques aimed to reproduce normal and familiar clinical practice, i.e. if repeated movements caused a centralisation of pain, that directional preference was used as a

treatment technique in line with the McKenzie approach, or if there appeared to be a compressive mechanism of pain implying a zygapophyseal origin, targeted mobilisation and manipulation was used as the first line of treatment.

Patients in the disc herniation with radiculopathy group, and in the non-reducible discogenic group received a graded functional exercise programme modified for the presence of discogenic pathology with a focus on specific motor control training targeting the local stabilising muscles. The multi-factorial persistent pain subgroup received graded functional exercises with a focus on neurophysiological and psychosocial factors comprising:
- education in relation to the neurophysiology of pain
- progressive exercises
- goal setting
- cognitive restructuring and behavioural strategies targeting key psychosocial barriers.

The primary interventions for each group were compulsory but the physiotherapists could add other optional treatment modalities at their discretion. For example, in the zygapophyseal group mobilisation and manipulation of comparable segments along with education were compulsory and optional elements included such things as sleep and relaxation strategies, discussion about psychosocial barriers to improvement, pain management

strategies and posture and ergonomic advice. Training was provided to maintain consistency of delivery.

RESULTS

Individualised physiotherapy resulted in significantly better outcomes than education on the Oswestry disability scale (figure 1). It also resulted in better outcomes on back and leg pain at 5, 10 and 26 weeks (Ford *et al* 2016). Mean differences between groups were statistically significant in 71% of the primary and secondary outcomes measured in the trial. Participants receiving individualised physiotherapy achieved the same reduction in pain rating in between five to eight weeks that those receiving advice only took 12 months to achieve (Ford *et al* 2019). Further, despite the increased costs of providing 10 sessions of physiotherapy compared to two sessions of education, overall healthcare costs were found to be similar due to factors such as the additional healthcare costs required for the advice group, and lower incidence of work absence in the active treatment group (Hahne *et al* 2017a).

Participants receiving individualised physiotherapy had between 1.8-1.6 times the chance of improving by at least 50% baseline on back and leg pain respectively, and 1.5 times the chance of improving by 50% baseline on the Oswestry disability questionnaire. Those receiving individual physiotherapy had
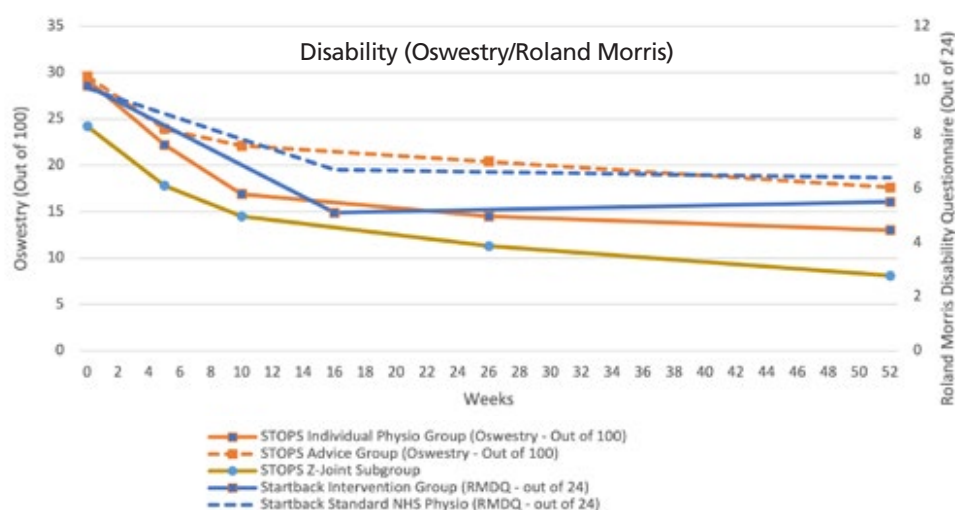

FIGURE 1: Improvement in disability for the STOPS trial (Oswestry disability scale) and the STarT Back trial (Roland Morris disability questionnaire)
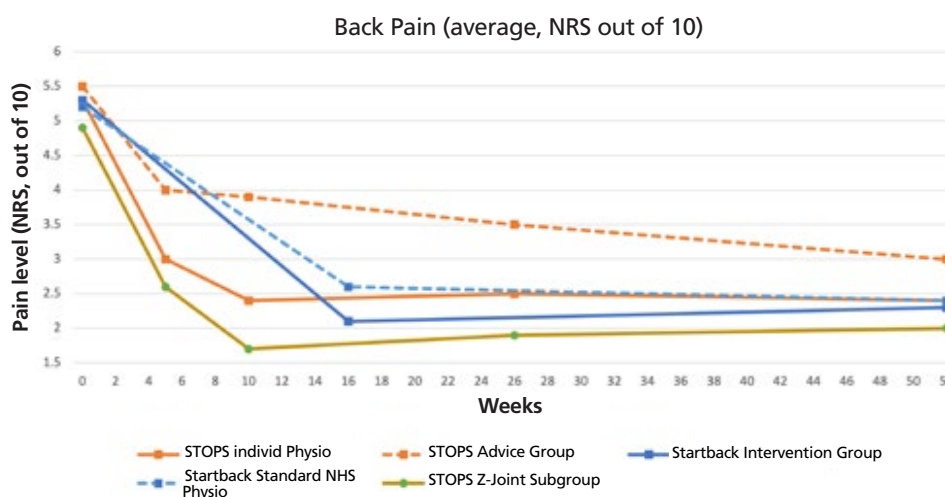
Back Pain (average, NRS out of 10)

FIGURE 2: **Pain drop in the back pain score for both the STOPS and STarT Back intervention groups**

between 1.3-4.1 times the chance of achieving a clinically important change in their condition (Ford *et al* 2019a).

Comparison with the STarT Back trial

Given the STarT Back protocol's central place in the UK's NICE guidelines on back pain, it seems relevant to compare the results to the STOPS trial. The premise of the STarT Back trial was in many ways similar to STOPS with patients being subgrouped and receiving individualised or "stratified" care. In the STarT Back trial, the "stratification" of care was based on a questionnaire of physical and psychological factors shown to affect prognosis. The focus was to tailor more psychological aspects of care to those scoring highly on a psychological subscale. This stratification of care and the addition of psychologically informed physiotherapy gave cost savings and better outcomes in terms of

disability when compared to standard UK NHS physiotherapy care. When the intervention was repeated in a US healthcare setting, however, it was found to be "a resource-intensive intervention" that had "no effect on patient outcomes or healthcare use" (Cherkin *et al* 2018).

The STOPS trial applied a subgrouping strategy that included physical, psychological and pathoanatomical factors.

In comparing the STOPS and STarT Back trials it is important to note that STarT Back patients were an average of five years older, and more of them presented with long-term pain, i.e. 46% of patients had a symptom duration beyond the six-month upper limit of the STOPS trial. In both trials there was a significant and early reduction in disability in the intervention group that lasted to 12-month follow-up, but differences in

measures preclude direct comparison. There was also an average pain drop from 5-6/10 to around 2/10 in the intervention group for both trials (figure 2). On the measured global rating of change and patient satisfaction, 71% of participants considered themselves "very satisfied" with their physiotherapy care in the STOPS trial at the 10-12 week mark compared with 40% of the intervention group in the STarT Back trial. Global rating of change of "much improved" or "completely recovered" was also reported by 71% of those participating in the STOPS trial versus the 40% in the STarT Back trial (figure 3).

The control groups of both trials also improved over the 12-month follow-up period with comparable improvements in pain and disability, but the two education sessions in STOPS outperformed in global change (53% better and above versus 35% respectively), the four treatment sessions (average) of standard UK NHS physiotherapy care, were used as the control in the STarT Back trial. There may, of course, be cultural explanations for this difference (figure 4).

One of the celebrated outcomes of the STarT Back trial was the cost benefits of the approach, when overall healthcare costs, including the cost of medication, medical appointments and interventions, are considered. Average savings of approximately £34 / US$48 (12.5%) per patient per year accrued mostly on the basis that people in the low-risk category were offered one session of education and advice and no physiotherapy treatment. In the STOPS trial, 10 sessions of physiotherapy, a lot in a UK treatment context, were provided. Nevertheless, similarly calculated overall healthcare costs over a 12-month period showed only a 3% increase in the average cost of £23 / US$30 per patient for the intervention group compared with the overall healthcare costs for the control group patients.

Challenging concepts of prognosis and back pain

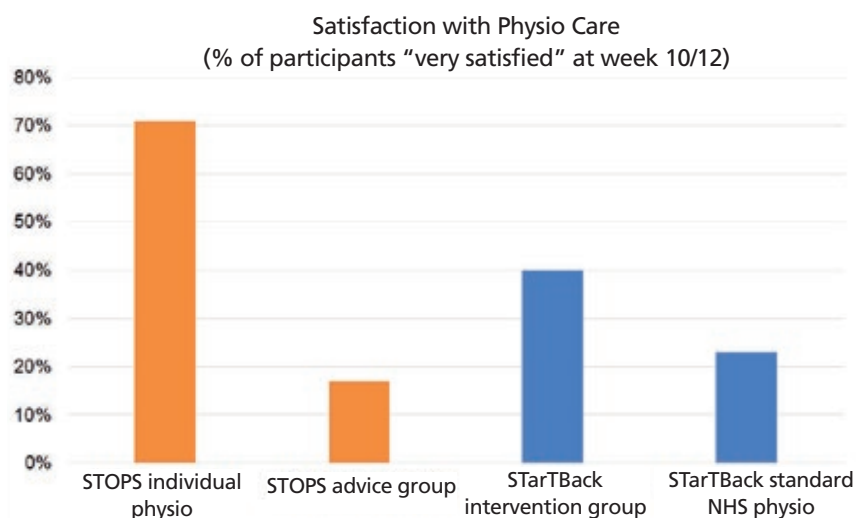As some people respond to treatment and some do not; some people get better ⊙

Satisfaction with Physio Care
(% of participants "very satisfied" at week 10/12)

FIGURE 3: **Satisfaction scale of physiotherapy intervention**

and some go on to develop chronic pain, the prognosis of an individual presenting with back pain has been the focus of much research attention and is the basis of the STarT Back protocol. Patients who scored highly on a subscore of psychosocial factors were considered at high risk of chronicity (Hill *et al* 2008, 2010a, 2010b) and it was hoped that tailoring psychologically informed physiotherapy to this high-risk group might improve outcome and provide cost savings. In fact, the high-risk intervention group did show improvements in disability at four-month follow-up but it was not maintained at 12 months in comparison to the standard NHS physiotherapy group. There were, however, improvements in the intervention groups at 12 months in work attendance and scores for anxiety, depression and fear-avoidance.

The STOPS trial adds to this knowledge base and challenges some long-standing assumptions. Looking at indicators of good and bad responses in this intervention group, the influence of a comprehensive range of biomedical, including pathoanatomical, psychological and social prognostic factors were evaluated (Ford *et al* 2018), and it was interesting that the participants who gained the greatest benefit from individualised physiotherapy were exactly those with features that are generally considered to indicate a high risk of chronicity,

> *"*THE PARTICIPANTS WHO GAINED THE GREATEST BENEFIT FROM INDIVIDUALISED PHYSIOTHERAPY WERE EXACTLY THOSE WITH FEATURES THAT ARE GENERALLY CONSIDERED TO HAVE A HIGH RISK OF CHRONICITY*"*

i.e. higher back pain intensity, high disability scores and a longer duration of symptoms. Further, other features normally considered to be associated with poor outcomes such as counterproductive beliefs, fear-avoidance, low expectations for recovery, work factors, age, psychological stress and depression, and poor general health, were not independently prognostic of poorer outcomes in their study. Nine of the 15 proven prognostic factors were pathoanatomical in nature, with only six psychosocial factors. The implication is, therefore, that if specific and comprehensive treatment is provided, these commonly assumed negative factors may not be as important to outcome as has been assumed.

Conclusion

The STOPS trial adds to the work of the STarT Back trial in attempting to tailor care specifically to subgroups of patients. While the STarT Back process involves the evaluation of physical and psychosocial factors, STOPS also considered pathoanatomical ones and shows that pathoanatomic specific care

results in significantly better outcomes than two sessions of guideline based advice and education for disability at 10, 26 and 52 weeks, and for pain at five, 10 and 26 weeks. In contrast, similar previous UK studies comparing standard NHS physiotherapy care to guideline based care showed no difference in outcome (Hill *et al* 2011; Hay *et al* 2005).

Pathoanatomical diagnosis has been criticised as being unnecessary, unhelpful or counterproductive. The treatments associated in clinical practice with pathoanatomical diagnosis, such as manual therapy, have been criticised as being incompatible with efforts to encourage self-efficacy in patients because they are seen as "passive" and encouraging dependence upon the treating therapist. These appear to be widespread beliefs that encourage a reductionist view of pain, considering only psychosocial factors of management and seeing intervention through the lens only of its psychological impact. The inherent rejection of traditional skills and practices inevitably leads to deskilling in the fundamental practice of physiotherapy (Monie *et al* 2016; Moore & Jull 2000; Hancock *et al* 2011), whereas we should surely be embracing this important knowledge of the biopsychosocial nature of pain into our normal practice to complement rather than replace our existing skill-base. There is little evidence that considering pathoanatomy has adverse effects (Ford & Hahne 2013) and the STOPS trial subgroup that received the most "manual" of therapies, e.g. the zygapophyseal group received mobilisation and manipulation as core interventions, responded significantly well both in terms of pain and disability, and in activity levels and their degree of psychosocial distress, with no indication of reduced self-efficacy
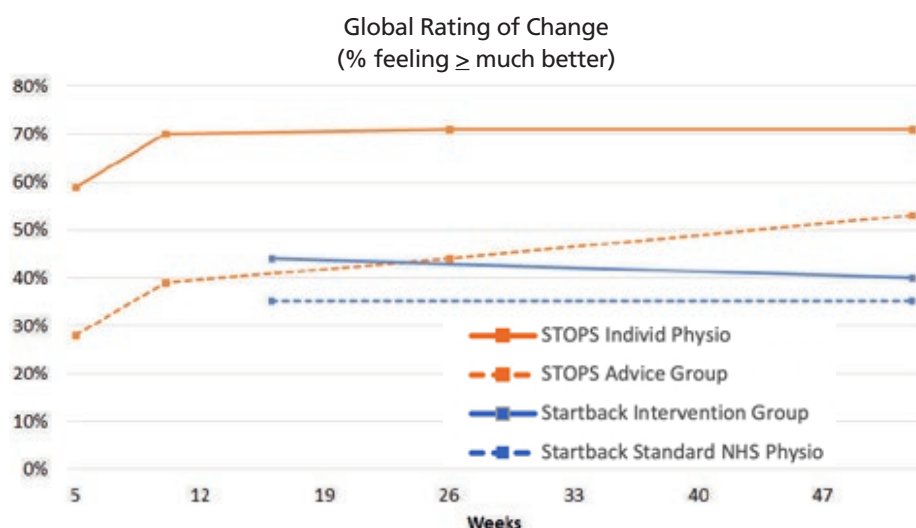

FIGURE 4: Global rate of change improvement for all patient groups

(Ford *et al* 2019b). The STOPS trial encourages us, therefore, not to discount pathoanatomical considerations.

Perhaps one of the most interesting outcomes of the STOPS trial is its analysis of prognostic factors. The results challenge the conventional wisdom, finding that only six of the 15 factors proven to be prognostic were psychosocial in nature, with the rest being of a pathoanatomical nature. The provision of specific and comprehensive care may eliminate the prognostic influence of such things as low expectations, fear-avoidance and depression, among others.

Pain is complex and, as always, more research is needed and with a depth and breadth not yet seen in order to establish subgroup differences. Nevertheless, both the STarT Back and STOPS trials add to evidence that superior outcomes may be achievable with a pragmatic, individualised, comprehensively multimodal treatment approach.

💬 QAP REVIEW

This is a really interesting discussion with detailed evidence about treatment modalities and outcomes in patients presenting with low back pain; a condition that is in the highest percentage of patients seen in private physiotherapy practices as evidenced by the data inputted in Data for Impact (DfI).

It reinforces the clinical reasoning behind the use of manual therapy techniques and why it is a modality that shows in the data as leading to good outcomes for patients, something that is against the conventional wisdom of avoiding hands-on techniques in favour of activity and exercise based intervention. The modalities talked about in this article are all relevant in the treatment of low back pain and would assist any MSK practitioner in working towards and achieving QAP status using the strategies outlined from evidence-based research.

Reviewer
Amanda Marsh

CONTACT DETAILS
hmt@wilmslow-physio.co.uk

About the author

Howard Turner is a private practitioner in Wilmslow, Cheshire, a part-time teaching fellow at Bath University. He has taught extensively on shoulder, knee, back, hip and groin pain, disorders of the pelvis, and clinical reasoning. He is a reviewer for Physiotherapy in Sport. Howard has worked for various sporting bodies and teams. He holds a physics degree from Melbourne University and a physiotherapy degree from La Trobe University in Melbourne.

Additional resources

https://startback.hfac.keele.ac.uk/ - STarT Back information on evidence-based implementation of stratified care

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Sporting spine

LIBA SHEERAN PhD MSc BSc (Hons) Physiotherapy MCSP HCPC FHEA

Consultant Physiotherapist, Welsh Athletics; Reader in Physiotherapy, Cardiff University

Low back pain (LBP) is a common complaint that significantly impacts training, competition and sport performance development. In many athletes LBP can evolve into a persistent problem associated with fear and anxiety related to training, poor training practices and the ongoing long-term demands of medical and therapeutic interventions. For some, LBP alone or associated with spinal injury can become career ending. This article aims to demystify LBP in people participating in sport and exercise. It provides a review of its prevalence and common causes, together with research-based clinical diagnostic criteria. A summary on modifiable and non-modifiable risk factors associated with LBP and spinal injury in athletes, followed by a review of evidence behind some of the common mitigating strategies used clinically with recommendation for practice is included.

LEARNING OUTCOMES
TO SUPPORT PHYSIO FIRST QAP

1 Understand the effects of low back pain on people participating in sport and exercise.
2 Be aware of the prevalence and common causes of sport-related LBP.
3 Be aware of the risk factors associated with LBP and spinal injury.
4 Understand common mitigating strategies for use in clinical practice.

Epidemiology

For many individuals, low back pain (LBP) is predominantly a self-limiting symptom rather than a diagnosis. In athletes, life-time prevalence of LBP varies between 18%–65% with some sports more affected than others (Trompeter *et al* 2017). There are so-called "high-risk" sports in which participation is associated with higher rates of LBP compared to the age-matched general population. Athletes participating in activities such as rowing, cross-country skiing, dancing, fencing, gymnastics, and track and field events appear to be more commonly affected (Trompeter *et al* 2017).

The full list of sports with high prevalence of LBP (Trompeter *et al* 2017) is:
- rowing
- dancing
- fencing
- gymnastics
- underwater rugby
- water polo
- shooting
- basketball
- hockey
- ice hockey
- athletics
- figure skating.

Causes of LBP and diagnostic criteria

Unlike in general populations, where the majority of LBP has no identifiable pathoanatomical structural cause, aetiology of LBP in athletes is more likely to be associated with structural changes in the spine (Schroeder *et al* 2016). This is thought to be a result of athletes exposing their spine to high and repetitive loads over long periods of time. Subsequently, differential diagnoses, including serious pathology and specific injury, must be considered when assessing an athlete with LBP (Jakes *et al* 2015).

Unremitting LBP lasting longer than three to four weeks, particularly in a younger athlete (< 20 years), is considered a "red flag" and should be considered serious until proven otherwise. Therefore, although many athletes and support staff may consider LBP as "a sign training hard", management of young athletes with LBP should include a thorough investigation to establish a diagnosis, with a "simple or non-specific" LBP to be considered as a diagnosis of exclusion only.

SERIOUS PATHOLOGY
LBP caused by a serious pathology is relatively rare (1%) but has to be considered in younger athletes in particular. Onset, duration and nature of LBP will help a clinician to differentiate non-specific LBP, i.e. unrelated to pathology, from a serious pathology including malignancy and tumours (e.g.

"IMPROVING OUTCOMES: UNDERSTANDING THAT LBP MUST BE TAKEN VERY SERIOUSLY IN YOUNG ATHLETES CAN POTENTIALLY DRAMATICALLY IMPROVE THE OUTCOME FOR THE ATHLETE"

"IMPROVING OUTCOMES: A THOROUGH UNDERSTANDING OF 'RED FLAGS' IS ESSENTIAL FOR ENSURING THAT WE ACHIEVE BEST PATIENT OUTCOMES. MISSING THEM CAN HAVE SERIOUS CONSEQUENCES FOR THE PATIENT"

osteoma, osteoblastoma, bone cysts, osteogenic sarcoma), infection (osteomyelitis, discitis), inflammatory spondyloarthropathies (juvenile arthritis, ankylosing spondylitis, psoriatic arthritis), enthesitis or visceral pathology such as pyelonephritis (Jakes *et al* 2015). Focused questioning to explore the existence of "red flags" is important and by definition, presence of any serious spinal pathology warrants a referral for further investigation (Jakes *et al* 2015).

Features that indicate serious pathology, and should be seen as red flags:
- age <20 years, especially pre-pubertal
- sudden onset of severe back pain
- duration of >4 weeks
- thoracic spine pain
- night pain, or pain that wakes patient from sleep
- unremitting pain, even when supine
- fever, chills and / or night sweats
- unexplained weight loss
- immunocompromise, e.g. HIV
- previous malignancy
- corticosteroid use
- recent trauma
- progressive neurological deficit
- bladder or bowel dysfunction
- saddle anaesthesia
- disturbed gait or limp, tripping and / or unexplained falls
- vertebral deformity.

STRUCTURAL INJURIES OF THE SPINE

Compared to the general population, athletes with LBP were found to have higher prevalence of structural pathology regardless of the sport they participate in (Schroeder *et al* 2016). However, the patterns of injuries vary between adults and adolescent athletes. In young adults with LBP, 46% had radiological evidence of bony injuries such as pars interarticularis defects compared to 6% in older adults

(Purcell & Micheli 2009). On the other hand, older adult athletes with LBP tend to have disc related injuries and these are seen in 48% of cases compared to 11% in adolescents (Purcell & Micheli 2009).

Consideration needs to be given to the relationship between spinal injury and LBP. Spinal injuries such as spondylolysis were found to be the most significant risk factor for LBP in NFL players (Iwamoto *et al* 2004) and MRI evidence of lumbar degenerative disc disease increased risk of LBP in gymnasts (Koyama *et al* 2013). There is other research, however, that demonstrates significant pathology in fully active individuals who are free of pain, for example, multilevel disc degeneration and pars stress lesions were found in asymptomatic and fully functioning cricket bowlers (Ranson *et al* 2005). While this conflicting research triggered a debate on the role of screening for structural abnormalities in asymptomatic athletes, it is important to maintain that establishing accurate diagnosis, particularly in young athletes participating in "high-risk" sports, is critical. The additional clinical indicators of possibility of a structural injury to the spine are a sudden onset of focal LBP, cessation of training / competition and disturbed sleep (Kalpakcioglu *et al* 2009).

NON-SPECIFIC LOW BACK PAIN

While athletes may be more susceptible to developing structural injuries of the spine, many will have symptoms that are benign and self-limiting. Importantly, just like in the general population, LBP in athletes can develop into a persistent pain disorder with associated loss of function driven by cognitive, lifestyle and behavioural factors rather than, often co-existing, structural changes in the spine. In such instances, the success of the management is dependent on a broader approach in the identification of dominant factors contributing to the disorder and a subsequent, individualised treatment pathway aimed at addressing the dominant pain drivers.

Risk factors for LBP in athletes

A multitude of risk factors are thought to be associated with LBP in athletic populations (table 1). The type of sport, for example, appears important when it comes to LBP in younger athletes. Biomechanical factors and muscle dysfunction have been associated with risk of LBP across the age groups (Nourbakhsh & Arab 2002). From recent evidence, training load and years of exposure appear to be among the most significant risks for LBP in athletes (Wilson *et al* 2020), as do non-modifiable factors such as age, skeletal maturity and a previous injury (Trompeter *et al* 2017).

TRAINING LOAD

There is some evidence that excessive training volumes, periods of load increase and years of exposure are risk ⊙

	MODIFIABLE	NON-MODIFIABLE
INTRINSIC FACTORS	Biomechanics of the spine, hip, shoulder Muscle function (strength, neuromuscular control, endurance/capacity) Psychological factors (mood, behaviours, cognitions)	Gender (more common in females) Age (young/mature athletes) Previous injury (+ return to play) Skeletal maturity status (growth spurt)
EXTRINSIC FACTORS	High training load Sport rules/regulations Coaching and training cultures Playing time Playing surface Equipment	Type of sport (high risk sports) Level of play (elite more affected) Playing position (bowlers, pitchers)

TABLE 1: Described risk factors for LBP in athletes

STRATEGY	DESCRIPTION	CLINICAL CONSIDERATIONS
Avoid rapid changes in weekly training load (TL) (>10% guideline)	**Weekly increases in TL should not exceed 10% (Gabbett 2016).**	**The 10% guideline is a rough estimate that can be higher/lower depending on the athlete's level, i.e. novice/elite. For example, athletes with very high or very low chronic workload (CW) may not be able to tolerate even 10% weekly increase, whilst a seasoned athlete with moderate or high CW may be able to tolerate weekly increases greater than 10% (Gabbett 2016).** **The "weekly" time window is an estimate which may differ depending on training schedule that can vary across different sports (Wang *et al* 2020).** **Consideration also needs to be given to what unit of load is used and its measuring accuracy, e.g. rate of perceived exertion, or minutes, training or distance covered (Wang *et al* 2020).**
Maintaining acute:chronic workload ratio low (ACWR)	**ACWR should be kept between 0.8 – 1.3 (Gabbett 2016)**	**Some athletes may sustain injury when ACWR is lower than 0.8, others may tolerate ratios higher than 1.3.** **Given that the CW is defined as training average over four weeks with each week weighted equally, athletes with very different training patterns over that time may, in fact, have the same ACWR, i.e. the same perceived injury risk, even though their injury risk would likely differ depending on how they spread their load over the four weeks. Calculating ACWR using exponentially weighted moving averages may therefore be more sensitive measure (Wang *et al* 2020; Maupin *et al* 2020).**

TABLE 2: Strategies to monitor training load and considerations for its use

factors for LBP in adult athletes (Fett *et al* 2017; Wilson *et al* 2020). Also, a larger epidemiological study in adolescent athletes showed that those training less than six hours per week kept LBP prevalence to levels similar to age matched non-sporting controls, i.e. 20.7%, but when weekly training was increased to 12 hours the LBP prevalence rose to 40.5% (Sato *et al* 2011).

Clinically, it is important to consider that while LBP in athletes may not necessarily indicate an injury, ongoing back pain complaints may potentially be an early indicator of inappropriate training load. Therefore, as well as ruling out serious / structural pathology and other risk factors, a review of training load and training practices may be useful to gain an insight into its relationship with the athlete's back problem.

Risk mitigation strategies related to training load

Tracking the training load of an athlete was proposed as a useful method allowing for adjustment of the training stimulus to ensure minimal injury risk and concurrent fitness gain (Gabbett 2016). Training load monitoring has initially gained popularity in sports such as Australian football and soccer given its relationship with reducing injury risk (Gabbett 2016).

Training load can be measured by quantifying (i) the external training load or "the dose" using e.g. GPS devices and (ii) the internal training load or "the response" using e.g. heart rate monitoring, blood lactate measures or simply by rating perceived exertion (RPE) (Halson 2014). To monitor injury risk, the International Olympic Committee recommended using the acute chronic workload ratio (ACWR) that compares the size of the acute (recent) load divided by the chronic (long-term) load (Soligard *et al* 2016). Since then, several limitations

have been identified with how the ACWR is calculated and used to mitigate injury risk (Maupin *et al* 2020; Wang *et al* 2020). Table 2 lists two main practical strategies for training load monitoring in athletic populations (Gabbett 2016), together with clinical considerations in light of the ACWR limitations (Wang *et al* 2020).

AGE

Both young and older age was demonstrated as a risk factor for LBP in athletes (Purcell 2009; Fett *et al* 2017). In adult athletes, increased LBP risk may be a result of high cumulative biomechanical loads imposed on the spine over time. This is supported by high rates of the radiological abnormalities found in seasoned wrestlers, rowers and gymnasts (Lundin *et al* 2001). The problem is compounded in younger athletes by the imposition of high, repetitive loads on spines in the immature skeletal structures and underdeveloped neuromuscular system (Purcell 2009).

Risk mitigation strategies related to age

While load monitoring described above may be appropriate mitigating strategy in seasoned athletes with LBP, monitoring skeletal maturity, combined with training load management, is an important risk management strategy in young athletes. Main indicators (table 3) of skeletal maturity status are:
- chronological age
- anthropometric characteristics
- skeletal age.

The choice of method is dependent on the resources available, the setting, e.g. club, academy etc, the athlete, and the LBP / injury risk profile of the sport.

GROWTH CHARACTERISTICS	GIRLS	BOYS
Age at start	9-10 y	11-12 y
Age at maximum growth	12 y	14 y
Age at which growth slows	>12 y	>14 y
Age until growth continues	16-18 y	18-20 y
Age at maximum height growth (PHV)	11-13 y	13-15 y
Approx rate of growth during PHV	7-9 cm / year	8-10 m / year

TABLE 3: Growth characteristics during the adolescent growth spurt for girls and boys (adapted from Birrer & Cataletto 2002)

Chronological age monitoring

Chronological age (CA) provides an estimate of growth during adolescence (table 3). An important indicator of skeletal maturity is the period of peak height velocity (PHV), which is a period of maximum growth during adolescence. Girls reach PHV at around age 11-13 years and boys between 13-15 years (Birrer & Cataletto 2002). While CA offers a quick and easy estimate of growth periods, only two-thirds of adolescents fall within "normal" age ranges for skeletal status, with as much as a five-year discrepancy (Birrer & Cateletto 2002).

Skeletal age monitoring

Annual screening techniques such as x-rays, ultrasound and magnetic resonance imaging (MRI) are accepted gold standard methods to establish skeletal maturity status (Bergeron *et al* 2015). Wrist and hand x-rays are most commonly used. Limitations include radiation exposures and the resources to cover the cost of annual screening. Nevertheless, this could be a highly effective and efficient risk management strategy for young athletes participating in sports associated with higher prevalence of spinal injury, e.g. gymnastics, rowing and cricket, to inform training load management on a case by case basis (Bergeron *et al* 2015).

Anthropometric screening

Anthropometric screening involves measuring, e.g. height, weight and leg length on a regular basis throughout the adolescent age (table 4). Athletics skills model (ASM) offers a digital growth calculating algorithm available at **www.athleticsskillsmode.nl/en/growth-calculation**. It is a quick and easy method to identify the onset of adolescent growth spurt from a set of basic variables, such as gender, date of birth, standing / sitting height, and weight. Providing the standardised measurement is followed, ASM is shown to be a valid and reliable estimate of growth (Mirwald *et al* 2002). Its one limitation is that its accuracy is dependent on access to the athlete's measurements for a period of more than four years.

METHOD	MONITORING FREQUENCY	ADVANTAGES	LIMITATIONS
Chronological age	Annually	Easy method of monitoring requiring limited resources	Reliability is limited with 5 year+ discrepancy between individuals
Skeletal age (e.g. wrist, hand x-rays)	Monthly, 6-monthly or annually	Highly accurate not affected by puberty Gold standard of skeletal maturity	Invasive, potential exposure to radiation and associated costs
Anthropometric screening (e.g. ASM growth monitor)	Monthly	Considered useful, non-invasive method to help identify the onset of adolescent growth spurt	Series of data needs to be taken every 3-6 months for period of 4+ years, a period that may be difficult to achieve

TABLE 4: Summary of skeletal maturity status monitoring

BIOMECHANICS

How people with LBP move was observed to differ from those without LBP in a number of ways. These include smaller range and lower speed of lumbar motion, reduced proprioception and stiffer movement strategies (Laird *et al* 2014, 2019). It was unclear, however, whether these movement alterations precede the development of, or contribute to, the perpetuation of LBP. A systematic review of prospective studies showed that restricted lateral flexion and limited lumbar lordosis predicted the development of LBP in general populations (Sadler *et al* 2017). The picture is less clear in athletes. In cricket, for example, coupling of lateral flexion and axial rotation, also called the "crunch factor", was implicated in the development of contralateral spine injuries in cricket fast bowlers (Glazier 2010). More recent, prospective and retrospective evaluation found no differences in biomechanical measures of those senior and junior cricketers with and without history of LBP, or in those who did and did not go on to develop LBP (Senington *et al* 2020).

There is some evidence to suggest that load-sharing between neighbouring anatomical regions may be important. Senington *et al* (2020) observed that cricket fast bowlers with no history of LBP had four times greater thoracic rotation during the back foot impact, serving as a "wind-up" mechanism to generate pace on the ball, when compared to those with history of LBP. Golf, squash and tennis are other examples of sports where players with LBP demonstrated significantly restricted range of motion (ROM) at the hip, and L-R hip ROM asymmetries compared to their pain free counterparts (Van Dillen *et al* 2008). Interestingly, a recent study of in-line hockey players suggests a cut-off point with hip external and total rotation ROM of less than 56.5 and 93 degrees, respectively, to predispose players to developing LBP (Cejudo *et al* 2020). ⊙

BODY AREA	BIOMECHANICAL RISK	ASSESSMENT METHOD	TESTED POPULATIONS
Spine	Restricted lumbar lateral flexion (Laird *et al* 2014; Sadler *et al* 2017)	Tape measure assessment of the difference between middle finger position on ipsilateral thigh to most distal position of middle finger achieved in max lateral flexion	General public
Hip	Restricted internal rotation (IR) (Sadeghisani *et al* 2015) L-R asymmetry External rotation (ER) < 56.5 deg and total hip rotation (TR) < 93 deg (Cejudo *et al* 2020)	Passive hip rotation in prone using inclinometer or goniometer	In-line hockey Rowing Hockey Golf Squash and tennis

TABLE 5: Clinical assessment tests screening for potential risk of LBP in selected sports

Mitigation of biomechanical risks

Biomechanical screening to assess LBP risk is frequently established on laboratory based movement analysis systems (Elliott 2000; Vad *et al* 2004), rendering them of limited clinical use. A summary of clinical assessment tests indicating a biomechanical risk for LBP in athletes in selected sports is summarised in table 5.

When it comes to biomechanical modifications, there appears to be insufficient evidence for these to successfully manage LBP in athletes (Thornton *et al* 2020). There is some low-level sport-specific evidence such as addressing hip asymmetries leading to a reduction of LBP in golfers (Reinhardt 2013), and moving rowers from end-range flexion in catch phase that coincided with a reduction in their LBP (Ng *et al* 2015). Coaching interventions in cricket also showed some promise, demonstrating that bowlers with mixed bowling action can be successfully moved towards a safer bowling technique, resulting in reduced incidence or progression of their lumbar disc degeneration compared to those who continued to use the mixed bowling action (Elliott & Khangure 2002). While these studies indicate that coaching modifications towards safer technique are possible, the Elliot & Khangure (2002) research took three years of intensive coaching input to produce their results. Furthermore, the impact of such interventions on the clinical outcomes, such as level of function or training time lost to LBP, has not yet been studied.

MUSCLE FUNCTION

Impaired muscle function of the lumbo-pelvic-hip complex seems to be a hallmark of LBP (Nourbakhsh & Arab 2002). This appears important both in athletes and non-athletes with LBP demonstrating similar levels of trunk extensor deconditioning compared to pain free controls (Moreno Catalá *et al* 2018). The role of muscle function as a predisposing risk factor for LBP is, however, less clear. From sport-specific literature, tennis players with LBP had lower abdominal endurance and less co-contraction compared to matched pain free controls (Correia *et al* 2016). Also in tennis players, those with erector spinae neuromuscular imbalance were more likely to develop LBP, while completing back extensor programmes proportionately reduced their symptoms (Renkawitz *et al* 2006). Elite golfers with a side bridge endurance score of less than 12.5 seconds had increased risk of LBP (Evans *et al* 2005), and cricketers with LBP undergoing neuromuscular training were shown to reverse their impairments in neuromuscular control of transversus abdominis and multifidus, and this coincided with a reduction in their LBP (Morton *et al* 2014).

Importantly, the role of muscle function in predicting LBP appears to vary depending on the age of the athlete. While reduction in trunk muscle strength was predictive of LBP in adult athletes (Noll *et al* 2016), this was not the case in adolescent athletes whose trunk flexion and extension peak torque didn't discriminate between those with and without LBP (Mueller *et al* 2017). This is likely to be a reflection of the multifactorial nature of LBP in athletes where factors such as training load may pose a greater risk for LBP than does their muscle strength.

Mitigation of risks related to muscle function

Optimal muscle strength and neuromuscular control is considered critical in compensating for external forces placed on the spine in athletic populations. Trunk muscle function screening is therefore a frequently clinically utilised risk management strategy. The Sorensen test (figure 1) is suggested as a useful proxy of trunk muscle endurance with good reliability, reproducibility and discriminative validity between athletes with and without LBP (Evans *et al* 2007). An important consideration is that this test was developed to measure muscle endurance in a single movement plane and thus may not be sensitive to detect unilateral or multiplanar deficiencies.

Functional movement screen (FMS) also demonstrated some utility in identifying athletes at risk of LBP. A study on collegiate female rowers found that those scoring ≤16 on FMS with a shorter plank test hold time (mean time 109.5 seconds) had a 1.4 times greater risk of developing LBP (Gonzalez *et al* 2018). While this study is promising in FMS utility to screen for LBP risk, the size of the risk was relatively small and

FIGURE 1: The Sorensen test: The Roman chair variant typically used in sport (image reproduced with permission)

was shown only in females, so further research is required in broader athletic populous.

PSYCHOSOCIAL FACTORS

Psychosocial factors including low mood, anxiety, distress and depression have long been considered strong predictors of LBP in the general population. Low mood, psychological complaints and catastrophising were also found to be among the factors associated with significant injury in elite dancers (Cahalan et al 2014). Noll et al (2016) studied athletes from Brazil and found that "feeling lonely" and loss of sleep were among the highest contributors to LBP in the range of demographic, socioeconomic, hereditary, exercise-level, anthropometric, strength, behavioural and postural factors.

Mitigation of psychosocial risk

The diagnostic uncertainty and often long-term impact of LBP can become career limiting for athletes, causing distress and anxiety that may impact on their recovery. Early screening of psychosocial risk factors was therefore recommended as a means of preventing chronicity in athletes (Wippert et al 2017a).

The Örebro Musculoskeletal Pain Screenings Questionnaire (ÖMPSQ) (Boersma & Linton 2005) and the STarT Back Tool (Hill et al 2016) are two examples of how the risk of LBP chronicity is assessed in the general population. However, these tools have not been validated in athletes as it is argued that they operate in a different "pain" context (Wippert et al 2017a).

Alternative tools have therefore been developed for athletes (Wippert et al 2017b, 2020). These include the Risk Stratification Index (RSI), which gives an estimate of the risk of LBP chronicity in the athlete, and the Risk Prevention Index (RPI) that offers personalised recommendation for management (table 6). However, while the RPI demonstrates clinical benefit in the general population (Wippert et al 2020), its effect in athletic populations is yet to be determined. Nevertheless, both tools outperform the ÖMPSQ in demonstrating excellent transferability, sensitivity, specificity and discriminative validity (Wippert et al 2017a), and these are the first validated tools offering a promise to assess LBP chronicity risk in athletes.

Summary and conclusions

The impact and associated burden of LBP in athletes is comparable, if not excessive, to that seen in the general population. The nature of LBP in athletes, however, is different. In young athletes, LBP is atypical and, as such, needs to be considered serious until proven otherwise. Athletes also have higher rates of structural injuries of the spine, although the relationship with LBP is unclear. The potential impact of spinal injury and LBP on longer term health outcomes and on a sporting career is yet to be determined. The aim of this article is to summarise the evidence of potential risk factors for, as well as management of, LBP and spine injury in athletes to guide clinicians in helping to maximise the spinal health, and ensure long and thriving sporting careers of their athletes.

About the author

Dr Liba Sheeran is Reader in Physiotherapy and Population Health Research Theme Lead at Cardiff University. She is also a Consultant Physiotherapist for Welsh Athletics working with elite track and field athletes. Her interest is in enhancing health through physical activity and exercise with focus on musculoskeletal disorders (MSDs). Liba's research track record is in the development and implementation of active physical interventions using health technologies. Her research involves exploring utility of wearable sensors, video-tracking and artificial intelligence (AI) models for assessment and personalised exercise management of MSDs. She speaks nationally and internationally and runs expert workshops in management of complex low back pain. ⊙

CONTACT DETAILS
sheeranl@cardiff.ac.uk
@libasheeran

TOOL	ITEMS	DOMAIN	PREDICTION	TIME	TARGET GROUP
Risk Stratification Index (RSI)	8-17	Biopsychosocial (e.g. fear avoidance, catastrophising, depression, lifestyle, work situation, financial incentives, exercise status)	Predictor of LBP chronicity	6-12 months	General population, athletes
Risk Prevention Index (RPI)	3-16	Biopsychosocial (e.g. fear avoidance, catastrophising, depression, lifestyle, work situation, financial incentives)	Identification of risk profile groups: (pain experience; social environment; stress; medical environment)	6-12 months	General population, athletes

TABLE 6: Overview of chronic LBP screening tools suitable for athletic population

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Cauda Equina Syndrome: the essential facts every physiotherapist should know

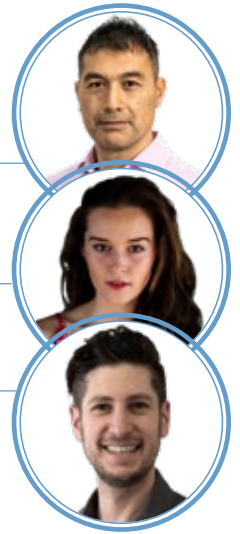**SOLOMON ABRAHAMS** PhD MCSP SRP FOIMM
Consultant Physiotherapist

LARA FOSTER BSc (Hons) FOIMM
Postgraduate Intern

ROBERT HARRIS BSc (Hons) MCSP SRP FOIMM
Extended Scope Physiotherapist

Cauda Equina Syndrome (CES) has potentially devastating effects not only for the patient, but also for the practitioner, should misdiagnosis and litigation occur. Deciphering which patients require an emergency referral from those who need close monitoring can be troublesome, given that there are a number of differential diagnoses that can masquerade as CES symptoms. It is no wonder, therefore, that practitioners are frequently over- or under-referring patients. This article will aim to establish a greater understanding of how CES symptoms progress and how to physically examine for these. The impact of this could dramatically reduce misdiagnosis and improve management in CES patients.

LEARNING OUTCOMES
TO SUPPORT PHYSIO FIRST QAP

1 Appreciate the different definitions of CES.
2 Understand how CES progresses and why the symptoms aren't always clear.
3 Be aware of the differential diagnoses that can cloud the subjective history in CES patients.
4 Know the importance of the objective examination and what signs to look for during it.

Introduction

Cauda Equina Syndrome (CES) is a serious pathology which normally requires emergency surgery. However, much debate remains amongst authors as to the most consistent way to diagnose this life-changing pathology.

Good quality, consistent research is seriously lacking, creating confusion amongst clinicians where mistakes are often made and, ultimately, misdiagnosis. This may partly also explain the high incidence of patients presenting at Accident & Emergency (A&E) departments for urgent MRI scans with suspected CES, where up to 90% of cases prove negative (Bednar 2016). This is confirmed by other authors who are looking at ways of improving our clinical diagnosis of CES within a primary care setting.

Sadly, large numbers of patients are still being referred to emergency departments based on clinical findings where the majority of urgent scans requested are either not CES or are normal, suggesting other causes of the symptoms (Harrop et al 2004). This article highlights the current literature available to help physiotherapists identify CES more reliably.

Definition

CES occurs when there is compression of the cauda equina, a group of lumbar and sacral nerve roots that originate from the conus medullaris of the spinal cord (Dionne et al 2019; Woodfield et al 2018). This serious pathology can manifest into symptoms such as altered or reduced lower limb sensation, weakness of the lower limbs, reduced motor innervation, bowel dysfunction, sexual dysfunction and bladder dysfunction (Ahad et al 2015; Bednar 2016; Dionne et al 2019; Gleave & Macfarlane 2002; Gitelman et al 2008).

The British Association of Spine Surgeons (BASS) published guidelines for clinicians managing patients with CES, defining patients suspected of having CES as "...presenting with acute back pain and/or leg pain with a suggestion of a disturbance of bladder or bowel function and/or saddle sensory disturbance". The guidance goes on to state that most patients with these presentations will not have critical compression of the cauda equina. However, in the absence of reliably predictive symptoms and signs,

"IMPROVING THE UNDERSTANDING OF CES AMONGST CLINICIANS REQUIRES STRONGER AND MORE SPECIFIC DEFINITIONS BASED ON CURRENT EVIDENCE"

there should be a low threshold for investigation with an emergency scan (Germon *et al* 2015).

Arguably, to improve an understanding of CES amongst clinicians and reduce inappropriate referrals, stronger and more specific definitions should be described based on the current evidence. In an alternative definition, Ahad *et al* (2015) succinctly described CES as "the squeezing of multiple lumbosacral nerve roots below the conus medullaris causing specific symptoms". In a comprehensive review in 2009, Fraser *et al* (2009) suggest many variations and definitions exist on CES with little consistency. This is supported by another clinical review (Srikandarajah *et al* 2018) that critically appraised 61 CES studies and reported that 33% had no definition of CES at all. However, not all authors agree, further suggesting that CES should be divided into the following two distinct categories based on the severity of pressure on the spinal cord.

1. CES-Incomplete
When the compression is incomplete (CES-I), the patient often experiences altered urinary sensation, poor urinary stream, loss of desire to void and strain on micturition (Gleave & Macfarlane 2002; Gitelman *et al* 2008). Additionally, saddle paranaesthesia deficit may be present (Gleave & Macfarlane 2002; Gitelman *et al* 2008).

2. CES-Retention
Complete compression or CES-Retention (CES-R) is characterised by overflow incontinence, i.e. an unexpected leakage of urine due to a full bladder from an inability to empty the bladder, with or without an awareness the bladder is full, or painless urinary retention. This is usually accompanied by extensive saddle paranaesthesia and deficient trigone sensation (Gleave & Macfarlane 2002; Gitelman *et al* 2008).

Prevalence
There is a relatively low prevalence of CES in patients with chronic lumbar spine pain. The Clinical Standards Advisory Group (CSAG) suggests that, of all patients with lower back pain, less than 1% represent a serious pathology such as CES (Greenhalgh *et al* 2018). In Slovenia, a comprehensive retrospective review estimated the incidence of CES resulting from lumbar disc prolapse to be 1.8 per million of the population (Gardner *et al* 2011). However, there is a high dichotomy in the figures reported, where an alternative study found this prevalence to be 1:100,000 (Bin *et al* 2009). Some authors even suggest CES is so rare that a General Practitioner (GP) in a general setting may only see CES once in their career (Greenhalgh *et al* 2016). These epidemiological inconsistencies may be distorted by the quality of available studies and a multitude of CES definitions available.

More specifically, Buchanan (2013) reviewed 753 patients with lower back pain, 28% of whom reported altered bladder and bowel function, but only one of the 753 patients had a radiologically confirmed CES that was managed with surgery. A comparable study published in 2015 retrospectively analysed 79 A&E referrals in the United Kingdom (UK), all referred for suspected CES (Ahad *et al* 2015). The authors reported just five of these patients had radiologically confirmed clinical CES (Ahad *et al* 2015), and that this 6% accuracy rate is inefficient and suggests more research is required to improve the use of referral pathways.

Historically, males and females have been affected equally by CES (Bednar 2016). However, some authors have suggested male CES instances are slightly higher, especially if the patient is obese or has a history of lower back pain (Ho 2003). Moreover, some studies suggest that patients between the ages of 30 to 50 are most at risk of CES (Verhagen *et al* 2016; Fuso *et al* 2013). This is most likely considering that discogenic herniations are suggested to be a primary cause of CES (Woodfield *et al* 2018; Gardner *et al* 2011) and patients in this age group are frequently affected by this (Ho 2003; Korse *et al* 2017a).

Causes
A number of structures, iatrogenic processes and pathologies can instigate the onset of CES (Gleave & Macfarlane 2002), the most prevalent being lumbar disc prolapse (Woodfield *et al* 2018; Gardner *et al* 2011). However, it should be considered that approximately just 2% of all herniated discs result in CES (Bydon *et al* 2016). Therefore, careful consideration of the symptoms present should be evaluated before CES is indicated. Other causes attributed to damaging the cauda equina include: trauma (Harrop *et al* 2004); spinal stenosis (Gitelman *et al* 2015); other space occupying lesions (Fraser *et al* 2018); sarcoidosis (Kaioriboon *et al* 2005); infections, such as meningitis (Cooper & Sharpe 1996); diabetes, associated with chronic inflammatory demyelinating polyradiculoneuropathy (Lai & Ubogu 2007); abdominal aortic dissection, albeit being exceedingly rare (Small *et al* 2005); and other complications post-surgery (Fraser *et al* 2018). In all, there are a multitude of origins of CES; thus, any patient presenting with the conditions aforementioned should be thoroughly examined.

Another potential cause of the presentation of listed signs and symptoms might include side effects induced by the medication, rather than CES itself, something which will be discussed later in the section on differential diagnosis.

Pathophysiology
The cauda equina nerve roots are known to respond poorly to mechanical pressure and the poorly developed epineurium, the outermost layer of peripheral nerves, histologically predisposes this area to injury (Reina *et al* 2020). Furthermore, traumatic ⊙

exposure at the cauda equina may trigger vascular occlusion, physiological stress, physical compression or indeed a combination of these factors (Eames 2020). The current research on macrophages in the Wallerian cycle, and the role of serotonin in nerve degeneration seen within CES is summarised here.

MACROPHAGES IN THE WALLERIAN CYCLE

Initially, when physical nerve compression occurs, it disrupts the regular functioning in the axon and schwann cells; this upregulates chemokines and cytokines in order to recruit macrophages into the affected nerve (Chen *et al* 2015; Napoli *et al* 2012). Macrophages are mononuclear phagocytes that originate from the bone marrow (Doulatov *et al* 2010). The relocated macrophages contribute to Wallerian degeneration by removing any myelin debris (Chen *et al* 2015). Wallerian degeneration was originally described by Waller as the cycle in which nerve fibres separate from their respective cell bodies (Waller 1850).

Following the initial phase of degeneration, the macrophages are then polarized into their anti-inflammatory M2 phenotype (figure 1); these changes are most prevalent at days three and four post-trauma and are essential for promoting nerve regeneration (Chen *et al* 2015). However, the more recent theories of macrophage function, as summarised by Chen *et al* (2015), have been based upon the understanding that the instigator of the neurological injury is eliminated in the acute phase. In CES, if the compressive force upon the cauda equina is not removed urgently, then regeneration of the nerve endings may never occur. It should be noted that in humans it takes up to a week following nerve compression for the initial phases of axon, or Wallerian degeneration to be completed (Chaudhry & Cornblath 1992; Beirowski *et al* 2005). Furthermore, detached axon segments are still able to transmit action potentials when stimulated during this period (Tsao *et al* 1999). Given axon degeneration is asynchronous (Beirowski *et al* 2005), this may provide evidence for why CES patients frequently experience inconsistent symptoms.

Serotonin and nerve degeneration

Blood flow to the compressed cauda equina nerve roots is largely thought to be altered (Gitelman *et al* 2008; Sekiguchi *et al* 2004, 2002; Lee & Wolfe 2000) but the pathophysiology of this is controversial. Sekiguchi *et al* (2002) investigated the role of serotonin which, in CES, has a vasodilative effect in healthy nerves (Gitelman *et al* 2008). When the cauda equina nerve roots are compressed, the associated blood vessels are thought to be forced into vasoconstriction, even in the presence of serotonin (Gitelman *et al* 2008). This abnormality led Sekiguchi *et al* (2002) to propose that the endothelial cells which form the blood-nerve barrier (Maiuolo *et al* 2019) and mediate hormone secretion (Flammer *et al* 2012; Deanfield *et al* 2007), may malfunction following nerve compression. Results suggested that changes to the serotonin cause this vascular deviation, not a physical impingement of the blood vessels from a compressive force .

In a similar fashion, Jonsson *et al* (2015) also reported deviations in the expression of serotonin. These authors postulated that disc herniation, as commonly seen in CES (Gardner *et al* 2011), decreases the expression of serotonin receptor 2c (Jonsson *et al* 2015), potentially then triggering vasoconstriction. However, with a sample size of five individuals, Jonsson *et al* (2015) failed to demonstrate any statistically significant data to support this. In addition, Sekiguchi *et al* (2002) used inflated balloons inserted deep into the lamina of dogs to simulate cord compression. While theoretically this allows the authors to examine the effects of the chronically compressed cauda equina in vivo, the true nature of CES is multifaceted and non-uniform in origin. Therefore, to confirm the reliability of this proposed theory, future research should obtain larger sample sizes and compare different mechanisms of CES.

Signs, symptoms and physical examination

Generally, CES patients present with a series of clinical features including perineal anaesthesia, lumbosacral root sensory deficit, lower extremity weakness, difficulty with bladder or bowel control, sexual dysfunction, lower back pain, and bilateral or unilateral sciatic nerve pain (Ahad *et al* 2015; Bednar 2016; Dionne *et al* 2019; Gleave & Macfarlane 2002;
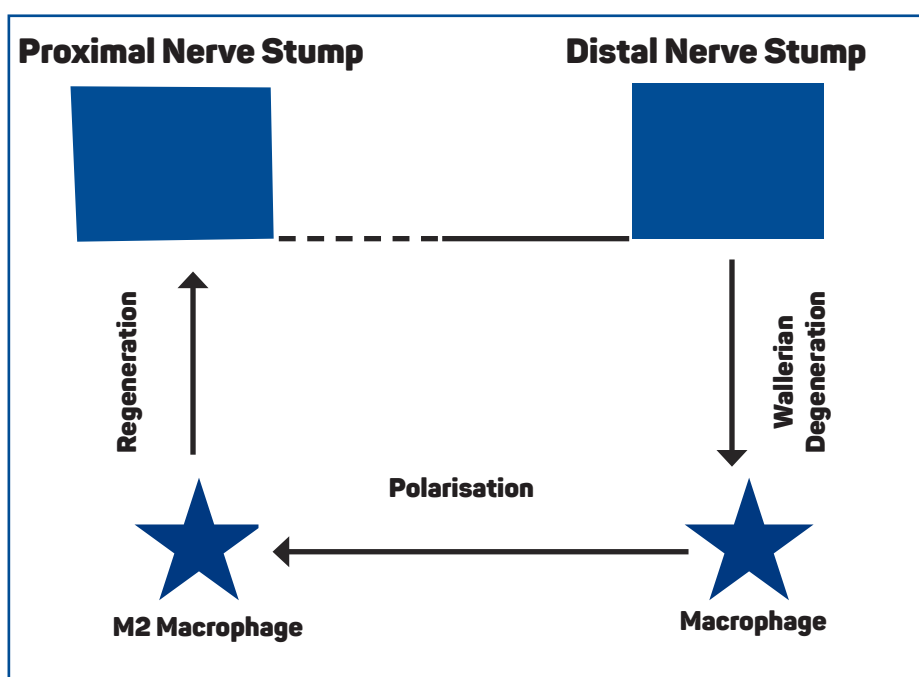

FIGURE 1: Macrophages and the Wallerian cycle

Gitelman *et al* 2008). More specifically, CES-R cases usually present with saddle anaesthesia and bladder / bowel retention / incontinence, while CES-I patients often have saddle anaesthesia but minor bladder / bowel dysfunction (Gleave & Macfarlane 2002; Gitelman *et al* 2008).

However, the Cauda Equina Foundation (2020) recommends a cautious approach because it is estimated just 19% of patients with genuine CES will present with these characteristic signs and symptoms. The Foundation confirmed in their 2020 review of the evidence that there is little consensus amongst authors upon these clinical symptoms, further perpetuating the ambiguity in CES diagnosis (Cauda Equina Foundation 2020).

What does the evidence suggest?

Patients with CES normally present with severe, acute lower back pain and radiculopathy (Ahad *et al* 2015; Bednar 2016; Dionne *et al* 2019; Gleave & Macfarlane 2002; Gitelman *et al* 2008). Furthermore, sciatic nerve pain can be present in up to 97% of patients (Korse *et al* 2017a). Suspected CES frequently presents with bilateral neurogenic sciatica in most cases (Greenhalgh *et al* 2018; Gardner *et al* 2011; Bednar 2016; Korse *et al* 2017a), but this can be unilateral in some cases, or even absent in others (Gardner *et al* 2011). For the minority of patients presenting with unilateral leg pain, many were found to have a better prognostic outcome, compared to those with bilateral leg pain (Fraser *et al* 2009). Bilateral leg pain should sound alarm bells as this may indicate a serious centralised disc prolapse (Fraser *et al* 2009).

Whether acute or indeed chronic in origin, CES is generally quick to progress (Gardner *et al* 2011; Gitelman *et al* 2008);

rather than in months or years, this can be within just a few days (Bednar 2016). A 2017 study involving 75 patients with confirmed CES found the average onset of acute symptoms was just 84 hours (Korse *et al* 2017a). However, a comprehensive review and evaluation of 105 studies by Fraser *et al* (2009) found there was little agreement on the level, intensity, onset or duration of pain symptoms. Moreover, the literature presents a great dichotomy in the time frames of CES symptoms (Fraser *et al* 2009), thus further confounding the already unclear information provided to clinicians.

As CES, in most cases, is primarily caused by intervertebral disc prolapse (Gardner *et al* 2011; Woodfield *et al* 2018), the pain symptoms of CES are predicted to be similar in nature and intensity of such. The Cauda Equina Foundation (2020) recommends that clinicians should recognise pain as the main presenting feature in genuine CES. However, as pain is subjective and interpretation is inconsistent culturally, this may also explain the variance in clinical approach. Good communicative skills (notwithstanding that English may not be the first language of the patient or clinician) are an essential factor in ensuring the patient understands the clinician, failure to do so is arguably the most common reason for misdiagnosis (Greenhalgh *et al* 2018).

A history of trauma can be involved in many cases (Greenhalgh *et al* 2018; Gitelman *et al* 2008; Fraser *et al* 2009), but this should not monopolise a CES diagnosis as many of the aforementioned chronic pathologies can also cause CES (Gleave & Macfarlane 2002).

More reliably, saddle anaesthesia (or numbness between the legs), and genital sensory disturbance, are common complaints in most of the

more acute cases (Verhagen *et al* 2016). A comprehensive review (Fraser *et al* 2009) reported saddle anaesthesia, along with sensory disturbance of the lower limbs, particularly the upper posterior thigh, groin and perineal region, are the most significant and consistent finding seen in patients with CES. However, a number of patients may not admit these symptoms during the subjective examination. Therefore, a physical examination will be required (Finucane *et al* 2017). This is further supported by the Cauda Equina Foundation (2020) who suggests that it is imperative to test the saddle area because many patients may not realise the deficit themselves or may be embarrassed to articulate this.

CES can also present with bladder and bowel dysfunction that gradually deteriorates over a number of days or weeks (Greenhalgh *et al* 2018; Gitelman *et al* 2008; Chau *et al* 2014). Lost or reduced anal tone can be accompanied by the inability to control bowel motions (Greenhalgh *et al* 2018), but the clinical evidence of this remains debateable. A retrospective study of CES patients in the UK found only 9% presented with bladder incontinence, 8% with urinary retention and 4% with faecal incontinence (Ahad *et al* 2015).

Interestingly, in the review by Fraser *et al* (2009), of the 105 studies, only 29 (27%) found any mention of bladder dysfunction, and 12 suggested bladder involvement was not always present. The authors concluded there was no general consensus on bladder or bowel clinical presentation, and if unsure a cautious and careful monitoring approach is recommended (Fraser *et al* 2009). The ⊙

❝FAILURE TO COMMUNICATE WELL IS, ARGUABLY, THE MOST COMMON REASON FOR MISDIAGNOSIS OF CES❞

Cauda Equina Foundation (2020) suggests one of the reasons for the ambiguity in this area could be the miscommunication and misinterpretation of the subjective examination, something that has been supported by other authors (Greenhalgh et al 2018).

Erectile Dysfunction (ED) is generally considered to be an uncommon symptom and is often confused with other pathologies, thus making it a prognostically poor symptom (Greenhalgh et al 2018; Gitelman et al 2008). This assertion is conflicted by data from Korse et al (2017a), involving patients with confirmed CES. Of the sample studied, only 26 were asked about ED and 25 admitted to having experienced some level of dysfunction (Korse et al 2017a). It remains unclear, however, how this process was reviewed. Furthermore, the subject numbers in this study are low, there is debateable reliability and the findings generally conflict with much of the evidence available.

Subjectively, male patients may not always admit or accept ED to be an issue, notwithstanding the difficulty itself in asking what can be an embarrassing question. Some authors also suggest that other more common causes of ED can include vascular disease, coronary heart disease or even psychological factors which should be considered first (Bin et al 2009).

The Cauda Equina Foundation (2020) advises that this ambiguity and lack of clarity in the symptoms of CES, especially considering some clinicians only examine subjectively, could be one of the reasons leading to high rates of misdiagnosis, misunderstanding and over- or under-referring. Some authors suggest a mandatory objective examination should be required before initial referral for MRI scan (Eames 2020).

Physical examination

Arguably, a patient with suspected CES requires a full objective examination. A subjective examination alone may not be effective in determining a diagnosis (Germon et al 2015). The

British Association of Spinal Surgeons (BASS) recommends the most essential features in the objective diagnosis of CES may include sensory examination of the saddle region, tests for bowel disturbance and a neurological examination (Germon et al 2015). Sometimes, these symptoms cannot always be communicated effectively during a subjective examination, and hence require a thorough physical examination.

A physical examination should consist of lumbar spine movements, findings of which may be consistent with discogenic pathology where movement is limited by pain (Abrahams 2014). Straight leg raise (SLR) is normally painful / positive unilaterally or bilaterally, depending on the severity of the CES (Ho 2003). A positive SLR on the ipsilateral side may indicate a possible disc involvement, especially if pain refers below the knee into the lower leg, and can be 40% specific for a disc herniation (Ho 2003). If a SLR test reproduces pain on the contralateral side of the spine, it may indicate a potential (90% specific) disc herniation (Ho 2003). Considering disc herniation is the most common cause of CES (Gardner et al 2011; Woodfeld et al 2018), it is imperative that clinicians examine these objective measures. Sciatic nerve pain is a common finding in patients with CES, with one study suggesting a 97% prevalence in confirmed cases (Korse et al 2017a).

Palpation may not always be possible in prone as some patients with CES may not feel comfortable in this position. Side lying may be more comfortable and palpatory findings may be consistent with severe disc pathology such as tenderness over the lower lumbar region (Markham 2004). Sadly, there is little evidence in this area and hence findings should be mostly consistent with stenotic or disc type pathologies.

A neurological assessment is essential and should check all dermatomes, myotomes and reflexes. The most common sensory deficits are light touch and pin prick (Cauda Equina Foundation 2020). It is widely recommended that

these are used for the lower limb dermatome examination especially over the inner thighs, back of the thighs and perineal area (Fraser et al 2009). Perineal numbness is the most commonly seen symptom in both CES-R and CES-I cases (Gardner et al 2011). An estimated 75% of all patients with CES present with reduced sensation over the S1-S3 region, with some authors suggesting it would be negligent not to at least check perineum sensation (Markham 2004). Similarly, a clinical study involving patients with confirmed CES found 93% had some altered sensation in the saddle area (Korse et al 2017a). Given that saddle anaesthesia is sometimes difficult to comprehend or understand subjectively by the patient however, a physical examination is considered essential for diagnosis (Germon et al 2015). This is further supported by evidence that suggests saddle anaesthesia is the most reliable and consistent diagnostic finding amongst CES patients objectively (Fraser et al 2009; Finucane et al 2017; Todd & Dickson 2016). Further, the Cauda Equina Foundation (2020) suggests that failing to do an objective / physical examination over the saddle region could expose the clinician to legal disputes if there is a misdiagnosis.

Myotomes and power in the lower limbs should be examined, although this may not always be conclusive. The origin of CES is thought to generally depict how serious any myotomal loss will be (Small et al 2005). For example, a significant central disc prolapse may cause bilateral myotome loss (Small et al 2005). However, approximately just 50% of cases of CES have been reported with associated myotome loss, and the levels presented were extremely inconsistent (Fraser et al 2009). A study of 79 CES patients found motor weakness to be the most defining factor of CES (Ahad et al 2015), but this motor deficiency represented only 27% of the sample, with even lower numbers for other signs and symptoms (Ahad et al 2015). The most common area for disc prolapse in CES patients is L5/S1 and L4/L5 (Korse et al 2017a), supporting the notion that the lower myotomes, especially dorsiflexion of the

ankle and big toe (L4/L5), and plantarflexion (L5/S1) of the ankle, are most likely to be affected (Korse *et al* 2017a).

Anal sphincter tone is normally loose or absent (Finucane *et al* 2017). However a retrospective study over a 12-month period in an A&E department found only 8% of patients with genuine CES had decreased anal tone (Kaioriboon *et al* 2005). Nonetheless Korse *et al* (2017a) found altered anal tension was invariably linked with perineal / saddle anaesthesia. Patients who had a lack of perineal sensation were more likely to have a positive anal sphincter test, whereas those patients who had normal sensation in this area demonstrated negative tests (Korse *et al* 2017a). The authors concluded there was 100% specificity, with strong correlation between a positive anal sphincter test and abnormal sensation in the saddle region (Korse *et al* 2017a). However, it was still recommended that clinicians use this objective test even if the patient demonstrates normal saddle sensation (Korse *et al* 2017a). Some authors have suggested that failure to evaluate rectal tone is one of the most common causes of litigation in the event of misdiagnosis (Kaioriboon *et al* 2005).

Reflexes should be considered in a neurological examination. A comprehensive review found varying degrees of reflex deficit in 31 clinical studies (Fraser *et al* 2009). Even though the consensus was inconsistent regarding which reflexes were absent, it was agreed that including them within the examination gives more ammunition to a correct diagnosis (Fraser *et al* 2009). The most common reflex to be found weak, or even absent, was the ankle jerk reflex, identified in approximately 50% of cases (Ho 2003; Gitelman *et al* 2008).

Some authors suggest a functional assessment is necessary for patients who may have CES (Ahad *et al* 2015). The Cauda Equina Foundation (2020) advises that difficulty in ambulation, alongside pain, is the main clinical symptom in patients with genuine CES. Patients who can ambulate at presentation will generally remain ambulatory (Ahad *et al* 2015). Similarly, patients with paresis, but who are ambulatory with assistance, will have a 50% chance of walking again (Ahad *et al* 2015). Therefore, observing functional capabilities may be a useful baseline measure to post-operatively evaluate surgical outcomes.

An objective and functional assessment not only strengthens diagnosis but can also improve prognostic outcome.

Differential diagnosis

The clinical diagnosis for CES is often unclear (Finucane *et al* 2017). A study in the UK reviewed patients referred for urgent MRI scans with suspected CES and found 10% of the patients' MRI scans were actually completely normal, suggesting other causes of the symptoms, with no apparent pathology at the lumbar spine (Ahad *et al* 2015). However, as previously mentioned, Buchanan (2013) suggests, of 753 patients with lower back and leg pain, 28% experienced altered bladder and bowel function with suspected CES and, quite astonishingly, only one of those patients had radiologically confirmed CES (Buchanan 2013) which represented less than 1% diagnostic accuracy.

Lower back pain and subsequent sciatic pains are common in the general population, with up to 80% of UK residents experiencing these symptoms in their lifetime (Rubin 2007). It is therefore conceivable that many of these patients, potentially some with symptoms associated to CES, have other pathologies not involving compression of the cauda equina.

Adding to this confusion, some medicines used for spinal and / or lower limb pain can cause some symptoms of CES. Opioid medicines, such as Tramadol, are a common medicine prescribed to patients with lower back and sciatic pain that can reduce bladder sensation and increase constipation (NHS 2018). Anti-convulsant medicines, such as Pregabalin or Gabapentin, are frequently prescribed for nerve pain down the legs and can significantly increase the risk of urinary incontinence (Germon *et al* 2015).

Amitriptyline, a commonly prescribed medication for sciatic or radicular pain, can similarly cause urinary retention and sexual dysfunction (NHS 2020). Patients taking hormone replacement therapy (HRT) or diuretics can also experience urinary incontinence as a side effect (NHS 2019). In fact, most disturbingly, the majority of common pain control medicines, used in primary care to treat and manage lower back and leg pain, can masquerade as symptoms of CES in some cases (Germon *et al* 2015).

Urinary incontinence or retention is commonly seen as a red flag for CES in patients with lower back pain (Verhagen et al 2016). It can also occur in patients who present with one of the following conditions: extreme obesity, urinary tract / bladder infections, alcoholism, Parkinson's disease, multiple sclerosis, bladder stones, bladder fistulas, bladder /prostate / ovarian / uterine cancer(s), chronic constipation, prostatic hypertrophy, age related muscular weakness of the pelvic floor, post-hysterectomy complications, cerebral vascular accidents, peripheral neuropathy (diabetes), Alzheimer's, and even patients who have a chronic cough with a weak pelvic floor and, of course, post-natal women (Bin et al 2009; NHS 2019; NICE 2019). A full past medical history should therefore be documented with any onward referral highlighted. Medicines that can cause ED include diuretics, antihypertensives, antihistamines, antidepressants, Parkinson's disease medications and antiarrhythmics (Grant *et al* 2013).

In summary, there is a plethora of medications and pathologies that can cause CES symptoms, and this highlights the importance of obtaining a comprehensive subjective history to avoid misdiagnosis. Clinicians are advised to remember that patients presenting with severe lower back pain, leg pains and accompanying bowel / bladder / sexual dysfunction should not be ignored if the history shows use of any of the listed medications (Greenhalgh *et al* 2016), but that diagnosis should be cautious. ⊙

Treatment and management

The first course of action for any patient suspected of having CES should be to seek an urgent MRI scan to confirm diagnosis (Gardner *et al* 2011; Barraclough 2021). For many this would mean speaking to a GP or transferring the patient directly to an emergency department where high-dose intravenous steroids may be given on admission to reduce pain and inflammation (Small *et al* 2005). If CES diagnosis is confirmed, then an emergency surgical decompression is recommended (Eames 2020; Barraclough 2021; Bin *et al* 2009).

The appropriate surgical intervention is predominantly determined by the underlying cause of CES but the aim is always to reduce the risk of permanent neurological damage (Bin *et al* 2009). Some authors have investigated the efficacy of conservative treatment options for CES that include vasodilative agents (Yone *et al* 1999), and steroid or non-steroidal anti-inflammatory drugs (Nakano *et al* 1998; Della-Giustina 1999). However, the research quality of these studies is extremely poor, with sample sizes as low as 11 per investigation, and many investigations being on animal subjects alone. The majority of the literature advocates for surgical intervention to achieve the best outcome possible (Eames 2020; Barraclough 2021; Bin *et al* 2009).

Prognosis

There can be devastating consequences for an individual with CES and if diagnosis is overlooked there can be permanent dysfunction to bladder, bowel and sexual health (Greenhalgh *et al* 2018). Consequently, litigation and CES are frequently concomitant with the average compensation payment being as high as £336,000 (Fairbank & Mallen 2014). It is well established that patients who receive treatment in the CES-I stage often have a more optimistic prognosis compared to those who have deteriorated to CES-R (Gardner *et al* 2011; Gleave & Macfarlane 2002). However, up to 70% of CES-R patients will still attain a socially acceptable

"THE QUALITY OF RESEARCH ON THE EFFICACY OF CONSERVATIVE TREATMENT OPTIONS FOR CES IS EXTREMELY POOR"

post-operative conclusion (Gleave & Macfarlane 2002), although this may be more due to the patient's ability to adapt to compensatory strategies than as a result of genuine neural regeneration.

Other long-term adverse outcomes reported in the literature include altered sensation in the lower limbs and long-standing back pain (Eames 2020). Additionally, the mental health impacts associated with CES are significantly under-reported. Patients will likely have experienced a sudden, rapid deterioration in symptoms and a traumatic rush to the operative theatre. Furthermore, the patient's ongoing symptoms of dysfunction can have considerable psychosocial impacts (Gardner *et al* 2011), and therefore a referral to psychological therapies may be necessary.

The following is analysis of current prognostic data available for outcomes in bowel function, bladder function, sexual function and mental health following CES, together with a brief summary of the current evidence regarding time to surgery.

BOWEL FUNCTION

There is a lack of reliable data to predict prognostic outcome of bowel function following CES. The most recent investigation of post-operative outcomes testified that 100% of the sample population had improved control functionality after the first year (Shah *et al* 2021). In this sample, more patients had waited longer than 48 hours between the onset of their symptoms and surgery than had received surgery within a 48-hour window. This led the authors to conclude that the timing of the operation may have no bearing upon the outcome of bowel function (Shah *et al* 2021). However, no definition of what specifically indicates an "improved outcome" was provided. Furthermore,

the sample consisted of just 10 patients and no consideration was given to whether these patients were in the CES-I or CES-R phase.

In a more comprehensive study, Korse *et al* (2013) collated the outcomes of defecation function in a total of 238 patients, of which a mean 49.6% experienced some dysfunction 17 months post-surgery. Prognosis of bowel function after CES is under-reported (Fraser *et al* 2009) but it is potentially just as prevalent as bladder dysfunction Korse *et al* (2013). Future research should aim to better quantify outcome measures compared to the phase of CES-I or CES-R degeneration, in order to improve patient and clinician education.

BLADDER FUNCTION

Jha *et al* (2021) and Shah *et al* (2021) are the most recent authors to analyse the outcome of bladder function following CES. Jha *et al* (2021) reported optimal recovery in 73.7% of patients, albeit average recovery time was correlated to the time between symptom onset and decompressive surgery. Nonetheless, time to decompression was found to have no significant effect on the likelihood of positive outcome in the long run for the 19 patients (Jha *et al* 2021). Therefore, length of follow-up should always be considered when reviewing the literature of CES because symptoms can continue to ameliorate for years after surgery (Chang *et al* 2000). In similar fashion, Shah *et al* (2021) recorded symptom improvement in the bladder function in 100% of the patients studied. However, both of the aforementioned investigations used small sample sizes and they only represent CES patients from lumbar disc herniation. The severity of symptoms at the time of surgical intervention is largely thought to dictate prognosis (Qureshi & Sell 2007).

Korse et al (2013) evaluated the outcome of micturition in 409 patients post-decompression. The mean prevalence of incontinence 17 months after surgery was 42.5% (Korse et al 2013). However, this figure may be excessively high. Urodynamic evaluation after CES decompression has been known to show serious disturbance to normal bladder function even in the absence of any symptoms felt by the patient (Hellström et al 1986). In their 2017 follow-up study, Korse et al (2017a) reported that in 75 patients, 48% were still experiencing micturition dysfunction 63 days post-surgery. Consideration must be afforded to the understanding that some of these individuals will recover further in years to come (Chang et al 2000). However, a comparison of these figures from 2013 to 2017 may highlight how little progress has been made in the treatment of CES. Given that such a large proportion of CES sufferers may never fully regain urological function, significantly more research is required to develop strategies to improve these outcomes.

SEXUAL FUNCTION

Sexual function is seldom discussed in practice or the literature (Fraser et al 2009; Shah et al 2021); a study involving 75 patients (Korse et al 2017a) reported only 34.6% of the research available even considered this. Dysfunction is more frequently reported in males than females (Korse et al 2013; Pronin et al 2020), and the two major indications of sexual deficit are ED and decreased sensation in the genitals during sex (Pronin et al 2020). Moreover, unlike urinary dysfunction, the probability of sexual health being restored post-operatively can be slim (Korse et al 2013). Some authors suggest more than 50% of CES sufferers may be left with permanent sexual dysfunction (Korse et al 2017a, 2017b). However, this conclusion has been drawn from limited data. It is suggested that many patients, therapists and doctors lack effective communication or confidence to bring up the subject (Korse et al 2016). Furthermore, ED is thought to be a prognostically poor symptom in the diagnosis of CES due to the number of other pathologies that may cause

this (Gitelman et al 2008; Greenhalgh et al 2018). Therefore, from the limited data available, it is difficult to make any conclusive predictions about ED prognosis in CES.

MENTAL HEALTH

Presently, there is a paucity of research into patient mental health following CES (Hall & Jones 2017). As presented by the National Health Service (NHS) England (2020) in their 2020 Spinal Cord Injury Service Specification, the multidisciplinary team are charged with a duty of care that includes the patient's mental wellbeing. In a series of interviews conducted by Hall & Jones (2017), a number of CES patients expressed dissatisfaction with their quality of care with comments such as "I felt very abandoned", followed by "Nobody knows, it's horrible". This provided an insight into the emotional trauma experienced by patients with CES (Hall & Jones 2017). In addition, and rather alarmingly, one patient reported they had been made to feel like they were making up their symptoms (Hall & Jones 2017). To evaluate these accounts objectively it must be considered that all patients were reimbursed with a small fee of £20 (Hall & Jones 2017) and, consequently, a conflict of interest could have been indicated. These accounts do, however, possibly highlight a significant area for development within outpatient care, and improved communication about symptoms during assessment, using a language accessible to the patient, may offer succour. More research into the psychological impacts of CES could develop the understanding of the clinician, and ultimately improve patient care.

TIME TO SURGERY

Prompt diagnosis and timely surgical intervention are factors universally thought to have a life-changing impact for CES patients (Dionne et al 2019; Gardner et al 2011; Fraser et al 2009; Gleave & Macfarlane 2002; Gitelman et al 2008; Bin et al 2009; Greenhalgh et al 2016; Pronin et al 2019; Chau et al 2014). However, the exact time frame for an optimum patient outcome remains controversial (Pronin et al 2019). Gleave

& Macfarlane (2002) proposed that spinal surgery 48 hours or less post-symptom onset would afford the patient maximal chance of recovery. Although some authors claim this number may be greater (Shah et al 2021) or lesser (Todd 2005), the overall consensus agrees a prompt surgical intervention is preferable (Dionne et al 2019; Gardner et al 2011; Fraser et al 2009; Gleave & Macfarlane 2002; Gitelman et al 2008; Bin et al 2009; Greenhalgh et al 2016; Pronin et al 2019; Chau et al 2014). The unanimous principle that biological systems generally deteriorate in a continuous linear progression can be applied to CES (Furlan et al 2011). Therefore, earlier intervention theoretically prevents more damage to the compressed nerve roots and this may support Gleave & MacFarlane's (2002) conjecture that timing is particularly important to outcome in CES-I, given there is opportunity to prevent further degeneration.

On the other hand, Pronin et al (2019) recently wrote a review analysing the histopathology and vascular changes associated with CES prognosis. The authors concluded that outcomes post-surgical decompression were determined by the degree of compression, resulting in excessively high systolic local blood pressure and irreversible lesion (Pronin et al 2019) and found that the histologic effects created by the amplitude of compression to be more consequential to the outcome than the duration of compression. However, these conclusions were based on predictions from limited, short time points. The compressed nerves were afforded just 90 minutes of recovery time and, therefore, all conclusions past this time were purely predictive. Furthermore, whilst the electrophysiological studies used may provide key information about changes in nerve firing, this study type lacks any pathological or neurobehavioral measurement. The implications for CES management in practice are therefore limited because no clinical features can be highlighted. There is a multitude of information available on the subject of time to surgery for CES. However, ⊙

the approach currently advocated for the best prognosis is a prompt surgical intervention and decompression of the nerve roots (Dionne *et al* 2019; Gardner *et al* 2011; Fraser *et al* 2009; Gleave & Macfarlane 2002; Gitelman *et al* 2008; Bin *et al* 2009; Greenhalgh *et al* 2016; Pronin *et al* 2019; Chau *et al* 2014).

Conclusion

There is a distinct lack of consistent and reliable evidence available to guide clinicians in the accurate diagnosis of CES and misdiagnosis is common as a result (Ahad *et al* 2015). This may explain both the high incidence of patients with suspected CES arriving at A&E departments where 90% of cases are not CES (Bednar 2016), and also the high value of litigation where the CES diagnosis has been missed. The literature does, however, support both thorough subjective and objective examinations with some recommended tests, which may currently sit outside of the normal boundaries of physiotherapy, unless the clinician can demonstrate CPD competency.

Future research should evaluate the efficacy of virtual triage, currently utilised by many clinicians during the coronavirus pandemic (Mehrota *et al* 2020), compared to physical patient appointments. As seen from the current evidence, a complete thorough examination is required to avoid misdiagnosis. Additional research may also be required to demonstrate the diagnostic accuracy of CES with some changes made to current practice, utilising some of the key facts outlined within this article.

Some recommendations can be drawn from the current data available which is depicted below.

RECOMMENDATIONS

- Key symptoms include subjectively:
  - Acute lower back pain with bilateral / unilateral neurogenic sciatica in 90% of cases.
  - Reduced perineal sensation / saddle anaesthesia in most cases.
  - Altered bladder / bowel function is not 100% reliable so detailed and comprehensive questioning recommended.
  - ED is not a reliable indicator.

- Key signs include objectively:
  - Positive straight leg raise in 90% of cases.
  - Reduced dermatomes specifically over S1-S3 and perineal region in 75% of cases.
  - Reduced lower myotomes, especially over L4/L5 and L5/S1 in 50% of cases.
  - Positive anal sphincter tone test can be found in most patients who also present with perineal / saddle anaesthesia. This obviously may require a chaperone.

- Clinicians are advised, where possible, to complete a cautious, thorough and easy-worded subjective examination AND an objective examination. The subjective examination needs to be clear and understood by both parties and should not be rushed.

- The clinician must document everything found, including a full subjective history, full medical history, current medicines in addition to the physical examination.

- Differential diagnosis should always be considered cautiously, specifically with regard to current medication and when they were prescribed, past and current medical history and, arguably, family history.
- Where the clinician is unsure, a second opinion must be obtained immediately, the decision documented, and the patient referred for an urgent MRI where necessary.

CONTACT DETAILS
sol@anatomie.co.uk

About the authors

Dr Solomon Abrahams, PhD, MCSP, is a Consultant Physiotherapist with more than 20 years of clinical experience, primarily working in private practice and primary care. He also lectures at University College London, and formerly at King's College London and Cambridge Medical School. He has published several textbooks including *Musculoskeletal Medicine in Primary Care* and more than 50 clinical research articles. He is Honorary Professor with the Foundation of International Musculoskeletal Medicine (FOIMM) and sits on several UK and US editorial boards.

Ms Lara Foster, BSc(Hons) is a postgraduate intern working in private practice and currently in her first year of her MSc Physiotherapy. Lara is a keen academic and passionate about incorporating evidence based research into clinical practice.

Mr Robert Harris, BSc(Hons), MCSP is an experienced Extended Scope Physiotherapist in spines working in primary care. He leads a team of other spinal specialists with more than 15 years in clinical practice. He is partner in a private practice and is lead CPD and is instrumental in helping to develop the FCP role. Along with Dr Abrahams, he teaches subjects outside the normal boundaries of physiotherapy to help advance our profession ready for primary care.

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Understanding disc herniation: a historical perspective

TOM JESSON

The disc herniation is such a familiar diagnosis and such a familiar sight on an MRI that it is easy to forget what a complex and remarkable thing it is. In looking back at how the disc herniation was first discovered, and at the efforts made thereafter to understand why they are painful and why they occur in the first place, we can gain new insight into a familiar topic.

LEARNING OUTCOMES
TO SUPPORT PHYSIO FIRST QAP

1 Understand how physicians first realised that disc herniations are often responsible for radiculopathy.
2 Be aware of the historical perspective on complexity of herniation-related pain.
3 Be aware of the historical perspective on the complexity in the causes of disc herniations.
4 Appreciate that our current understanding of musculoskeletal pain is historically contingent and subject to change with the discovery of new information.

Introduction

Surgeons first began removing disc material from spines in the early 1900s – although they didn't realise it at the time.

The first surgeon to describe this operation seems to have been Krause who, in 1905, performed an L3 laminectomy on a man with what we would now call cauda equina syndrome (Postacchini 1999). When Krause removed the mysterious whitish mass of disc material, he thought it was a cartilagenous tumour or "enchondroma". Surgeons would continue to describe and remove these "enchondromas" for the next 30 years.

Some physicians did notice that radiculopathy could be caused by a herniated intervertebral disc, but these cases were always considered curiosities. For example, in 1911 Middleton & Teacher described the case of a workman who, on lifting a heavy plate from the floor to a bench, felt a "crack" in the small of his back. That evening, the man felt "a sudden, agonising pain" in his legs, "with peculiar sensations as if the limbs were 'sleeping'". Soon after, he "found he could not move either limb […] His bladder and bowels now became paralysed […] Two days later he was sent up to the surgical wards as a case of intestinal obstruction. His bowels moved in the ambulance van without his knowledge" (Middleton & Teacher 1911).

Since nobody at the time knew much about cauda equina syndrome, the workman was left in a hospital bed untreated. He developed bedsores which became infected, and he died.

In the post-mortem examination, Middleton & Teacher found that a segment of the man's spinal cord had been compressed by "an irregular, roughly circular flat mass of firm white tissue, which looked rather like the pulp in the centre of the intervertebral discs" (figure 1). They performed further experiments to confirm that such an injury could be caused by bending and lifting, as the man had been doing when he felt a "crack" in his back. Middleton & Teacher concluded by saying that "the rupture of an intervertebral disc during muscular effort may prove to be a very rare injury, but it may prove to be the explanation of certain cases the nature of which has been regarded as quite obscure".

FIGURE 1: Artist's impression of herniated disc material. Herniated disc material often contains not only nucleus but also annulus, endplate and even bone (tomjesson.com/thebook)

That same year, Goldthwait also noticed that radiculopathy could be caused by a herniated intervertebral disc (Goldthwait 1911). He described the case of a man who, on lifting a heavy suitcase, felt a pain in his back. The next morning the man took a bath and, "on trying to get out of the tub, in leaning forward and straining to get up, something slipped in his back". The man developed a lateral shift and pain down both legs.

Goldthwait was sure the man had displaced his sacroiliac joint, but a manipulation did nothing to relieve his symptoms. So the physician directed ⊙

that a plaster of Paris jacket be applied to keep the patient's spine fixed.

As he was being moved in bed for the application of the jacket, the patient "felt an intense pain followed by a quick relaxation". For a happy moment, Goldthwait thought that the patient's sacroiliac joint had been relocated. But then, "gas escaped from the bowel, there being a complete paralysis of the rectal and bladder sphincters, as well as complete sensory and motor paralysis of the legs".

They turned the man on to his left side and, "in a few moments power and sensation began to return in the legs so that within two hours the use was normal, and for the sensory disturbance there was left only a slight numbness in the dorsum of the right foot". As he was sleeping, however, the patient rolled twice on to his back and exacerbated his radiculopathy, resulting in "much pain of an explosive or lancinating character" in his legs.

After six weeks, an exploratory operation could find nothing to explain the patient's condition. Fortunately, he started to improve soon after the operation and made a reasonable recovery.

Goldthwait pondered the case. Perhaps because he was used to diagnosing and manipulating "slipped" sacroiliac joints, he thought that maybe the patient's facet joint had slipped, causing a kind of one-sided spondylolysthesis. As a consequence, the disc too would separate from the bone which, Goldthwait surmised, would allow the high-pressure nucleus to escape outward "and project beyond the edge of the vertebra [...] the result must be

that the detached portion of the disk is crowded backward and must narrow the spinal canal".

Although the 1911 observations of Middleton & Teacher and Goldthwait were astute and their conclusions approximately correct, such isolated case studies proved little about sciatica or disc herniations generally.

In 1929, Dandy went one step further. He described his findings from operating on two cases of cauda equina syndrome. On opening the back of the dural sac and viewing the cauda equina, he noticed "a sharp knuckle of the roots protruding backward [...] Palpation revealed a hard tumour lying beneath the roots. The roots were then retracted to the left, and a bulging tumour mass was seen. It was round, about as large as a big hazelnut [...] and entirely covered by dura. [...] After some hesitation I thought it advisable to incise the dura, and much to my surprise a border of loose cartilage protruded through the opening." On closer inspection, Dandy found the cartilage was in fact disc material.

Dandy showed that disc herniations cause cauda equina syndrome and sciatica. But disc herniations were still considered mere curiosities. Surgeons would continue to perform discectomies on "enchondromas" not realising that they were removing the disc. It was not until the 1930s that Mixter & Barr would show that it is disc herniations that are the most common proximal cause of sciatica (Mixter & Barr 1934).

At last, the breakthrough

In 1930, a 25-year-old man fell while skiing and twisted his back. Soon after, he felt a radiating pain down the back of

his left thigh into his calf. After several months of recumbency on a Bradford frame, the same restricting structure used for people with polio, the pain had not improved. The patient's physician, Joseph Barr, referred him to Massachusetts General Hospital, under the care of the surgeon William Mixter. Mixter performed a laminectomy from L2 to S1 and found, at S1 (figure 2), what he described as "evidently an enchondroma" (Parisien & Ball 1998).

Barr pointed out, however, that the patient's pain had not developed insidiously as one would expect with an enchondroma, but had come on suddenly after his skiing trauma. Mixter & Barr investigated this discrepancy by reviewing the biopsies from all of Mixter's previous patients who had been diagnosed with enchondroma. To their surprise, they found that most of the lesions Mixter had excised consisted of disc material.
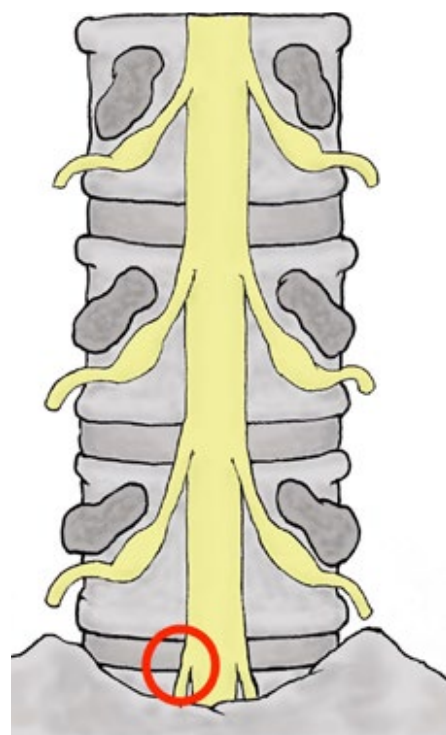

FIGURE 2: Posterior view of the thecal sac. The red circle indicates the location where Mixter & Barr found the first identified disc herniation. This paracentral location is the most common place for lumbar herniations (tomjesson.com/thebook)

On publishing their findings, Mixter & Barr (1934) wrote that "the enchondroma is a well-recognized lesion to be

> **"WHEN A DISC HERNIATES ITS INTERIOR CELLS, ENZYMES AND INFLAMMATORY CYTOKINES SPILL ON TO A NERVE ROOT AND CAUSE IT TO BECOME INFLAMED"**

treated by excision and with a distinctly favourable prognosis […] investigation of these cases of spinal cord tumour […] has shown a surprisingly large number of these lesions, classified as chondromata, to be in truth not tumours of the cartilage, but prolapses of the nucleus polposus or fracture of the annulus".

Their discovery quickly became common knowledge. In 1938, Love & Walsh wrote that "it may be safely said that, today, protrusion of intervertebral discs constitutes one of the major causes of sciatic pain". In 1952, Armstrong wrote that "the diagnosis of a 'prolapsed' or 'retropulsed' intervertebral disc is so commonplace that it is difficult to remember that the condition thus described has only been recognised during the last 17 years" (Armstrong 1952).

Thus began the "Dynasty of the Disc" (Allan & Waddell 1989). Although Mixter & Barr (1934) had written that "treatment is surgical" they did not anticipate just how popular that surgery would become. In time, they both became concerned that their colleagues were too aggressively resorting to the laminectomy. Of their first patient, Mixter would later say with wry affection, "he is the man who started all the damn trouble" (Frymoyer & Donaghy 1985).

'Some other cause besides': beyond compression

Mixter & Barr assumed that disc herniations cause sciatica because of root compression. But, as the Swedish physician Olov Lindahl and others would point out, this couldn't be the whole story (Lindahl & Rexed 1951). After all, everyday experience tells us that when a nerve trunk is compressed the immediate consequence is numbness,

weakness and pins and needles (think of sitting awkwardly or falling asleep on your arm), not prolonged, sciatica-like pain. Lindahl (1966) noted that, when compressed by tumours, nerve roots often behaved the same way. "Since pressure on a nerve root does not necessarily involve pain", he wrote, "it would seem reasonable to suppose that the sciatica [caused by disc herniations] must be due to some other cause besides."

To find this "other cause", Lindahl & Rexed (1951) studied 10 nerve root biopsies taken during operations for sciatica. In seven of the 10 they found evidence of inflammation. Where had this inflammation come from?

One explanation, first proposed in the 1960s, was that disc herniations caused inflammation by inciting an auto-immune reaction (Di Martino *et al* 2013; Sun *et al* 2020). In adults, there is no blood supply to all but the outer few millimeters of the intervertebral disc. This means that, similar to the insides of the testes or the eyeballs, the inside of the disc is cut off from our immune system. Gertzbein (1977) wrote "since the isolated tissues are considered 'foreign', an immune response is mounted in the form of a chronic inflammation."

The auto-immune explanation received early support from an experiment in which researchers extracted nuclear material from rabbits and applied it to the animals' ears, which quickly became inflamed (Bobechko & Hirsch 1965). In another similar experiment, researchers harvested nuclear material from a human cadaver and injected it into the preserved lung of a guinea pig (Marshall *et al* 1977). They observed a "severe reaction" of bronchoconstriction

and edema in the lung. They then tried the same thing on preserved guinea pig intestines, which reacted by constricting repeatedly. To test their theory that these events were caused by an autoimmune reaction, the researchers measured serum levels of circulating antibodies in their patients with disc herniations. What they found also pointed to an auto-immune reaction.

Another explanation for how discs cause inflammation is that it is endogenous to the disc (Jones *et al* 2008; Molinos *et al* 2015; Phillips *et al* 2013). Although the interior of a disc has no blood supply, it is metabolically active, with destructive cells and enzymes that break down waste tissue, and inflammatory cytokines that build up as by-products. When a disc herniates, these irritants spill out on to a nerve root and cause it to become inflamed.

As an example of this endogenous theory, Saal and colleagues harvested disc material from five patients undergoing discectomy (Saal *et al* 1990). They found "extraordinarily high levels" of the enzyme phospholipase A2, which is also found in the venom of snakes, insects and spiders, and triggers the inflammatory cascade that includes COX1 and COX2.

Both the auto-immune and endogenous explanations seem to play some part in how disc herniations cause nerve root pain. But we don't yet know which one, if either, is dominant. And we should not forget that even very slightly prolonged compression will cause nerve root pain by depriving the nerve of oxygen (Olmarker 1991). It is, however, clear that Lindahl was right that pressure on a nerve root cannot explain the full clinical picture of sciatica and that it must be due to some other cause (Lindahl 1966; Mulleman *et al* 2006) (figure 3).

Our changing understanding of why discs herniate

Besides the issue of how these lesions cause pain, the other mystery surrounding disc herniations was why they happen in the first place. ⊙
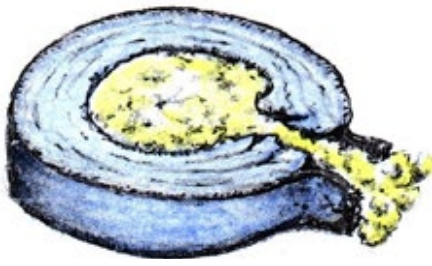
FIGURE 3: Artist's impression of a herniated lumbar disc. This herniation is an extrusion, as the 'head' of the herniation is wider than the 'neck' (tomjesson.com/thebook)

At first, although many physicians were careful to emphasise how little they knew (Nachemson 1960), the dominant assumption was that herniations are a form of injury. Love & Walsh (1938) wrote that "it is probable that repeated trauma may be necessary in many cases to produce sufficient protrusion of the nucleus polposus to cause clinical symptoms". In 1952, Newman proposed the "sprung back" theory, according to which the posterior ligaments are a weak point in the spine and that when they fail, the spine over-flexes which tears the disc open.

It was impossible to ignore the fact that, as studies in the 1950s began to confirm, most people who have disc herniations cannot actually recall any trauma. Naylor (1962) reported that "70% - 80% of patients give no history of injury" and "it is reasonable to assume that injury is never the sole causative agent and, when evident, is only an additional factor".

Instead of injury, physicians began to consider herniations as one consequence of "disc degeneration" (Friberg & Hirsch 1949), the name they had given to the process by which many discs become yellowed, dehydrated and fissured. They considered degeneration not a macrostructural injury, but a microstructural disorder, perhaps a cartilaginous disorder (Naylor 1962). More recent understandings of disc degeneration state that it occurs when cells and enzymes in the disc behave in an aberrant and disregulated manner (Pinheiro-Franco et al 2016). In time, the disc degeneration paradigm so comprehensively displaced the injury paradigm that "some advocates of

degeneration appear to deny the possibility that intervertebral disks can be injured at all!" (Pinheiro-Franco et al 2016).

The causes of disc degeneration

Degeneration was first thought to be a consequence of age and over-use. This changed with a series of studies in the 90s. The most well-known of these examined the spines of Finnish twins to find whether their appearance on MRI was mostly explained by their occupation, hobbies and lifestyle or by their common genetic inheritance (Battié et al 2009). In the analysis, 61% of the variance in disc degeneration in the upper lumbar spine, and 32% in the lower lumbar spine, was explained by heredity. Leisure time and physical loading explained next to nothing. The studies also produced a series of memorable photos in which twins who had taken very different occupations – journalist and farmer, programmer and plumber – nevertheless had very similar-looking spines. Battié and colleagues (2009) wrote that "disc generation appears to be determined in great part by genetic influences". Others argued that Battié's conclusions were too strong and that it was a misuse of their results, which measure the observed variance between participants, to make causal claims (Walls et al 2019).

In their aim to understand why discs herniate, researchers first focused on trauma and injury, then age and loading-related degeneration, and then genetically determined degeneration. More recently, researchers have re-integrated the role of injury; in fact, it appears that discs can herniate as a consequence of injury, whether traumatic or gradual, without ever

degenerating. As a consequence, there is no simple narrative of why discs herniate, and different researchers emphasise different factors. It is safe to say however that the common underlying theme of all explanations of why discs herniate is that, because they don't have many blood vessels, nerves or cells, discs are not that good at recovering from stress compared to most other tissues. This means that as discs are weakened, whether by injury, degeneration, age, or bad genetic luck, nuclear material is allowed to nose through the annulus unchecked until it emerges as a herniation (figure 4).
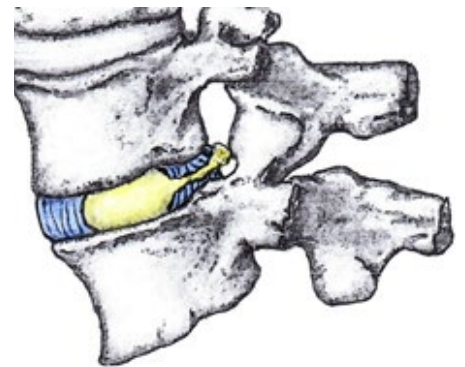
FIGURE 4: Lateral view of a disc extrusion (tomjesson.com/thebook)

'The man who started all the damn trouble'

Researchers have made a lot of progress since the 1900s in their understanding of why disc herniations happen and why they hurt.

Although Mixter & Barr's discovery did usher in some of the worst excesses of "The Dynasty of the Disc", it also allowed surgeons to refine their laminectomy and discectomy technique, which to this day remains one of the most effective interventions

> "THE 25-YEAR-OLD SKIER SEEN BY MIXTER & BARR IN 1930 WAS, IN 1985, DOING WELL WITH NO LOW BACK OR LOWER LIMB PAIN AND WAS PERHAPS THE LONGEST FOLLOW-UP IN MUSCULOSKELETAL MEDICINE"

for pain (Bailey *et al* 2020; Peul *et al* 2007). It also allowed therapists to move away from bed rest and bracing and towards a more sophisticated understanding of radicular pain that accounts for the fact that disc herniations are a form of spinal injury that can cause nerve pain and nerve damage, while also accounting for the fact that such herniations often resorb with time (Chiu *et al* 2015) and are not, on average, made worse by tolerable exercise (Jesson *et al* 2020).

In 1985, the physician John Frymoyer reported on the case of a 75-year-old man whose gait had become unsteady (Frymoyer & Donaghy 1985). The man's case would hardly have warranted a write-up except that, 50 years prior, he had fallen while skiing, twisted his back and, after a number of months, undergone an operation at Massachusetts General Hospital to remove what his surgeon thought at first was an enchondroma. The man was the injured skier in whom Mixter & Barr had found the first confirmed disc herniation. "There is no doubt", wrote Frymoyer, "that this patient is the first one in whom Mixter & Barr diagnosed the lesion now known as a herniated intervertebral disc."

Unsteady gait aside, the patient was doing well with no low back or lower limb pain. This, perhaps, is the longest follow-up in musculoskeletal medicine.

Summary

Our knowledge that disc herniations cause radicular pain, not only through compression but also through chemical irritation, allows us to look beyond surgery as the only treatment option and begin to explain otherwise confounding presentations such as people who have radicular pain, but with little or no apparent herniation. Finally, the finding that herniations occur not only because of injury but also because of age, microstructural degeneration and genetic bad luck allowed us to move beyond simplistic "protect your spine" advice and educate our patients that load, within reason, is good for discs.

CONTACT DETAILS
@thomas_jesson
tomjesson.com

About the author

Tom Jesson is a physiotherapist from Northumberland, currently living in Houston, Texas. He has a special interest in radiculopathy and is the author of *The Clinician's Guide to Sciatica*. More of his writing can be found at **tomjesson.com**

Tom's book, *Sciatica: The Clinician's Guide. Book 1: What Is It,* can be purchased at **tomjesson.com/thebook**

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Bodies of evidence?

JULIAN BAKER

Director of Functional Anatomy Ltd

This article examines how anatomy and the world of the anatomical sciences has changed little in 300 years and suggests that what is being taught to medical professionals is outdated, reductionist and missing elements that are regarded as problems for healthcare within an allopathic model. With examples of how human anatomy is missing vital detail relevant to physiotherapy, this article proposes a radical change in the way anatomy is taught and considered by healthcare practitioners from all disciplines.

LEARNING OUTCOMES
TO SUPPORT PHYSIO FIRST QAP

1 To introduce an anatomical paradigm and present a variation on current thinking.

2 To consider how relatively narrow and limiting anatomical thinking influences research methodology and thereby diagnosis and treatment.

3 To propose a broader and more functional and holistic view of traditional anatomy and encourage a more logical and scientific perspective within anatomical teaching.

The paradigm shift

Until the early 1970s, the biggest watch makers in the world were the Swiss (Anon 2020). Famed for their precision, craftsmanship and quality with the mainspring being the heart of the product, the Swiss were comfortable and confident of their place at the top of the horological hierarchy, controlling more than 50% of the world market and employing 1,600 watchmakers.

Just 10 years later the number of watchmakers in Switzerland had dropped to just 600 and the Japanese became the biggest watchmakers in the world. The attitude of the Swiss was simply that there was no alternative to

what they had been doing for hundreds of years and that nothing needed to change. This was in spite of all the evidence that was mounting up and knocking at their door. Sometimes it pays to embrace a new order if the old order is to survive.

The world of anatomy is an overwhelmingly academic one. It is not a profession that anyone can simply decide overnight to enter. Where anatomy teaching with cadavers or dissection is concerned, the field becomes even more limited and niche and the opportunities are harder to come by. The practice of human dissection in the UK is confined to medical schools and regulated by the Human Tissue Authority in England and Wales (**www.hta.gov.uk**) and by the Inspector of Anatomy for Scotland in Scotland (**www.gov.scot**). Medical schools teach anatomy to those engaging in medical science degrees, and it is taught following a standard curriculum with specific texts that have changed little in 200 years.

It must be considered that, of late, the teaching of anatomy might be facing something of its own paradigm shift with new methods and technologies that leave cadavers and dissection behind (McLachlan & Patten 2006). Where it has not wavered even slightly is in whether the current content that is disseminated is lacking, flawed or outdated and

whether it accurately reflects a new generation of those outside of medical schools, fascinated by the body and human anatomy and eager to take a different perspective of the human form.

Instead, the world of anatomy pushes on with its own version of the mainspring and, like the Swiss watchmakers, considers itself to be complete and therefore untouchable by the future or the outside world. Some medical schools have more recently opened their doors to others in various health professions such as massage and movement therapies, but there is an open resistance from the anatomy world to this, and a declared feeling that the dissection room should be open only to "medical professionals". Anatomical dissection in the UK is overseen by a small cabal who all know each other and it is therefore not hard for those who are deemed unworthy of admittance to be excluded. Even where access is granted, the approach to anatomy is still not adjusted to fit the student, but runs along the same standard anatomical methods that are taught to medical students. It is this attitude that I perceive as a problem.

While there is a wealth of potential donors for the study of anatomy and therefore no shortage of material from which to expand ideas and build new concepts on this subject, many who make the offer are rejected by UK universities for a whole host of reasons. ▸

These can include body size, the number of surgeries the potential donor has undergone, and excessive scar tissue. Such donors are deemed unacceptable because they lack uniformity, and any form that is not represented in the teaching material is deemed to risk throwing a "curve ball" at the student and the teacher alike. Scotland alone rejects almost half of those who offer their bodies for medical study (**www. gov.scot**). The purpose to which donations are used is specific and narrow and, for the manual therapist, turning away any donor who does not accurately represent the anatomy curricular represents an encyclopaedic level of lost opportunity to study and chart areas such as the effects on connective tissue of long-term scarring, and ageing. The loss of this potential to benefit human health is both endless and heartbreaking.

The chicken and egg

The representation of anatomy then becomes the next stage in the problematisation of the human form. The anatomist in a dissection lab does not generally undertake dissection. Prosectors prepare cadavers that have been selected as good candidates for anatomical teaching and prosect according to the specification of the head of department. They cut away the bits that are in the way of what needs to be taught by the anatomists, leaving behind the parts nominated.

This modelling process then creates unrealistic and impossible ideas that get repeated often enough for them to become a truth. An anatomist then becomes a high priest of the half-truth, perpetuating a series of myths that then become the status quo. Taken as

anything other than a starting point or an outline, these myths are unhelpful if presented as fact. Simply put, classical anatomy is incomplete at best and potentially harmful at worst

Once no-one questions anything or suggests an alternative view, reality becomes even more extenuated and researchers, taking on the mantle of anatomical actuality, study the individual parts that have been created, imputing meaning on them as separate structures, capable of independent function and with the ability to fail or become injured without reference to other tissues. Diagnoses, treatments, syndromes, therapies, exercises, equipment, workshops and entire approaches then get based around what is essentially a manicured and sculpted model that is missing much of what makes it functional in the first place.

It's worth bearing in mind that this is not the fault of the anatomist. They have been informed by what they are teaching, and teach what they have been informed by. An anatomist therefore knows a huge amount about the human body but, by necessity, needs to know nothing whatsoever of the human condition or humanity itself.

The cadaver contributes nothing except the anatomy in its possession. The behaviours, habits, functions, movements or lifestyles that have formed it are not considered or asked about. A cause of death may be given or availed of, but function in life holds no interest for the anatomist after it.

The act of dissection in anatomical study is rarely undertaken by undergraduate medical students as part of their

course. Where it is, the process is proscribed and defined by books such as *Grant's Dissector* which, according to one reviewer is "a vindication of the irreplaceably tried and true method of gaining anatomical knowledge" (Sperber 2006). In other words: "Cut here, tie here, reflect here, study this, do it the same way it's always been done and pass the exam".

Testing acquired anatomical knowledge in the dissection room comes in the form of "stations" where, for instance, the upper limb might be the subject. Upper limbs dissected at various stages with pins in them are arranged around the room and students are required to identify pinned structures with a certain amount of time allocated. An understanding of how these parts might relate to each other is passed by and the cycle is perpetuated.

Science: anatomy's missing link

It is the job of science to adapt and change as new information becomes available, and it ceases to be operative, functional or fit for purpose when it ignores that information or neglects to seek it out. Modern anatomy is a museum piece and its teachers are curators of a history that is no longer fit for purpose and can hardly be recognised as one the basic sciences. If the same anatomy is to be taught over and over again, then the debate on whether cadavers are needed is moot. A simple 3D synthetic model will indeed suffice if texture, context and related anatomical palpation and structural relationships are not to be taught.

With anatomy being one of the foundation stones upon which all modern medicine rests, its failures as well as its successes bear considering. Heart surgery, a life-saver in modern times, could not have been achieved without an anatomical foundation.

Back pain, however, presents as a bigger problem in an ageing population and costs society more (Hoy *et al* 2014).The study of anatomy has stood still and its status as a science has diminished (Dyer & Thorndike 2000). The fundamental failure of the basic understanding of back pain is rooted in, and maybe even perpetuated by, the consistent denial of structural relationships in the human form, and by conveniently ignoring of the nuisance that is basic physics.

The human head weighs in the region of 6kg. Standing upright and moving it around on its axis changes the load of this weight through the entire structure of the human form, in turn creating load transfer. Do this on a bicycle, a trained horse or a pair of skis and the direction of travel can be altered. It stands to reason and physics that a head held in a position away from the midline will create a different load through structures underneath it and that these structures will, in turn, require a different contractile input in order for function and balance to be maintained. Such is the simplicity of this model that it forms the basis of how you might teach a five-year-old to ride a bike, yet it has no basis in simple assessment of knee function prior to surgery, and is barely alluded to in many functional assessment or gait analysis protocols.

Crossing over

When considering medicine as a whole, it could be argued that medical specialisation removes the need to consider a more holistic model or wider implications. This, however, creates an unwieldy model, whereby each presenting issue needs specialist consideration, creating a logistical and interminable merry-go-round where opinion and science are often divided and rarely agree.

The anatomists are the servants of medicine rather than the other way around (Orsbon *et al* 2014). It is medicine that tells the anatomist what to teach to fulfil its responsibilities, instead of the anatomist informing medicine of where it is missing knowledge. Medical specialism will determine what it needs from anatomy to make it more successful and create the impetus for curriculum design (Orsbon *et al* 2014).

Extending the circle

Should we be teaching the same anatomy to physiotherapists as we do to undergraduate medical students? The end results of the two learning paths are vastly different, and the need for one to step away from the classical "basics" and develop a more functional approach seems evident. While joint based biomechanical approaches are well established at BSc level education, the principles employed are still rooted in a regional anatomy curriculum that makes little sense and is limiting.

In trying to establish an "elevator pitch" to describe the problem, I use breath as an example that demonstrates the disconnection. The physical act of breathing is a remarkable physiological process and a tool that most physiotherapists will use daily in practice settings. Breathing to move, breathing to soften, breathing to allow for change, increasing blood flow, bringing attention to movement, resistance, stretch, mobilisation, hydrotherapy, as an assessment tool; the list is endless and affected by countless clinical elements (Han *et al* 2016). The descriptive anatomy and the visual power of inflating lungs, their effect on thoracic and abdominal organs and muscular structure and the ability for breath to be transformative, even at the most prosaic level, is a powerful and useful tool.

Learning to breathe, whilst related, is not the same us understanding respiratory physiology (Bintley *et al* 2019). Physiotherapy training and resources rely almost entirely on current anatomical models within basic training, and clinical anatomy resources constructed from standard anatomical teaching models

remain important reference tools when moving into outpatient settings (Farrell *et al* 2015).

Bigger is not better

Fat is not a friend to the anatomical dissector and overly large donors will be declined, as fat gets in the way of where a dissector needs to get to and there is a belief that it has little physiological or functional relevance. This is something that is held true for other connective tissues (Pratt 2019).

The fatty layer, the adipose, consists of adipocytes or fat cells held in place by a strong network of collagen fibres. Once we reach adulthood, adipocytes numbers are relatively static, but they expand and contract according to the variable energy changes experienced. The collagen fibres respond accordingly. It is unclear whether they physically grow in terms of fibre length or unwind when under tension but they have the ability to rapidly adapt according to environmental change.

However, these fat-holding fibres are not just a "layer". Instead, they blend in to the underside of the skin and become the fibrous element of the basal layer. When they move inwards towards the muscle, they meld seamlessly into the layer known as epimysium or deep fascia. It is this fatty layer, referred to by a variety of names, such as the panicular layer and the superficial fascia that is changeable and problematic for scarred tissue. It can create restriction and pain that will often be interpreted by patients as being deeper than it is, and it is familiar to the therapist when palpating as it feels lumpy and complex under our hands. Once it is removed, the smooth, striated fascia and muscle is alien to the touch. It is in this area that I believe we experience the phenomena known as muscle knots or trigger points, a principle rarely considered even amongst the trigger point sceptics (**www.PainScience.com**).

The separation and study of layers tends to be selective and, while the principle of cross-sectioning is hardly new, when it comes to considering function more widely, it is rarely employed to explanatory effect. ⊙

When considering the thoracolumbar fascia (TLF), there is a wealth of material and studies that examine everything from its histology to its location and where it begins, but the fibres of the superficial fascia are always conspicuous by their absence (Willard *et al* 2012). Yet, as we can see from figure 1, tensional fibres situated in the adipose region, between the skin and the deep fascia, are clearly distributed down and through the next "layer" of the TLF. An interesting "pocket" of tissue connects to the iliac crest and forms strong bonds across it and onto the gluteus medius.

If and how these formations contribute to stability, function or pain remains to be seen. There is, however, no doubt that they must have a functional role to play as they are there and, given the enormity of their presence, the fibrotic nature of their presentation and the strength of their attachment to bony landmarks, they must surely merit the same consideration and understanding as any other structure in the body.

The iliotibial band and swan carving

My friend John Webster, a skilled massage therapist, also dabbles in carving ice with a chain saw. Looking in awe at a carved ice swan, I asked him
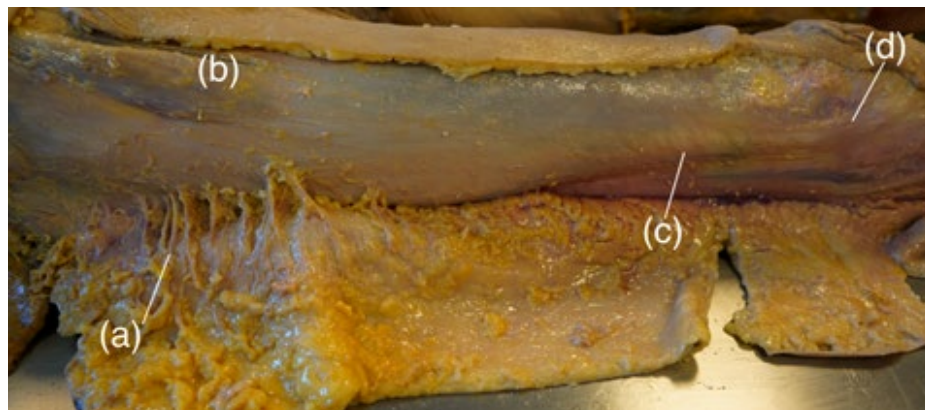

FIGURE 2: A dissection of the left leg showing (a) = collagen fibres of superficial fascia blending to become fibres of the fascia lata. (b) = Vastus lateralis is encased in a bag inside this. The iliotibial band will only be revealed when the leg is trimmed and the ITB fibres left behind. (c) = the guide of where this might happen can be seen in the thickened area. (d) = the ITB fibres diverge at the tibia and continue inferiorly, medially, and posteriorly, forming the external fascia of fibularis longus, the crural and anterior tibial fascia and in most cases, the extended fibres of plantaris, as well as many others (© Julian Baker 2020)

how he did it. In his sardonic Californian drawl he replied: "You just take a block of ice and cut away everything that doesn't look like a swan."

An iliotibial band (ITB) is made in much the same way. Cut away everything that doesn't look like an ITB and leave the rest. From there we can create any story we like. Syndromes, assessment techniques, foam rollers, research papers (Shamus & Shamus 2015), and weekend workshops. All for something that only exists because of the dissector's scalpel and a book illustration.

The thickening of the connective tissue through a lateral part of the fascia lata is not in question. However, the illustration and description of the structure, while occasionally referring to the tissue as a whole, consistently fails to consider any role of the crossing and weaving fibrous tissues that give the ITB its integrity (figure 2).

The hamstrings: keep doing what you've always done

Sports injuries tend to be repeat injuries. Even when considering "new injuries" the ability to assess overall function is limited. Localised treatment of specific injuries in regionalised areas is supported by the classical and localised model with little consideration of how any given injury will impact on other systems or functions.

A person who spends five days limping is teaching their functional system a way of behaving that is very quickly going to be part of who they are and how they move. A therapist who is unable to identify this new pattern and address it when treating the patient may unwittingly be prolonging the problem and allowing new ones to emerge.

A good example that combines the repetition of injury with the construct of a poorly represented anatomical structure is the hamstrings. The

FIGURE 1: The upper part of left gluteus maximus. (a) = the remains of loose areolar connective tissue (superficial fascia) lying on top. (b) = the skin of the lower back that has been removed and (c) = the strong fibres that connect superficial fascia through to the underside of the skin can be seen blending into the thoracolumbar fascia and the (hidden) fascia of the gluteus minimus. Some adipose has been scraped away to show the fibres more clearly (© Julian Baker 2020)

definitions of the grouping are set in stone, yet a different dissection approach reveals tissue connections that significantly change how we might widen our view of this structure (figure 3). The implications are staggering and should have contributed to a radical shift in the consideration of the anatomy of the hamstrings and how they are assessed, treated and described (Pérez-Bellmunt *et al* 2015). With only five citations listed in the citation database Scopus (**www.elsevier.com**) in five years, this has yet to create the impact it should.

Conclusion

When we study the body we have to start somewhere. Naming as many parts of the body as a point of reference, and understanding them as a process of function is a reasonable place to start but a terrible place to finish as it fails to translate the human body into the human being. The missing link in our approach to health from a medical perspective is something that we all understand and seem to accept, as if excusing medicine from needing to accept reality. This is demonstrably harmful. The sturdiness of any structure is based on a solid foundation and medical science is no different. If the foundations are missing essential parts, then everything set on top of them becomes unstable.

When you follow the trail to where the fault begins, it is to be found in the perpetuation of an anatomy that omits more than it teaches and an arrogant complacency that ignores the rest. The mainspring remains an integral aspect of a beautiful watch and has not
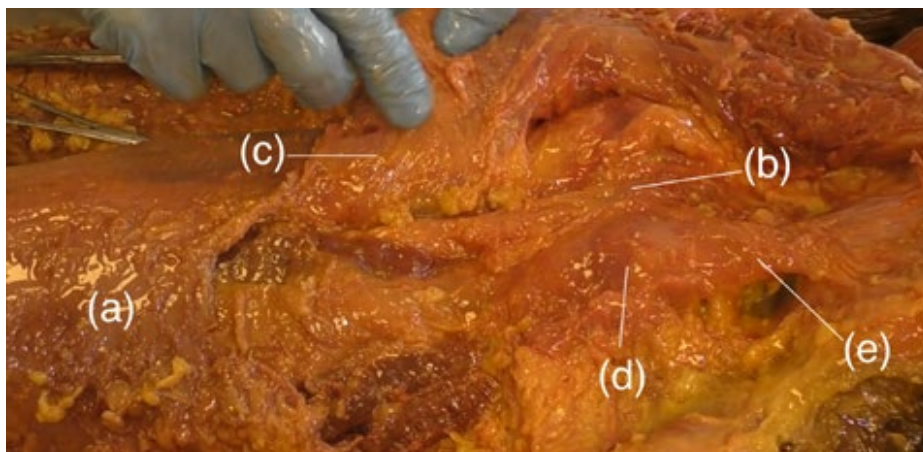

FIGURE 3: Shows a pre-dissected area of the hamstrings. (a) = the fascia lata has been cut on its upper portion and (b) = the path of the sciatic nerve can be clearly seen. (c) = the anterior fascia of gluteus maximus forms a lateral band over head of the (d) hamstring grouping and the (e) ischial tuberosity, demonstrating the potential capacity of the fascia to transfer load and information to from the lower limb

💬 QAP REVIEW

The Paradigms of what is accepted and relevant anatomy knowledge have been set in stone now for some time. This institutionalisation of knowledge has sometimes been to the detriment of knowledge advancement, as new ideas surface they struggle to gain mainstream acknowledgment. In clinical practice, how many diagnostic terms have changed over the years to reflect the advancement of our understanding, sometimes even in conjunction with a new appreciation of how global (dys)functional movements or abnormal loading create these 'pathologies'? This process of recognising the evolution in our learning has enabled our treatments and outcomes to change for the better. For me, this article highlights that 'anatomical truths' can and should be challenged, but only when the counter arguments are underpinned with sound scientific reasoning, inform our clinical decision making and improve our knowledge and patient outcomes.

Reviewer
Tobias Bremer

become obsolete. However, we now understand that it is not the only way to power a watch.

CONTACT DETAILS
julianmarkbaker@gmail.com

About the author

In 1993, Julian was responsible for introducing The Bowen Technique to Europe, and he remains the principle instructor of the College of Bowen Studies, **www.thebowentechnique.com**. He has taught thousands of ➤

students all over the world and, since 2008, has been running dissection classes for manual and movement therapists in the UK and the USA.

From 2007 to 2016, Julian organised and hosted Gil Hedley's dissection classes in the UK and he has taught with, amongst others, Tom Myers and Robert Schleip. He continues to regularly deliver lectures worldwide on anatomical paradigms during which he half-jokingly refers to himself as an Anatomy Anarchist as he challenges conventional ideas on anatomy and questions the myths, language and assumptions that surround manual therapy.

Julian is a member of the Anatomical Society, the Institute of Anatomical Sciences, the Fascia Research Society, and is an "expert" member of the Federation of Holistic Therapists. He has been on the board of the British Fascia Symposium since its inception.

During 2020, Julian ran a six-day live-stream dissection class that examined his approaches and, since April 2020, has created a series of over 20 webinars all of which can be found at **www. functionalanatomy.com**

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Physiotherapy changing lives... setting up a physiotherapy service in southern Rwanda

KATE HUNT MSc MCSP

Musculoskeletal Physiotherapist

Overwhelmingly, the drive for physiotherapists is to help others through our skills and knowledge. In some cases this is extended beyond the clinic and treatment of individual patients to volunteering to help promote physiotherapy for the benefit of wider communities. Here, Kate Hunt shares her experience of being part of a team of who travelled to southern Rwanda to help to set up a physiotherapy service for the community there.

Introduction

From the moment we started working in our first clinics eight years ago, to standing in the foundations of our unfinished building at the beginning of 2020, to now looking at pictures of our up and running purpose-built physiotherapy centre, it has so far been an incredible journey. We now have a 30-bedded building with inpatient and outpatient services up and running. The team and the patients moved in during August 2020.

Our journey

For me, it started with an email from my friend, and now colleague and fellow Trustee, Alison Hawksley asking if I was interested in going to Rwanda with her to do a series of pop-up clinics in the southern province, to explore the possibility of setting up a physiotherapy service for children with disabilities.

Alison is a wonderfully inspiring philanthropic person who I instantly wanted to say yes to and follow anywhere, but I had never been to Africa and like many people I suspect my only experience of Rwanda had been watching the news in my 20s and seeing the suffering the country and its people had endured during the genocide. I had a busy physiotherapy clinic to run, a husband and two children, a dog to walk and a house to look after. The sensible answer seemed to be thank you but no.

In a matter of weeks I was sitting on a plane with Alison, her daughter Brooke and a physiotherapist colleague Jette Jakobsen, heading to Kigali. With advice taken from the Foreign Office, a yellow fever certificate in my hand and a feeling that, at 48, I would never get the chance to take the passion I have for physiotherapy out to a place like Rwanda, I had said yes.

Between us, we had crammed as much clinical equipment as we could into our check-on luggage limit, meaning that I had to pack all that I would need for a two week trip to Africa into my hand luggage. I remember being horrified at that thought for my first trip but, over the years, we have become very adept at packing light for us and heavy for the centre. The equipment in our hold luggage was all donated; from patients, hospitals, schools and friends in the UK, and the continued generosity of people to help others so far away in another country has never ceased to amaze us.

Our first major achievement was to get all the equipment through customs and on to the truck that was to take us on ⊙

FIGURE 1: Jette Jakobsen with the Rwanda physio centre care team (with permission)

our five-hour journey south. To start the day at Heathrow and end in a place so remote we couldn't find it on Google maps was mind-blowing! Although we broke our first rule of "do not travel in the dark" on our first day, leaving the tarmac roads and bumping along tracks that felt like dried river beds, staring out into the darkness lit only by our headlights, was the most amazing experience. Added to this, I had met my new physio colleague Jette only once before we left and, as we drifted off to sleep under our mosquito nets on that first night, we felt like gap year students all over again.

Our patients

The first three pop-up physiotherapy clinics we held were a humbling experience. News had spread of our arrival and in each place that we held our clinics there were queues of people so long they stretched around the buildings we were in. Many of the patients had travelled for hours to see us, and I don't think I have ever loved my job more than I did on that first day. With the help of expert translation from a Rwandan medical student, we found that our physiotherapy skills were immediately transferable.

We have so many memories of the patients we have seen over the years. We all remember the incredible, positive spirit of a severely disabled 18-year-old boy who had spent his life lying in a basket on the floor in his father's tailoring shop. His sister had pushed him many miles in his wheelchair to see us. As he asked if we could help him get better so he could go to school and get an education we all had tears in our eyes.

Another patient with spinal and multi-joint TB did so well with physiotherapy

that, after a couple of years of treatment, she has gone on to become a lead member of our care team.

While there were many occasions that we were able to laugh, there have been times when we have cried meeting patients whose conditions have gone

untreated. We acknowledge over and over again how lucky we are to have the healthcare system we do in the UK. A particularly poignant moment was meeting a family with four sons with Duchenne muscular dystrophy.

There was always a varied start to the clinic day. My youngest patient was a two-week-old baby with talipes, and my oldest a lady of 75 who had a fractured hip and was carried in to clinic on a stretcher by her sons. One young girl attended with a bilateral lower limb oedema and, as we assessed her, my colleague Jette and I realised that we were seeing elephantiasis for the first time outside of a text book.

FIGURES 2-6: Above, a selection of photos of our patients attending clinic (with permission)

Our treatment service

Since we started our clinics in Rwanda, we have seen patients with many different conditions ranging from autism, cerebral malaria, Down's syndrome, hydrocephalus, osteomyelitis, TB, congenital talipes and genu valgum / varum deformities, among others. Our data collection process consistently showed that the most common patient diagnosis was cerebral palsy and developmental delay.

We responded to this by limiting our adult outpatient service in the short term so that we could focus on paediatric inpatient and outpatient services. We started with a small but solid outpatient service nestled into a multipurpose hall owned by our local Rwandan partners, but we quickly realised that, because so many of our patients travelled a long way to see us, outpatient treatment was difficult for some and impossible for most. This drove us to set up an inpatient service and within two years we had 10 inpatient beds. We very quickly became pushed for space, however, and realised that the sustainable future of our physiotherapy service relied upon accreditation from the Rwandan Government. That was when we really understood the project we had taken on was bigger than we had first thought, as we would need a dedicated building in order to be accredited.

FIGURE 7: Fine motor skill group led by the care team therapy assistants (with permission)

PLAY THERAPY

As part of our treatment service, Jette worked tirelessly to introduce therapy through play, and to promote the idea that for our patients their whole day is their therapy. Moving away from the old medical model that treatment is only happening when the child is with the physiotherapist has allowed us to engage our care team and our patients' parents in a totally different way. Our care team understands that getting dressed, eating, washing and playing are all part of the treatment programme, and that they are therapists when they facilitate those activities with the patient. Parents have taken a bit of time to accept this concept but, through this new understanding, we have been able to better support and empower them to know that they are key to the outcome of their child's treatment plan and goals.

By introducing play therapy, we could then move on from a situation where parents wanted to leave their children with us for unlimited months. Patients now stay with us for three-month admissions with varying periods of time at home in between, for which they have home treatment programmes and goals set. Parents and staff have noticed the very positive results of this approach and through this we have built more engaged relationships with the parents of our patients.

Teamwork

We are a small team and we all have our separate roles and responsibilities that ideally link with our individual skill bases, but sometimes the roles we perform are just based on urgent need. We have undergone a steep learning curve in flexibility and resilience, attributes that have been at the top of the list of what has been required of us.

There have also been many practical things to consider in designing our new building such as natural ventilation to provide heating and cooling to accommodate the changeable temperatures of a two rainy seasons climate, provision of toilets and plumbing for many more people, and power supply. Sitting watching the skies during one of the many spectacular electric storms, we quickly learned the importance of lightening rods in protecting the buildings that sit high on the hilly landscapes of Rwanda.

From an organisational point of view, my main role in the team is operational, and that included the responsibility for setting up the physiotherapy service.

Getting started

Setting up, and managing, a physiotherapy centre and service from scratch has certainly been a challenge. Looking back at what we took on from the distance of time, starting one in southern Rwanda is not something that I would have ever thought I would agree to do. However, despite it being an enormous undertaking, we have found that by taking small steps you can achieve great things. One thing has truly led to another and it has felt to be a very organic process.

The starting point was in treating our first patients on the very first trip. Everything about the way we work as physiotherapists made so much sense to me in those early days. Working in such a different place enabled me to view what I do with fresh eyes and to revisit what I had often found very dry and procedural. Our assessment format felt like an old friend, safe and reassuring in a bewildering new world.

Patient notes

A WhatsApp conversation with a new physio in Rwanda who was asking me to direct them to where notes for a patient might be while I was cooking supper for my family in my London made me resolve to change the system. On our next trip to Rwanda, after a week of evenings of working long into the night, we stood back to admire our new filing ⊙

FIGURE 8: Construction of the physio centre (with permission)

cabinet, purchased in Kigali, and our new system of perfectly filed patient notes.

While change is never easy, our colleagues quickly noticed the increase in efficiency that this change brought about and, with the clinic having only one physio and one physio assistant in a catchment area of 300,000 people, we badly needed to maximise efficiency.

It required gentle persuasion over time to change the accepted way of recording daily patient attendance in huge handwritten ledgers, repeating lengthy details over and over. Once we had our new documentation and notes storage systems in place, we were able to move on and introduce much more streamlined monthly statistics sheets, making information about our service more easily accessible. This was a crucial step in enabling us to develop informed policies and procedures.

We are trained to work with good systems at the heart of our practice and over the past eight years I have often felt, and relied upon, the safety net of having this ingrained set of high standards and processes in place. They are essential in protecting our patients and ourselves, and in the provision of a best practice physiotherapy service.

We were also lucky at the time when we were setting up our clinic, that we had a wonderful gift, in the form of UK physio Jessica Edwards who was living

in Rwanda and worked for us on the ground alongside our Rwandan team. She helped us to use the information we had collected to keep much more accurate waiting lists and develop admissions criteria, so we could direct care to those in most urgent need.

This gave us a framework into which we could introduce the gross motor function classification system (GMFCS) and the goal attainment scale (GAS) and these have been hugely important in helping us to prioritise patients and to provide safe staff / patient ratios for our level 4/5 complex patients. With these important cornerstones in place, we were on a roll, and as we moved onwards and upwards we needed to put in place many different policies and procedures such as first aid and resuscitation training, continuing professional development for our staff, appraisals and safeguarding and clinical governance reviews.

Safeguarding

Being in a different country, with different acceptable social and cultural practices, meant that safeguarding had an element of extra challenge. With Jessica's on the ground help we set up focus groups to explore what our own staff thought, and

what safeguarding actually meant to them. We shared experiences of our childhoods and lives, comparing and contrasting the UK and Rwanda. It was a truly bonding experience for both the Rwandan and UK teams to work on this together and develop a robust understanding of what safeguarding actually means on a daily basis for our staff and our patients. Having always had a dislike of only paying lip service to paperwork and processes, I found it exhilarating in Rwanda to be in a situation where processes I had previously found difficult to see the purpose of suddenly showed meaning, and their true value in directing change.

Continuing professional development (CPD)

Our monthly patient data collection is used to inform the in-service training for our care team staff. This involves weekly staff meetings and training sessions that include a safeguarding review of our current patients.

Some lovely and unexpected adventures and successes have occurred as our policies developed. As in the UK, CPD credits are required in Rwanda to retain professional membership. In a remote village, some five hours travelling distance from the capital with fluctuating internet connection, this was difficult and costly.

However, as our UK team was already providing training when we visited, I wondered if we could somehow get our teaching accredited. The Rwanda Allied Health Professions Council (RAHPC) was very encouraging and, following a couple of meetings with them, we set about becoming an accredited provider of CPD in Rwanda, something we achieved in 2018. We were excited to be told we are the only non-Rwandan accredited provider of CPD in Rwanda. We have run accredited courses on-site for our physio team each time we have visited, and ⊙

> *"*GOOD RECORDING SYSTEMS ARE THE HEART OF PHYSIO PRACTICE AS THEY ARE ESSENTIAL IN THE PROVISION OF A BEST PRACTICE PHYSIOTHERAPY SERVICE*"*

have also run two courses in the capital, Kigali, for physios countrywide. This has had the bonus of linking us to the wider physio network in Rwanda and that has helped to build the reputation of the centre. We had seven courses planned and accredited at the start of 2020 that sadly had to be cancelled due to Covid restrictions, but we hope to get these up and running again as soon as we can.

More recently, we were contacted by a Rwandan occupational therapist (OT) who had found us via our website asking for work. We have always known that we needed OT input but up until now we have not been in a position to add another staff member. Through a donation, we were able to give him a six-month internship which has been an immediate success. It has been hugely rewarding to see how specific OT skills can enhance what we do as physiotherapists, and we have quickly witnessed how this has benefitted our patients.

Our future plans

In March 2021, a container full of donated equipment that we had collected over the last few months in partnership with PhysioNet set sail for Rwanda, and the container ship that blocked the Suez Canal quickly became another of the many hurdles we have faced over the past year!

When the container reaches Rwanda, having the equipment safely installed in our clinic will be the last piece of the puzzle that will enable us to invite Rwandan government officials to visit and assess the centre. Accreditation will give us the sustainability we need for the future, and complete this leg of our journey.

Our goal has always been for the centre to stand alone in its ability to self-fund and, with the confidence and skills of the Rwandan team, to be a centre of excellence in Rwanda. At that point, our UK team will be able to step back from day-to-day financial and operational support, but we will stay on as clinical advisors. We will always be there in spirit though and, as we get closer to obtaining our accreditation, I dream of so many possibilities, including setting up a back pain programme, becoming a talipes / club foot specialist centre and having an orthotist in situ. There are always new challenges to be taken on.

Our project joined forces, in 2017, with UK charity Make A Difference for Africa (MAD4Africa) and I am proud to be working with a very dedicated team of trustees who guide our work in health and education. Our projects are completely funded by donations and all UK staff members are unpaid volunteers. We are always grateful for donations of money and equipment and, once the centre is accredited, we will be in a position to offer work for volunteers who are ready for adventure and challenge in a different environment and would like to share their clinical expertise with our colleagues in Rwanda.

About the author

Kate has worked as a musculoskeletal physiotherapist for 35 years, following her graduation from King's College, London. Before setting up her own practice she worked at The Royal London Hospital, St George's Hospital and in India.

While Kate continues to support the running of the physio centre in Rwanda, she balances that with her current role in London, working within two health and wellbeing departments of large news companies in London, with a particular interest in posture and muscular strain related to working with technology. She loves that physiotherapy allows for knowledge, skills and experience to be used in such different settings.

Acknowledgments

FIGURE 9: The care team receiving CPD certificates with (far left) Kate and (far right) Jessica (with permission)

FIGURE 10: The completed new building

First contact and advanced practitioner

AMANDA HENSMAN-CROOK FCSP

HEE AHP National Clinical Fellow, Consultant MSK Physiotherapist

Introduction

The Musculoskeletal (MSK) First Contact (FC) and Advanced Practice (AP) Roadmap to practice in primary care landed in the system in October 2020. This article explores how the Roadmap relates to the private physiotherapist, what opportunities it offers, and how it can help clinicians to develop a route to becoming a AP. It is important to mention at this point that the Roadmap is an educational pathway for all clinicians, regardless of how they are employed, and that all courses, resources and supervision are inclusive.

First contact practitioner explained

The title first contact practitioner (FCP) has created much discussion over the past few years and it can be argued that private practitioners, by the nature of how they see patients directly from their receptions, are already 'first contact practitioners'. However, in the context of the Roadmap, and as a nationally recognised job title in primary care, a FCP is synonymous only in the primary care setting, and with specific knowledge, skills and attributes requirements that are assessed and verified at master's level, both academically and through the application of that knowledge. You cannot hold the title of FCP if you work outside the primary care setting because the scope of practice is wider than MSK, and the FCP has direct access to diagnostics and onward referral.

A FCP is, therefore, a diagnostic clinician **in primary care and working at master's level** with undiagnosed and undifferentiated diagnoses. They will have a minimum of five years' postgraduate experience and manage complexity and uncertainty as the first point of contact. Recognised first contact clinicians will be listed on a first contact

directory held at the Health Education England (HEE) Centre for Advancing Practice, once their verified portfolio of evidence has been signed off at stage 1 and stage 2 in line with their profession specific Roadmap. These stages involve:

Stage 1 = a verified portfolio of evidence of working academically at master's level against the knowledge, skills and attributes document, plus completion of the two sets of e-learning modules, further details of which can be found on the MSK Roadmap.

Stage 2 = a verified portfolio of evidence demonstrating that their stage 1 academic master's–level knowledge has been applied to practice, using the Work Based Placement Assessment Toolkit in the primary care clinical setting. **The first contact practice sits in the transition between the enhanced and the advanced practice**.

The educational pathway is designed to develop the practitioner with the knowledge, skills and attributes that have been verified and signed off in the achievement of FCP, to then continue along the portfolio route towards attaining AP status.

Advanced practice explained

In order to achieve AP status, the FCP will have proven capability, with a portfolio of evidence at master's level and will have demonstrated at least some of the four pillars that are integral to AP and influence every intervention, i.e. leadership, clinical, research and education, throughout the process.

Advanced practitioners are clinicians who are multi-professional (across all professions), cross-organisational (across all clinical settings) and cross boundary (across regions), and who provide multi-professional supervision.

Recognised APs will be held on a directory at the HEE Centre for Advancing Practice.

A PowerPoint presentation that explains the Roadmap was recorded for the Primary Care Rheumatology and Musculoskeletal Medicine Society (PCRMMS) conference, and is available via the website address in the Resources section at the end of this article.

How does this relate to the independent practitioner?

The capability framework for the MSK FCP/AP Roadmap has been developed by using the International Federation of Orthopaedic Manipulative Physical Therapists (IFOMPT) educational standards which are recognised by the UK MSK educational standard of practice.

Research conducted by Birmingham University confirmed that the multi-professional advanced practice framework, and the MSK core capability framework sit inside the IFOMPT standards, which is why these standards are central to the Roadmap to AP.

In collaboration with IFOMPT, the Standards for Advanced Practice have been raised to master's level to be made specific to the UK by including injection therapy, independent prescribing, social prescribing, surgical options and psychological treatment approaches, explicitly across all four pillars of practice. The use of the IFOMPT standards to build the educational pathway means that **the Roadmap can be used as a portfolio route to AP for MSK clinicians regardless of whether or not they want to work in primary care.**

- **For those who want to work in primary care** directly for a primary

care network or employed by a GP practice, the Roadmap stages will need to be followed and signed off with a recognised Roadmap supervisor. Alternatively, the practitioner can opt to complete the FCP master's module for stages 1 and 2, and continue along the portfolio route to advanced level.

- **For those who don't want to work in primary care** they can map to stages 1 and 3 and, once the portal is public facing, submit their portfolio of evidence to the Centre of Advancing Practice. If the portfolio route is via the Centre of Advancing Practice portal, it is not necessary to locate a supervisor to become recognised as an AP, but if the practitioner decides they want to work in primary care at a later date, they will need to find a supervisor and complete stage 2.

How to develop a portfolio of evidence

The Physio First Quality Assured Practitioner scheme is a great way to prove the quality outcomes of your practice, and while PROMs can be used as a basis of a written reflection of treatment pathways, they cannot be used as standalone evidence to map against the portfolio route as they are not proof of capability at master's level. The Resources section lists two videos from Manchester University that explain what master's–level practice is, what constitutes as evidence, and how to build a portfolio and map to the FCP/AP framework.

It is worth noting that those practitioners with full membership of the Musculoskeletal Association of Chartered Physiotherapists (MACP), and who have undertaken the e-learning modules, will already have achieved stage 1 and will have almost completed stage 1. Also, any practitioner who holds a full diploma from the Society of Musculoskeletal Medicine (SOMM), and who has completed the e-learning modules and completed the top-up course, will fully map to stage 1. The SOMM is running a top-up day course that will enable completion of the stage 1 portfolio, full details of which can be found on their website **www. sommcourses.org**.

How to access taught routes

Independent practitioners have the same access as NHS providers to all taught courses, including FCP master's modules, the MACP taught and supported portfolio routes, SOMM and the PCRMMS GP working in extensive roles (GPwER) diploma courses, which also maps to stage 1. Independent providers, as well as NHS staff, are eligible for the HEE funded FCP masters modules and funded AP master's degrees.

Finding a supervisor for the Roadmap portfolio and working in primary care

There will soon be a public-facing page on the NHS HEE website with a national supervisors list and, as the Roadmap supervision is developed, regional supervisor lists will be available. The MSK professional networks will also be able to support individuals to find supervisors. Further advice can be obtained from the Advanced Practice Physiotherapy Network (APPN). Clinicians who hold a postgraduate master's degree are encouraged to attend a free two-day course to become Roadmap supervisors and verifiers in order to supervise and verify other clinicians along the portfolio route.

About the author

Amanda is a Consultant MSK Physiotherapist in Primary Care and a Fellow of the Chartered Society of Physiotherapy. She is currently seconded to HEE as its allied health professions (AHP) National Clinical Fellow leading on the training pathway to first contact and advance practice in primary care and the supervision and governance for the new AHP roles in primary care. She was central to the development of first contact practice from 2014 and created the national data template for standardised data collection for primary care computer systems in England to provide evidence of impact.

CONTACT DETAILS
Amanda.Hensman-Crook@hee.nhs.uk

💬 **PHYSIO FIRST REVIEW**

This is a great one-stop shop for information on the first contact physiotherapy pathway, and detail on what is required of a physiotherapist to reach the required level to become a FCP. Although private practitioners may find it difficult to locate a Roadmap supervisor, it's good to see that we can still register as advanced practitioners. Of particular interest is the major focus throughout the pathway on the four pillars of practice, and that one of these includes research.

Our Physio First Quality Assured status gives us the opportunity to demonstrate that, as practitioners, we are taking part in research as well as auditing our own work through our reports from the University of Brighton. As Amanda describes, a written reflection on our patient reported outcome measures (PROMs) can be used as part of reflection to help map against other areas.

Reviewer
Lucinda Brock

Resources

Advanced Practitioner Physiotherapy Network: https://www.appn.org.uk

Manchester University: mmutube – What is level 7 learning? https://mmutube.mmu.ac.uk/media/1_7ovbkilj

Manchester University: mmutube – Level 7 portfolio, where to start? https://mmutube.mmu.ac.uk/media/Kaltura+Capture+recording+-+January+25th+2021%2C+8A47A30+pm/1_cz6wfb91

NHS Health Education England: Advanced Practice https://www.hee.nhs.uk/our-work/advanced-clinical-practice

NHS Health Education England: What is Advanced Clinical Practice? https://www.hee.nhs.uk/our-work/advanced-clinical-practice/what-advanced-clinical-practice

Primary Care Rheumatology and Musculoskeletal Medicine Society: Powerpoint presentation on First Contact and Advanced Practitioners https://onedrive.live.com/view.aspx?resid=B510487705B1C6C2!1651&ithint=file%2cpptx&authkey=!AK-TNQEAJKBF_LA

Society of Musculoskeletal Medicine: SOMM top-up day course https://www.sommcourses.org/courses/top-up-day/

A view from the Chair

Looking to the future: where next for private physiotherapy practice?

Karen Lay, our Physio First Chairman, looks at what physiotherapists are likely to have learnt from changes during the pandemic

As we reflect on the massive changes the pandemic has wrought on our working lives, and on private physiotherapy in particular, it is tempting to wonder which of those changes we will keep and embed into our practices, and which will fall away as we return to post-pandemic healthcare; whether it will be a 'new normal' or an 'enhanced normal' in retaining the positive changes, while discarding those less desirable, such as social distancing, which have been necessary for infection control. The use of virtual platforms such as Zoom is here to stay, with an impact on all areas of both our personal and professional lives. Many Physio First members have embraced virtual consultations, both for individual and class-based applications, and may continue to offer this option as a client service in addition to face-to-face appointments.

From our client-base perspective, the impact of home working in poor set-ups has resulted in an increase of MSK conditions. Add to this the effects of long Covid and a backlog of deconditioned patients awaiting surgery on an overburdened NHS, and it is anticipated that demand for quality private practice services will continue.

So how might we position our practices to best meet the future needs of the population, and sustain successful practices with a reputation for evidence-based interventions and good outcomes?

One answer might be found in becoming a first contact practitioner (FCP) or advanced practitioner (AP). As Amanda Hensman-Crook highlights in her article on page 48 of this issue of *In Touch*, the Roadmap to Advanced Practice is now live and has a strand examining a portfolio route to advanced practice which may be particularly suitable for private practitioners. While not everyone will want to explore working in primary care, and the FCP role is one of triage rather than treatment, the elements of advanced practice require demonstrated competence in all four pillars, one of which is research. For our Physio First members who are considering applying for FCP and / or working towards AP, our DfI tool and Quality Assurance schemes are the perfect examples of how day-to-day practice can be used to involve research and reflection, as well as identifying areas to address the potential training needs for each practitioner.

In England, the Best MSK Health Programme will be the main vehicle for influencing the national policy direction of the recovery and restoration of MSK services to improve patient outcomes. The NHS White Paper proposes the establishment of Integrated Care Systems in England, with the potential to influence services at a local level, and similar changes may follow for the devolved countries. If, due to the pressure on MSK services, self-management is to become the NHS first intervention of choice, will it increase demand on private practices of all AHPs? An ageing population with multiple co-morbidities will require us, as clinicians, to have a greater breadth and depth of understanding of our patient base in order to help them to achieve good outcomes. This may result in the private practitioner moving from specialist towards more generalist practice, something that I suspect many of our Physio First members increasingly do anyway, as the essence of being in private practice includes an element of not knowing what the next patient's story will reveal, and so it is always necessary to have a broad overview of conditions and pathology outside MSK.

The pandemic has shone a spotlight on the NHS areas of silo working, as well as on potential areas for collaboration. While private sector efforts to engage with the NHS have, in the past, sometimes been met with distrust and anxiety around expertise and competencies, if service provision is moving to a more local specific response, it makes sense to make connections and build a relationship with your local NHS teams. Being a trusted colleague, we can offer options for where NHS teams can signpost their patients for continuing recovery and rehabilitation once NHS provision has been completed. An important element of building trust in these relationships is in demonstrating good outcomes, so Physio First will continue to promote our DfI tool and Quality schemes, and highlight their significance in fulfilling part of the advanced practitioner portfolio.

Physiotherapy UK 2021

At the Physiotherapy UK conference in November this year, we are leading the theme of 'Transforming Provision', and our symposia will be on data collection and Quality Assurance. We also aim to showcase some examples from some of our members who demonstrate innovative practice.

This is another way in which we are able to champion examples of quality, value-based treatment delivered by you, our members, and we will continue to do so wherever and whenever we can.

And finally, calling Facebook fanatics, terrific Twitterers and Instagram sensations. If any of these apply to you, Physio First needs your social media skills and expertise to amplify our online communications. Helping our communications team is as easy as clicking 'like' and sharing our posts on your chosen social media platform, and commenting encourages greater engagement with our message, which is always appreciated.

@PhysioFirstC
@PhysioFirstOrg
@PhysioFirstUK

A message from our membership team

New beginnings with familiar friends and new ones

Our 2021/22 membership year started on 1 April and, considering the year we have just had, our renewal period went really well, with a high percentage of members rejoining. Thank you for demonstrating your continued faith in our Physio First community and the benefits it presents to all of us.

In addition, many of you have taken advantage of our monthly membership option, and we are pleased to welcome more new PAYG members every month. Your input into our community is really appreciated; whether you are attending and engaging in our community Zoom meetings, participating in our online forum, or communicating your thanks and support to our volunteer and office teams for all the timely marketplace information we share in our bulletins.

As a reminder, please do make sure that you are signed up to receive our email bulletins. Important updates are sent out regularly, with an average of more than two a week delivered to our members' inboxes in the past year! So if you haven't been receiving them, contact our office team and we'll get it sorted for you.

We are also seeing an increase in members signing up for our Physio First Data for Impact (DfI) data collection scheme. The fact that more private physios are becoming engaged with the need to collect data and demonstrate quality is brilliant for our profession, and hopefully those who have joined us specifically for this benefit will discover many more benefits from your Physio First membership.

During this next membership year, we will be inviting everyone who would like the opportunity to be part of our volunteer community to become more involved through initiatives aimed at accommodating as wide a range of interests, skills and available time as possible; please look out for our communications to see what might appeal to you. We also have new developments coming within our DfI tool that will be much more than just about meeting Bupa's requirements, and designed to meet other marketplace pressures. We **can** influence our marketplace if we work together.

Thanks a lot for being part of our community. It's great to have you with us!

Richard Hulbert
Honorary Membership Officer

PPEF update

As we all know, physiotherapy plays a major role in the treatment of stroke. You will not be surprised to learn, therefore, that as a charity exclusively supporting the development of physiotherapy research and practice, the PPEF has worked for several years with the Stroke Association, a national charity funding research into the prevention and treatment of stroke, and supporting those who have had a stroke.

The PPEF has attended and contributed to lectures and seminars run by the Stroke Association and funded several physiotherapist-led projects highlighting the various aspects of stroke. Recent projects we have part-funded include one to identify the common treatable causes of early post-stroke shoulder pain, and another is investigating whether high repetition arm strength training undertaken early after a stroke occurs encourages new brain connections.

Information on these and other projects will soon be available on our website **www.ppef.org.uk** which is in the process of being updated and expanded.

More information specifically on stroke can be found at **www.stroke.org.uk**

Gillian Jordan MSc BA MCSP Dip TP
PPEF Trustee

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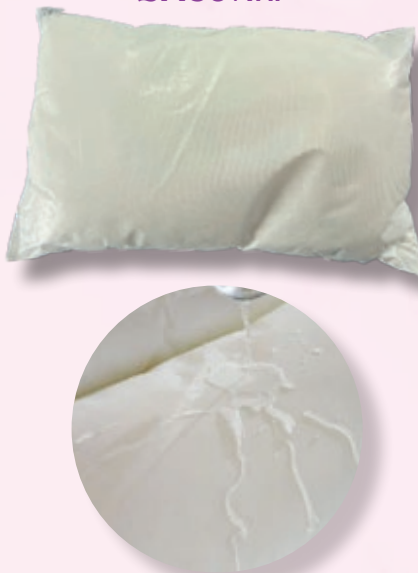
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