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Editorial

Quality Assured Practitioner...

I have pleasure in inviting you to enjoy another cracking edition of *In Touch*. Once again, all our articles are delivered thanks to the generosity of our gifted and giving authors who want to share their knowledge with the private practitioner community and, in doing so, help support our Physio First members who are, or who are working towards becoming, Quality Assured Practitioners (QAPs).

This Physio First initiative makes more sense now than it ever did. Quality may well be the defining factor in the future of private medicine. The June 2018 Private Healthcare Summit (*The Core* July 2018) noted among other things that quality needed to be a major part of the private medical experience; price alone won't cut it, which is something that Physio First has recognised for a long time. Our patients want the best quality care and they know that this is not about price. The experience of best care from the first hello to the last goodbye and the feeling of being cared for in a trusted way from start to finish is driven not by price, but by the desire of our members to provide a quality service.

We are aware that our Physio First QAP scheme does not fit all business models. For those able to participate, there is a personal marketing advantage that could be the edge in our rapidly changing healthcare marketplace, and we hope all Physio First members recognise that this translates as a huge asset for our organisation as a whole. Even if you are unable to participate in QAP, we all need to measure ourselves as clinicians; from patient-recorded experience measures to patient-recorded outcomes measures, all data shows what we do, and why we are doing it. This data may be the defining factor in the choice a patient makes in who they go to for physiotherapy. Personally, I really do want to be the quality-driven, evidence-based private physiotherapy practitioner they choose.

"Adapt or die" might be the adage to use here. We need to be innovative, strong and use the technology available to us, i.e. tweet exercises to our patients' smart watches as a way to encourage adherence and as a quick education nugget to keep them on-board. Evidence shows that it is not one or two individual factors that make the patient's experience a good one, instead it is a rounded package of care that can include education and advice, exercise therapy, hands-on treatment and providing the best context in which the patient can find a route to get better. From the wonderful "hello" and support from the practice front-of-house team, to the room they sit in, to the when and how interaction of the physio team; it **all** matters. Pain is a construct of the brain, so we need to use that knowledge every time we give care.

PAUL JOHNSON | MSc BSc MMACP MCSP | EDITOR

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Editorial Panel

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Sarcopenia: epidemiology, challenges and opportunities for multidisciplinary practice

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Sarcopenia, the loss of skeletal muscle mass, strength and function with age, is widely recognised as a major clinical problem for older people that is associated with serious health consequences in terms of frailty, disability, morbidity and mortality, as well as high health care costs. Recent diagnostic algorithms have provided a systematic approach to case-finding. This article reviews the epidemiology and pathogenesis of sarcopenia and presents an overview of comprehensive geriatric assessment (CGA) as a multidisciplinary-based approach to the assessment, management and follow up for the older patient.

LEARNING OUTCOMES
TO SUPPORT PHYSIO FIRST QAP

1 Be aware of the characteristics of sarcopenia.
2 Understand how sarcopenia affects our aging population.
3 Understand the challenges and opportunities presented.

Introduction

Demographic transformations across the world dictate that survival to older age is anticipated to be the norm for all of today's younger population (Chatterji *et al* 2015). While this is a cause for celebration, these cumulative demographic changes pose significant challenges for delivering health and social care to older people.

Sarcopenia is a particularly deleterious expression of aging that impacts on individuals across a range of health and social care settings, as well as on the consequent health economy (Fuggle *et al* 2017; Janssen *et al* 2004). Sarcopenia is characterised by generalised and progressive loss of muscle mass, reduction in muscle strength and resultant functional impairment. The condition is associated with poor health outcomes from disability and morbidity, e.g. diabetes, osteoporosis and consequent frailty (Gale *et al* 2007; Sayer *et al* 2005, 2007; Ferrucci *et al* 2014; Morley *et al* 2014). It is also predictive of mortality not only in older people, but also those who are middle aged (Cooper *et al* 2010; Studenski *et al* 2011). In 2016, sarcopenia was assigned an official disease classification based on the International Classification of Disease (ICD-10). This reflects the importance of recognising and managing this condition (Cao & Morley 2016).

Skeletal muscle across the life course

Skeletal muscle comprises 40-60% of total body mass, and plays an essential role in both physical and metabolic functioning, e.g. locomotion, thermoregulation, metabolism of glucose and amino acids (Sender *et al* 2016). Muscle is also a reservoir for proteins and energy that can be utilised in periods of stress or undernutrition, such as in any acute deterioration in health and / or hospitalisation.
Muscle development in humans begins at six weeks gestation and continues until approximately 24 weeks at which point the total number of Type I and Type II fibres is set. Any subsequent increase in muscle bulk occurs by hypertrophy (Maltin *et al* 2001; Brown 2014). Muscle mass increases during childhood and adolescence, remains relatively constant in early adulthood, and then is estimated to decline at 8% per decade after the age of 40 up to 70 years, followed by a more precipitous loss of 15% per decade thereafter (Janssen 2011). Loss of strength appears to be of a higher magnitude with a reported 10-15% loss of leg strength up to 70 years, followed by a greater decline of 25-40% per decade thereafter (Goodpaster *et al* 2006; Grimby & Saltin 1983; Hughes *et al* 2001). In this regard, direct proportionality between loss of muscle mass and impaired strength / function cannot be inferred as evidence from longitudinal, as well as cross-

"EXTRINSIC AND INTRINSIC FACTORS AFFECT MUSCLE QUALITY IN AN OLDER PERSON"

sectional studies show that younger individuals can be weaker, and older individuals stronger, than would be predicted by their muscle mass alone (Kallman *et al* 1990; Newman *et al* 2006). Consequently, the health of skeletal muscle in an older person is not only a result of the peak levels they may have attained in mass during their early to mid-life years, but also the extrinsic and intrinsic changes that affect muscle quality, the force generated per unit of muscle area, i.e. patterns of physical activity, nutrition, disease, disuse and hormonal changes that may have taken place during their middle years into old age.

Defining sarcopenia

Accepted definitions of sarcopenia in research, as well as in clinical practice include loss of muscle mass, strength and physical performance, collectively referred to as function (Cruz-Jentoft *et al* 2010). Diagnostic algorithms include those proposed by the European Working Group on Sarcopenia in Older People (EWGSOP) (Cruz-Jentoft *et al* 2010), The Foundation for the National Institutes of Health (FNIH) Sarcopenia Project (Studenski *et al* 2014), and the Asian Working Group for Sarcopenia (AWGS) (Chen *et al* 2014).

The EWGSOP definition is most commonly used within the UK and Europe (figure 1). It incorporates slower walk speed (≤0.8m/s), or weaker strength (grip <30kg for men, <20kg for women) in combination with low muscle mass (defined as appendicular lean mass normalised by the square of body height [ALM/ht^2] ≤7.23 kg/m^2 for men and ≤5.67 kg/m^2 for women). From a clinical point of view, this and other algorithms facilitate sarcopenia case-finding, and can conceptually identify stages of sarcopenia that may allow early detection and intervention. For example, and as illustrated in figure 1, the pre-sarcopenia stage is characteristic of low muscle mass without impact on muscle strength or physical performance, the sarcopenia stage is characterised by low muscle mass, plus low muscle strength or poorer physical performance while severe sarcopenia is when all three criteria within the algorithm are met (Cruz-Jentoft *et al* 2010).

MEASURING MUSCLE MASS

The most common approach in measuring muscle mass is through portable bioimpedance scanning (BIA) and, where available, dual energy x-ray absorptiometry (DXA) scanning. Computerised tomography (CT) and magnetic resonance imaging (MRI) can also be used (Cooper *et al* 2012) but high operational costs and radiation in the case of CT, limits their use. Anthropometric measures such as skin fold thickness are prone to error, especially in hospitalised older people

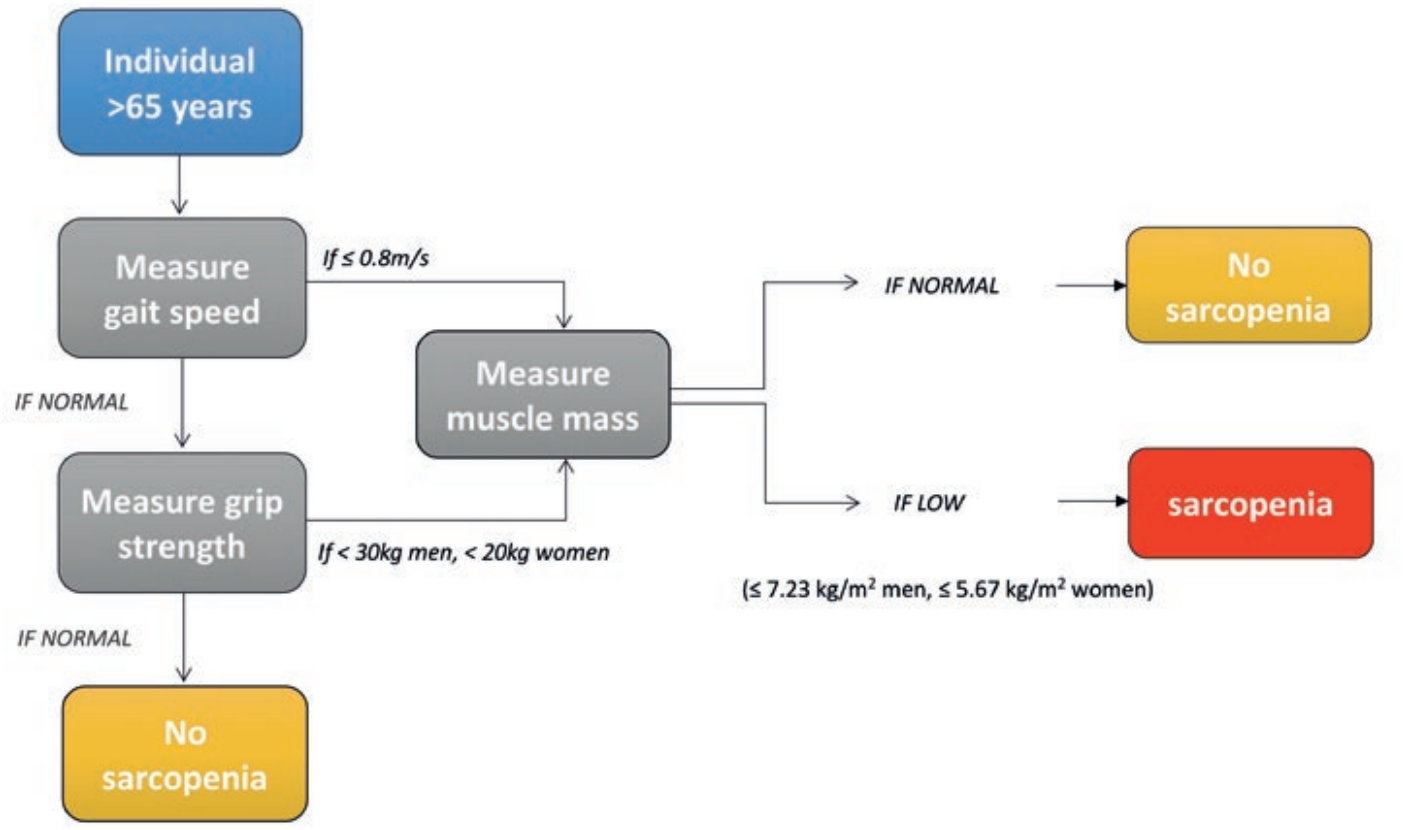

FIGURE 1: The European Working Group on Sarcopenia in Older People (EWGSOP) diagnostic algorithm for sarcopenia case finding adapted from Cruz-Jentoft *et al* (2010)

and are not suitable for assessing muscle mass in this population.

MEASURING MUSCLE STRENGTH

Hand-held dynamometry has gained wide acceptance, across healthcare settings, as a reliable and valid measure of muscle strength (Roberts *et al* 2011, 2012). Other methods include ascertainment of knee extensor power, isometric knee strength and quadriceps torque, but these require static and bulky equipment that can be impractical for use in routine practice.

MEASURING PHYSICAL PERFORMANCE

Slower gait speed is associated with risk of future morbidity and mortality and is therefore suitable for inclusion in diagnostic algorithms for sarcopenia (Vermeulen *et al* 2011). Measurement of gait speed requires intact co-ordination, and neural and joint control so may not be practical in context of acutely unwell, hospitalised older people. In this situation, grip strength measurements may have better predictive value and be more feasible (Roberts *et al* 2012; Ibrahim *et al* 2018).

Prevalence of sarcopenia

While it is clear that sarcopenia increases with age, estimates of its prevalence vary widely in different clinical settings, reflecting divergence in the approaches used for its definition, the ethnicity of the population studied, and the cut-off values for lean mass and function. In their systematic review Cruz-Jentoft *et al* (2014) reported rates of between 1% and 29% in community-dwelling populations and of 14-33% in long-term care residents. In the UK, the prevalence of sarcopenia, using the EWGSOP definition in the Hertfordshire Cohort Study was 4.6% in men and 7.9% in women, mean age of 67 years (Patel *et al* 2013).

QUESTIONNAIRES TO AID THE DIAGNOSIS OF SARCOPENIA

The SARC-F questionnaire was developed to predict poor muscle function (Malmstrom & Morley 2013; Cao *et al* 2014) and is based on five questions that ascertain how much difficulty an individual has in performing the following parameters:

- Ability to rise from a chair
- Walk assisted or unassisted
- Climb stairs
- Carry heavy loads (as a measure of strength).

It also contains an ascertainment on the number of falls a person has had over the previous 12-month period.

Each parameter is assigned a score: none (0); some (1); or a lot (2); the falls parameter score is none (0), 1-3 (1), 4 or more (2). A total score of ≥4 on a scale of 0-10 suggests that the subject is symptomatic of sarcopenia. While the SARC-F questionnaire has been shown to have excellent specificity, it has poor sensitivity for sarcopenia and so may be useful for case identification and subsequent diagnostic evaluation in a community-based setting rather than in hospital or care home based settings (Woo *et al* 2014), and therefore could be of practical value to therapists working in the community.

Despite the recent progress in refining and implementing criteria, at present no global consensus exists regarding the diagnostic criteria for sarcopenia based on cut-off values for skeletal muscle mass indices, grip strength and walking speed. However, ongoing collaborative efforts by the EWGSOP, FNIH and AWGS aim to harmonise the definition of sarcopenia for research and clinical use worldwide.

Pragmatic indicators of whether an individual is at risk of sarcopenia can be useful for the multi-discipline team (MDT), both in the hospital setting and within the community, and these can trigger a referral for more specialist assessment (Fielding *et al* 2011; Morley *et al* 2011):

- Measured gait speed < 1.0 m/s measured over a 6m course
- Post-hospitalisation as a consequence of disuse and deconditioning
- Recent weight loss
- Reduced levels of physical activity
- History of recurrent falls
- Self-reported functional difficulty, e.g. inability to get out of a bed or rise from a chair independently
- Decline in overall health
- Recent weight loss
- Presence of co-existent, comorbid conditions, e.g. Type 2 diabetes, heart failure, COPD.

> "DESPITE PROGRESS IN REFINING CRITERIA, NO GLOBAL CONSENSUS EXISTS REGARDING THE DIAGNOSTIC CRITERIA FOR SARCOPENIA"

Conditions such as malnutrition and cachexia can lead to muscle wasting and there can be considerable overlap in these entities with sarcopenia that can be difficult to disentangle, especially in hospitalised older people (Patel 2014). However, clues from the patient's history and examination can point to the predominant cause. Starvation leads to a loss on non-fat structures, as well as body fat, while cachexia is associated with severe muscle wasting as well as loss of body fat that is driven by a marked pro-inflammatory state, e.g. cancer or heart failure. Body fat in individuals with sarcopenia is often preserved or increased and while individuals with cachexia can be sarcopenic, individuals with sarcopenia are not considered cachectic (Malafarina *et al* 2012).

Pathogenesis of sarcopenia

Multiple intracellular cell signaling pathways execute environmental and cellular cues that determine myofibre size through protein synthesis or degradation. These include the insulin like growth factor -1 (IGF-I) signaling

pathways that promote protein synthesis, and pathways associated with protein catabolism that are mediated by inflammatory cytokines, e.g. tumour necrosis factor alpha (TNF) and interleukin 6 (IL-6), among others (Goodman *et al* 2011). While the acute inflammatory response can be beneficial to promote muscle repair and regeneration the persisting, chronic, low-grade production of inflammatory cytokines that appears to be present in older individuals can adversely impact on muscle mass and function and there is substantial evidence linking inflammation with sarcopenia (Beyer *et al* 2012).

Skeletal muscle aging is also characterised by a continuous cycle of denervation and reinnervation, as a consequence of the loss of alpha-motor neurones within the central nervous system (CNS). Withdrawal of nerve terminals from the neuromuscular junctions (NMJ) and axonal sprouting from neighbouring neurons collectively give rise to larger, inefficient motor units. Remodeling skeletal muscle tissue through neuropathic, neurohormonal and inflammatory pathways leads to a reduction in muscle cross sectional area, volume and a reduced rate of force generation (Hepple & Rice 2016). This is characterised by the presence of fewer Type I oxidative (slow twitch) and Type II glycolytic (fast twitch) myofibres, myofibre atrophy and concurrent increase in non-contractile material.

Challenges and opportunities for multidisciplinary practice

Sarcopenia is considered a core component of frailty, indicating loss of physiological reserve in the skeletal muscle system (Landi *et al* 2015). However, in the typical phenotype of frailty, the gradual decline in physiological reserves is accelerated and accumulates across multiple systems, including the brain, immune, endocrine, cardiorespiratory, renal and haematological systems, as well as skeletal muscle. This accelerated decline across multiple systems can lead to failure of homeostatic mechanisms which, in turn, leads to vulnerability to adverse outcomes following minor stressor events. There is strong evidence that individuals living with frailty have poorer outcomes and die sooner than expected (Clegg *et al* 2016, 2013). As weakness tends to be the first sign of frailty and given that slow walking speed and low physical activity typically precede weight loss and exhaustion, identification of sarcopenia may be an especially useful method of recognising those people at increased risk of progression in their frailty status.

INTERVENTIONS FOR INDIVIDUALS LIVING WITH SARCOPENIA

Nutrition

The synthesis of muscle fibres requires adequate protein. Physiological changes in the gastrointestinal system are associated with a blunted anabolic response to ingested proteins (Robinson *et al* 2012). As such, older people may require more protein to counteract the inflammatory and catabolic effects not only associated with the sarcopenic phenotype, but also with co-existent co-morbidities and their exacerbations (Wandrag *et al* 2015). Protein supplements vary in their composition and current evidence from trials is inconsistent to enable the development of evidence-based recommendations for protein supplementation in sarcopenia (Hickson 2015; Beaudart *et al* 2017). However, observational evidence does suggest that essential amino acids, i.e. leucine and beta-hydroxy-beta-methylbutyrate (a bioactive metabolite of leucine), stimulates muscle protein synthesis more than non-essential amino acids and may be useful for maintaining lean body mass and improving muscle function (Deutz *et al* 2013; Paddon-Jones *et al* 2004; Wu *et al* 2015; Flakoll *et al* 2004; Stout *et al* 2013). A recent consensus statement from the multinational PROT-AGE group recommend that older people have a protein intake of at least 1.2-1.5 grams per body weight (kg) per day to maintain muscle homeostasis (Bauer *et al* 2013). Low vitamin D levels are also associated with decreased muscle strength and vitamin D supplement,

> "COMBINING PHYSICAL ACTIVITY AND NUTRITIONAL INTERVENTIONS CAN BE ASSOCIATED WITH BETTER FUNCTION, STRENGTH AND REDUCED INFLAMMATION"

especially for those older individuals who are deficient, may have positive effects on muscle strength (Beaudart *et al* 2014). Ultimately, however, whole diet modifications rather than single agent supplementation may be more appropriate strategies when advising dietary changes in older people (Hickson 2015). At present there are no medicine-based treatments for sarcopenia.

Physical activity

Physical inactivity and sedentary behaviour are common among older people, and this can lead to an acceleration in muscle catabolism as well as reduced aerobic capacity. In conjunction with other personal, social and environmental factors, such as issues with access to food and social isolation, a decline in physical activity can create a spiral of further inactivity, muscle loss, weight gain, mobility problems and an increase in cardio-metabolic risk (Ford & Caspersen 2012; Ryan *et al* 2015).

Combining physical activity and nutritional interventions can be associated with better function, strength and less inflammation in older sarcopenic people (Rondanelli *et al* 2016; Churchward-Venne *et al* 2013; Naseeb & Volpe 2017). In terms of physical activity, progressive resistance and aerobic exercise have been shown to be the most beneficial for the prevention and 'treatment' of sarcopenia (Cruz-Jentoft *et al* 2014; Liu & Latham 2009; Pahor *et al* 2014; Valenzuela 2012). While progressive

resistance exercise improves lean mass, strength and function (Peterson *et al* 2011), optimising exercise capacity through aerobic activity improves metabolic control, reduces oxidative stress and insulin sensitivity, and can also stimulate a hypertrophic response on muscle fibres. Despite being shown to be safe and effective in older people (Fiatarone *et al* 1994; Liu & Latham 2009; Vincent *et al* 2002), implementing progressive exercise in clinical practice is not always readily achievable. However, recent evidence suggests even incremental elevations in habitual activity in older people may help decelerate age related declines in musculoskeletal fitness (Westbury *et al* 2018).

Falls are a serious, and sometimes fatal, complication of sarcopenia. A multicomponent approach that addresses balance and gait, flexibility and endurance, and lower limb strengthening is required to manage those susceptible to falls. These approaches are aimed at improving reaction time, gait, balance, strength co-ordination and physical and cognitive function (El-Khoury *et al* 2013; Stubbs *et al* 2015). Group- and home-based exercise programmes, which incorporate safety interventions, may reduce the rate and risk of falling (Robertson & Gillespie 2013). Moreover, targeted home-based or group-based exercise interventions can also improve mobility and functional outcomes for older people (Clegg *et al* 2012; Theou *et al* 2011).

Intervening earlier in the life course, before or at the onset of mild functional limitation, may have huge benefits for later skeletal muscle health. For example, Dodds *et al* (2013) ascertained that increased levels of leisure time physical activity in mid-life was associated with stronger grip strength in both men and women at age 60-64. This is consistent with optimising peak strength earlier on in life, thereby reducing the impact of sarcopenia. Regular physical activity in adulthood may, therefore, prevent a more precipitous decline in muscle strength in older age (Dodds *et al* 2013).

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Hospitalisation of older individuals is commonly associated with immobility and bed confinement, with a typical length of stay in hospital being between 7-10 days (Lim *et al* 2018). Studies have shown that during prolonged bed-rest there is significant reduction in muscle protein synthesis, loss of muscle mass, strength / function, and aerobic capacity (Coker *et al* 2015; Kortebein *et al* 2008). Rapid identification of these individuals and implementing interventions to maintain muscle function between periods of bed-rest should be a priority for the MDT and may provide opportunities for innovative ways of maintaining physical activity levels.

Comprehensive care planning

It is clear that the older person's care requirements are complex and that they often have co-existent functional, psychological and social needs. Ultimately, the treatment goals for an older person with sarcopenia revolve around improving physical function and maintaining independence and well-being. A useful method for planning the care of those living with sarcopenia and frailty is through a process of CGA, a multi-professional diagnostic process that involves medical, nursing, therapy, dietetics and social services, etc. It is focused on determining the psychological and functional capability of the older person in order to develop a co-ordinated, integrated and personalised care plan for treatment, and long-term follow-up (Ellis *et al* 2011). With the patient at the centre of their decision-making process, the purpose of CGA is to improve diagnostic accuracy, optimise treatment and minimise polypharmacy. Additionally, the aim is to improve outcomes and crucially, allow effective integrated case management that ensures that the care plan is enacted and remains continually responsive to the patient's needs. This involves effective communication between secondary and primary care and vice versa. The personalised care plan, both diagnostic and therapeutic, can be overseen by any member of the MDT. There is good evidence that CGA, when delivered to patients in the community and within the hospital setting, is associated with decreased mortality, and reduces the likelihood of the patient experiencing better function and cognition rather than a deterioration in health and / or being institutionalised (Ellis *et al* 2011; Stuck *et al* 2002; Cameron *et al* 2013). Equally, the CGA process can facilitate appropriate advanced care planning discussions with individuals who may be towards the end of their life.

Summary

Sarcopenia is a major health problem for older people. Clearly, early identification as well as a better understanding of mechanisms underpinning the development of sarcopenia will create a basis for clinical trials and therapeutic interventions. Physical activity and nutrition are currently the cornerstones of interventions as currently no drug is licenced for the treatment of sarcopenia.

Conflicts of interest

The authors declare that they have no conflicts of interest.

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Preserving muscle strength and function in old age

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For years it has been assumed that muscle wasting was an inevitable part of growing older, but John Starbrook, who at 87 completed the 2018 London Marathon is, together with several other masters athletes, forcing us to reconsider this view. While the media is full of how sedentary lifestyles and poor nutrition are making our youth ill, the research also seems to suggest that it may be an important factor influencing how our neuromuscular systems age.

LEARNING OUTCOMES
TO SUPPORT PHYSIO FIRST QAP

1 Understand that maintaining muscle mass and strength in old age is dependent on several variables.
2 Be aware of how different lifestyle choices, especially exercise, can influence neuromuscular aging.
3 Help patients to understand that they have a degree of control over how they age through knowledge sharing.

Introduction

Old age is commonly associated with a progressive loss of muscle mass, strength and performance. At its worst this can lead to physical disability, poor quality of life, and premature death.

This loss of muscle mass, also known as sarcopenia, and function is commonly regarded as a given, and researchers (Fujita *et al* 2007) often refer to these changes happening as a natural consequence of "healthy aging".

I would like to challenge this view. I believe that the current research is showing that sarcopenia and loss of muscle function is a result of the natural aging process of sedentary adults, not healthy ones. We know that a sedentary lifestyle is extremely unhealthy, contributing to a wide variety of illnesses including diabetes and heart disease.

While it is the case that a sedentary person may appear healthy, their physiology is affected in a very different way compared to that of active individuals. The research clearly shows that a sedentary lifestyle does not allow healthy aging of the neuromuscular system and it is my opinion that researchers should not refer to sedentary individuals as undergoing healthy aging (Passaro *et al* 2018).

There is currently no consensus with regard to a definition for sarcopenia. Most of the formulas used to detect it usually consider a person as being sarcopenic if their muscle mass deviates from the standard gender-specific mean muscle mass for young adults.

The prevalence of sarcopenia appears to vary greatly between different populations. In Taiwan, for instance, 26% of men and 19% of women over the age of 80 were found to be sarcopenic, while researchers in America have put the figures at 50% of men and 72% of women for a similar cohort (Mitchell *et al* 2012).

Differences in DNA, epigenetics, lifestyle, exercise and nutrition may all play a part in these differences as they have been shown to have an effect on muscle metabolism in old age.

Age-related changes in the neuromuscular system that affect performance

The following highlights some of the findings from a recent in-depth review of how aging affects skeletal muscle performance. For more detail on each of the topics, I recommend accessing the full Tieland *et al* (2018) research.

THE MUSCULAR SYSTEM

Studies have shown that, on average, muscle mass declines in the general population by around 0.37% per year in women and 0.47% in men. This increases to 0.64-0.70% and 0.80-0.98% respectively in people over the age of 75 (Mitchell *et al* 2012).

Several studies also show that the loss of muscle mass in old age is due to both a decrease in the total number of muscle fibres, and in the muscle fibre size (Tieland *et al* 2018). As it is mostly the Type 2 fast twitch, rather than the slow twitch Type 1 muscle fibres that appear to reduce in size, this may explain why elderly people in the general population often struggle to carry out movements that require quick, strong muscle contractions, e.g. climb stairs or get up off the floor.

Studies in rats have shown that there is also an increase in muscle fibrosis between muscles fibres which can influence the elastic properties of muscles. An increase

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in intermuscular adipose tissue also appears to be a symptom of old age, but the research has found that the amount can vary greatly between individuals (Tieland *et al* 2018).

A decrease in aerobic capacity of around 10% per decade has been observed. This is thought to be partly due to changes in the mitochondrial functioning and content of muscle cells (Tieland *et al* 2018).

CONNECTIVE TISSUE

The functional stiffness of tendons seems to decrease as we age, despite the tendon collagen becoming stiffer. The end result is a decrease in the spring-like properties of the muscle-tendon unit and a subsequent decrease in performance (Tieland *et al* 2018).

NERVOUS SYSTEM

It has been observed that, as we get older, the number of motor units in our muscles reduces. There also seems to be an increase in the number of muscle cells served by a single motor unit. This is thought to be due to compensatory sprouting of the surviving neurons (Tieland *et al* 2018) which the researchers suspect is the reason for the observed loss of Type 2, fast twitch, muscle fibres in some older people. This may be due to the denervation of these fibres and the subsequent re-innervation through axonal sprouting from slow motor neurons.

The good news is that the research has shown that healthy, active older adults can preserve their ability to activate their muscle motor units optimally (Tieland *et al* 2018).

The active, older population

Wroblewski *et al* (2011) investigated muscle mass and body composition in masters athletes. They found that chronic intense exercise, i.e. more than four sessions a week, preserved lean muscle mass into old age and, despite the age-related increase in total body fat, this did not lead to fatty infiltration into the muscles themselves.

The research concluded that the decline in muscle mass previously thought to be a natural part of aging was more likely to be due to lifestyle choices, e.g. a sedentary lifestyle and poor nutrition.

Mckendry *et al* (2018) supported these findings in their recent review and meta-analysis. They concluded that the current evidence suggests that chronic exercise training preserves physical function, muscular strength and body fat levels in old age, similar to that of young, healthy individuals. Interestingly, Piasecki *et al* (2016), found that masters athletes experience a drop in motor unit numbers similar to that of the elderly, sedentary population.

There is still a paucity in research investigating physically fit older people and it's not clear how many of the previously mentioned neuromuscular changes also applies to this group. What the current research does suggest is that, if fit older adults are not spared from these neuromuscular changes, it may be possible to combat their negative affects through other strategies such as exercise and nutrition.

Inflammation

Ageing is often accompanied by inflammatory disorders. Slight elevations in circulating pro-inflammatory mediators and decreases in anti-inflammatory cytokines result in chronic low-grade inflammation which can add to muscle loss in older people (Zhai & Xiao 2017). Strategies that decrease chronic, low-grade inflammation may thus be important to combat sarcopenia (Dalle *et al* 2017).

Researchers found that, in rats, low-grade inflammation attenuated their muscle synthesis response to dietary protein, leucine and that antioxidant supplements that reduced the inflammation were effective in improving the anabolic response to leucine.

Other strategies that might be considered in combating inflammatory disorders include Omega 3 fatty acids that have been shown to alleviate systemic inflammation and increase muscle volume and muscle strength in the elderly (Dalle *et al* 2017), and vitamin D which is important for the maintenance of skeletal health and the immune system and prevents muscle wasting (Dalle *et al* 2017). Supplements may be of use to those who are at risk of deficiency in either of these elements.

Building new muscle: effects of nutrition and aging

It has been established that the balance between skeletal muscle breakdown and synthesis is strongly influenced by the levels of amino acids circulating in the blood. The exact science involved in the process is still unclear, but results thus far suggest there is an intricate relationship between amino acid availability, skeletal muscle synthesis and aging.

Ingesting small quantities, i.e. between 7-10g, of essential amino acids can

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stimulate skeletal muscle synthesis in young individuals, but not in older adults. However, larger quantities, i.e. between 20-40g, of amino acids do stimulate muscle protein synthesis to the same degree in all age groups (Dickinson *et al* 2013).

Dickinson *et al* (2013) conclude that the data indicates that the "threshold" at which amino acids stimulate muscle synthesis is increased with age, and so this process could be enhanced by ingesting adequate quantities.

As previously mentioned, leucine has been identified to be important for muscle synthesis, having been shown to be a potent stimulator for muscle growth. In studies where 2g of leucine concentrate was included in just 7g of protein, it was found that both the younger (28/30yrs), and older (66/70yrs) subjects showed the same level of muscle synthesis despite the small dose of protein ingested (Katsanos *et al* 2006; Paddon-Jones & Rasmussen 2009). Thus, increasing the amount of leucine within their diet may promote muscle synthesis in older adults.

This, however, contradicts prior research in which no difference in muscle synthesis was found in response to 20g amino acid ingestion between age groups (Pennings *et al* 2010). This disparity may be due to cultural differences (Dickinson *et al* 2013) that lead to societal discrepancies in daily physical activity and lifestyle. Exercise has been shown to enhance older adults' anabolic response to amino acids and it could be that the results for the subjects in Pennings' study were due to them having more active lifestyles.

Both of these studies are also at odds with Verhoeven *et al* (2009) who found no increases in muscle strength or mass in healthy elderly men who had taken 2.5g leucine supplement for three months.

The subjects in this Verhoeven *et al* (2009) study consumed mixed meals, i.e. containing protein, carbohydrates and fat, which may have influenced how their muscles responded to the leucine, and which is further explored in this article in the section on insulin.

Another randomised control trial, conducted over three weeks, looked at the effect of 20g protein supplement on muscle mass post total knee replacement surgery. The researchers found that the control group showed attenuated muscle atrophy in all the muscles of the operated and the un-operated leg (Dreyer *et al* 2013). At two weeks post-surgery the placebo group showed a -14.3 ± 3.6% change in muscle mass from baseline, and at six weeks this had increased to -18.4 ± 2.3% compared to -3.4 ± 3.1% and -6.2 ± 2.2% for the control group's operated legs.

The variability in these results underlines how complex the mechanisms behind sarcopenia are, and that a multi-faceted treatment approach is needed, and Dickinson and colleagues (2013) warn that it is important to remember that exercise has been shown to play an important role in muscle synthesis, and that physical activity or base level fitness may be another important reason for differences in results.

I fully agree with this statement since the subjects in Dreyer's study were asked to administer their supplements an hour after physical therapy, while Verhoeven and colleagues (2009) did not report on the activity levels of their subjects. Why the timing of food intake may be important is discussed later in this article.

The role of insulin

There is evidence that a meal that includes 40g carbohydrates and 40g protein interferes in the ability of amino acids to elicit a protein synthesis response in older adults (Dickinson *et al* 2013).

This puzzled researchers since a meal containing carbohydrates causes an increase in circulating insulin, and insulin is known to stimulate muscle growth. Further investigation suggested this effect may be due to the inability of insulin to stimulate an increase in muscle blood flow in older adults. Artificially increasing the blood flow in the muscles of older adults caused the difference in muscle synthesis between age groups to disappear.

These results are supported by those of Fujita *et al* (2007) who found that a bout of aerobic exercise before eating a mixed meal could restore the normal muscle protein synthesis reaction to insulin.

Building new muscle: effects of exercise and aging

RESISTANCE EXERCISE

Early studies showed that resistance exercise could increase muscle size and strength in older individuals, but not to the same level as for younger individuals (Peterson *et al* 2011). This may not be entirely true, however, as in more recent research Deutz *et al* (2014) have shown that older individuals may be able to achieve similar muscle gains.

There is evidence that more protein is incorporated into muscle if resistance exercise is performed before a meal (Pennings *et al* 2010). Drummond *et al* (2008) also found that, by performing a bout of resistance exercises before ingesting 20g of essential amino acids, older athletes experienced a similar anabolic response to the younger ones.

This is thought to be due to the exercise decreasing the insulin resistance in the muscle cells and increasing the blood flow, allowing for a normal anabolic response to the circulating amino acids.

AEROBIC EXERCISE

Harber *et al* (2012) showed that a 12-week cycling programme was effective in increasing muscle mass in both young and old individuals. The researchers suggest that the secret may again lie in the ability of aerobic exercise to overcome age-related insulin resistance. It is important to note that the individuals studied were not trained athletes, so it may also be the case that the improvement was because of their initial low baseline muscle mass prior to undergoing the exercise programme.

In an earlier investigation, Fujita *et al* (2007) examined the effect of aerobic exercise on muscle protein synthesis and found that, 20 hours after a single bout of walking for 45-min on a treadmill, at 70% of maximum heart rate, the physiological anabolic response of muscle protein to insulin was restored in older individuals.

Dalle *et al* (2017) also argues that chronic aerobic exercise further promotes muscle synthesis by decreasing chronic low grade inflammation.

Conclusion

Several factors may influence muscle loss in older age. Sedentary older adults appear to have a blunted muscle synthesis response to dietary amino acids when compared with young controls, which is likely to be a result of insulin resistance. However, when older adults perform a bout of aerobic exercise or resistance training before a meal they experience a similar muscle synthesis response to young controls. This may explain why masters athletes do not exhibit the same declines in muscle mass and function as sedentary older adults.

It is clear, therefore, that physical fitness is a key factor for managing health during all stages of life and should be promoted. As healthcare providers we should take every opportunity available to educate and inform our patients of the long-term benefits of physical fitness.

Generic advice on exercise should be avoided in favour of helping our patients with fitness plans that are tailored to their lives, and that they truly feel comfortable with. By empowering patients to take responsibility for their own health we may one day be able to reduce the detrimental effects of chronic illnesses on our society.

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Radiculopathy, radicular pain and referred pain: what are we really talking about?

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Pain that radiates from the neck down the arm, or from the back down the leg, is a common presentation in clinical practice. However, despite the importance of standardisation in physiotherapy practice, there exists an abundance of terms to describe such symptoms, that may not only be confusing to patients, but may worry them unduly if explanations imply that their body is vulnerable or damaged. Therefore, communicating a diagnosis to the patient in a way that is honest, reassuring and empowering is very important. This article also aims to clarify these terms, explain the pathophysiology behind them, consider their limitations in light of recent developments in pain science.

LEARNING OUTCOMES
TO SUPPORT PHYSIO FIRST QAP

1 To clarify the terminology of radicular pain, radiculopathy and referred pain.
2 To consider these terms in light of the complexity of pain science.
3 To address how to explain radicular pain and radiculopathy to our patients.

Introduction

In 1941, the rheumatologist Jonas Kellgren wrote a paper in *The Lancet* lamenting that back pain with leg pain was, in his experience, too often diagnosed as "sciatic neuritis" when in fact it was nothing of the sort (Kellgren 1941). He had performed a series of experiments showing that noxious stimulation of the ligaments and muscles of the spine produced segmental areas of referred pain running down the buttock, leg, shank and foot; a referred pain which he accused the "neurological school" of physicians of over-diagnosing as nerve pain. Assessing 90 x-rays of patients diagnosed with sciatica, Kellgren judged that 50 of them were in fact suffering referred pain.

Later, in 1994, the radiologist Pierre Milette wrote to the journal *Radiology* on the issue of radicular and referred pain to ask simply, "what are we really talking about?" (Milette 1994). Like Kellgren, Milette accused his colleagues of over-diagnosing sciatic nerve root pathology, assuming that any back / leg pain was neurogenic. He pointed to confusion surrounding the terminology for back / leg pain not only clinically but even at an academic level. "If we seek to improve our understanding", Milette wrote, "it is mandatory to address this fundamental issue".

Today, despite Kellgren and Milette's pleas, there is still some confusion about the terms *referred pain*, *radicular pain* and *radiculopathy*. This article aims to address this confusion on two levels. First, at the level of *communication between clinician and clinician*: in order to reduce unwarranted variation in practice it is important to, as Milette put it, be clear what we are really talking about. Second, at the level of *communication between clinician and patient*: as physiotherapists are becoming more aware of the impact of the words we use, and their power to both harm and heal, we need to improve on our common lexicon so we can explain nerve root pain in a way that is clear, reassuring and empowering.

Communication between clinician and clinician

BASIC TERMINOLOGY (TABLE 1)

Referred pain, or *somatic referred pain*, is

	MANIFESTATION	MECHANISM
SOMATIC REFERRED PAIN	Dull, aching, gnawing and difficult to localise pain, with an inconsistent and non-dermatomal pattern	"The convergence of nociceptive afferents on second order neurons in the spinal cord that also happen to subtend regions of the lower limb" (Bogduk, 2009)
RADICULAR PAIN	Lancinating, shocking, electric feeling in a thin band	Compression and / or inflammation leads to ectopic (i.e. spontaneous, abnormal) discharges emanating from the dorsal root or its ganglion
RADICULOPATHY	Objective neurological signs: loss of function in reflexes, power and sensation. (Not necessarily pain)	Compression and / or inflammation leads to demyelination and axonal damage, causing reduced impulse conduction along the nerve

TABLE 1: Basic terminology of the manifestation and mechanism of pain (Bogduk 2009; Baron *et al* 2016)

"AS PHYSIOTHERAPISTS, WE ARE BECOMING MORE AWARE OF THE IMPACT OF THE WORDS WE USE"

a result of nociception (danger messages) arising from the structures of the spine (Bogduk 2009; Baron *et al* 2016). The proposed mechanism is the "convergence-projection model". According to this model, afferents from the lumbar spine and from the lower limb converge on the same second order afferent in the spinal cord, and so the brain cannot discriminate between the two sources of input when they are projected up. As a result, the brain creates a pain experience for both, even though the lumbar spine is the only significant source of nociception. This is the same mechanism by which visceral referred pain, like the shoulder and arm pain of a heart attack, is assumed to work.

Somatic referred pain is dull, aching, gnawing and difficult to localise (Bogduk 2009). Figure 1 illustrates the results of Kellgren's (1941) aforementioned experiments to find the referred pain patterns for each spinal segment.

Radicular pain is a result of an action potential emanating from the axon of a nerve at the dorsal root, or at the nerve root ganglion, as opposed to resulting from stimulation of the nerve's peripheral terminals (Bogduk 2009; Baron *et al* 2016). This discharge is "heterospecific", a barrage of AB, Ad and C fibre discharges, and so technically it is not nociceptive.

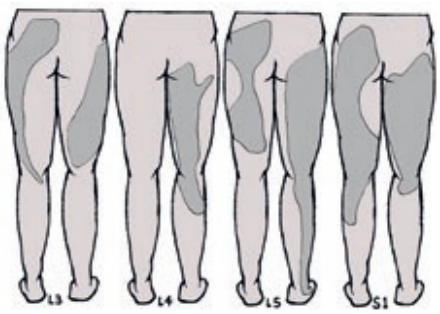

FIGURE 1: Patterns of somatic referred pain evoked by noxious stimulation of the interspinous ligaments at the segments indicated. Based on Kellgren (1941)

FIGURE 2: An illustration of the lancinating quality of radicular pain travelling into the lower limb along a narrow band.

Figure 2 illustrates the result of Kellgren's (1941) experiments to induce radicular pain. The subjects reported this pain as a lancinating, shocking, electric sensation.

The pathophysiological process is complex. Compression of the nerve, whether by a disc or its exudate, or by foraminal stenosis, reverses the downward pressure gradient necessary to maintain the nerve's blood supply causing it to become ischaemic. One way in which this leads to pain is by causing Schwann cell death and demyelination, which exposes a portion of axon from where spontaneous action potentials can emanate to be processed by the brain and experienced as pain (Schmid 2015). Another way in which compression and ischaemia lead to pain is by inciting intraneural inflammation, which alone is sufficient to sensitise a nerve to movement (Dilley *et al* 2005) and can spread to the spinal cord and the brain (Albrecht *et al* 2018). In addition, intraneural inflammation impairs axonal transport so that ion channel components being transported along the nerve bunch up in one place, creating a "hot spot" of excitability (Dilley *et al* 2013).

The mechanisms of radicular pain are not fully understood and more complex than is suggested by the neat description here; for a more comprehensive explanation see Schmid (2015).

As figure 1 demonstrates, experimentally induced radicular pain manifests in a clear line that approximates the territory innervated by the affected nerve root. Clinically, however, the boundaries of radicular pain are less clear. Bove *et al* (2005) found that, of 25 patients with lumbar radicular pain, all but one reported multi-dermatomal pain. Similarly, Furman & Johnson (2018) found that only radicular pain on the anterior, medial and lateral thigh had any correlation with its nerve root origin, whereas buttock, gluteal and calf pain had none (figure 3). The non-dermatomal presentation of radicular pain is likely because intra-neural inflammation has the potential to spread to the dorsal horn and beyond, which would sensitise neighbouring nerves (Schmid *et al* 2013a); and because nerve roots have multiple intrathecal anastomosis (cross linkages) (Furman & Johnson 2018).

Radiculopathy is the objective loss of function of a nerve resulting from damage to the nerve root (Bogduk 2009; Baron *et al* 2016). This manifests clinically as loss of muscle power, reflex response, and the sensation of light touch, hot / cold response and response to pinprick. As discussed, a compressive insult to the nerve can set off a chain events that cause it to become demyelinated and inflamed, in

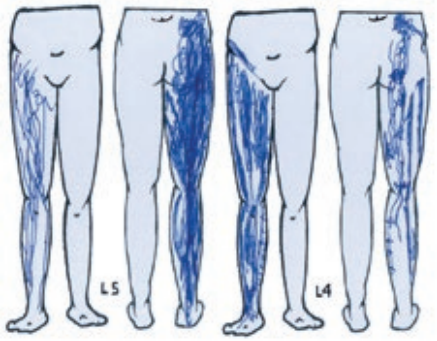

FIGURE 3: An illustration of the variation of induced radicular pain distribution in multiple patients. Based on Furman & Johnson (2018)

> "THE CLINICIAN SHOULD MAKE A DIAGNOSIS OF RADICULOPATHY BASED ON AN ACCUMULATION OF SIGNS RATHER THAN JUST ONE"

some cases there may also be axonal degeneration (Merskey & Bogduk 2012). A nerve in this poor state of health cannot readily conduct action potentials, leading to the loss of function we call radiculopathy. Radiculopathy is not a pain condition at all; it is a diagnosis that refers only to loss of nerve function and may or may not be painful (Merskey & Bogduk 2012).

NOTES ON MAKING A DIAGNOSIS

In order to diagnose a radiculopathy (as opposed to radicular pain), we need to see objective loss of nerve function, but it is important to remember that many of our tests for the loss of function of a nerve have imperfect specificity, i.e. will turn up false positives (Tawa *et al* 2017). This implies that the clinician should make a diagnosis of radiculopathy based on an accumulation of signs rather than just one (Tawa *et al* 2017).

When it comes to differentiating between radicular and referred pain there is no clear and validated method, although recent work suggests that a cluster of:

1. pain below the knee
2. leg pain worse than back pain
3. positive neurodynamic test
4. the report of pins and needles

is strongly associated with nerve root involvement (Stynes *et al* 2018).

As previously discussed, radicular pain is not necessarily a "line" of pain and rarely follows dermatomal patterns (Furman & Johnson 2018). In cases of doubt, the International Association for the Study of Pain (IASP) suggests simply documenting "lower limb pain" (Merskey & Bogduk 2012).

When documenting findings from an objective assessment, table 2 illustrates how it might be helpful to make the distinction between gain and loss of function (Schmid *et al* 2013a).

IMPORTANT CAVEATS

Having presented a classical way of categorising low back / leg pain and neck / arm pain it should be noted that, to make a pain diagnosis based on an underlying mechanism like "radicular" or "referred" is to reformulate something complex and emergent into something simple and linear, and as such is perilous.

The distinction between different kinds of axial / limb pain is not clear; for example, nerve entrapment pain is not necessarily neuropathic, as nervi nervorum (the nerves subserving the nerve trunk) can be a source of nociception (Teixeira *et al* 2016). Additionally, radicular and referred mechanisms can co-exist. Seemingly non-neuropathic pain can feature "occult" neuropathic symptoms (Spahr *et al* 2017), and, of course, pain itself is a higher construct. None of the above mechanisms "are" pain until this information reaches the brain (Brodal 2017).

Indeed, it is arguably not necessary to consider underlying mechanisms at all. They may in fact distract from variables like expectations, disability and pain severity, which appear to be more important for the prognosis and management of many musculoskeletal conditions (Croft *et al* 2015; Konstantinou *et al* 2017), but such considerations have to be held against the practical need for clinicians to communicate clearly and productively with each other, and between disciplines. For this reason, classification, though awkward, is arguably necessary.

Just as importantly, we must also consider how we communicate such labels as *radicular pain* and *radiculopathy* with patients.

Communication between clinician and patient

The clinical presentation of radicular pain has little association with findings on imaging (Karppinen *et al* 2001), but in a survey of people with a history of cervical radiculopathy, Weber *et al* (2017) found that 67% believed an MRI is more important than clinical findings in the decision-making process for their condition, and 47% said they would be willing to undergo surgery based on "abnormal" findings on an MRI alone, even if they had no or few symptoms.

It is no surprise that patients appear to have a biomedical understanding of radicular pain and radiculopathy, they share that perspective with much of the medical and allied health professions and with society at large. For our patients' sake, physiotherapists must promote a more evidence-based and less threatening understanding of these conditions (Moseley & Butler 2017). This is a delicate matter as there is a fine line between de-threatening a condition and trivialising it.

	DEFINITION	MANIFESTATION	TESTED BY	MECHANISM
GAIN	Abnormal excitability	Paraesthesia, spontaneous pain, hyperalgesia and allodynia, hyperreflexia, muscle spasm	Neurodynamic tests, Spurling's test, Tinel's test, pain on palpation	Demyelination, axonal degeneration and neuroinflammation
LOSS	Reduced impulse conduction	Hypoesthesia, anaesthesia, weaker / absent reflexes, muscle weakness, reduced sensation	"Hard neuro" deficits, pinprick, hot, cold testing	Demyelination, axonal degeneration but not neuroinflammation

TABLE 2: Distinguishing between gain and loss of function during objective assessment

There is no script for patient education, and where possible it should be tailored to the individual's prior knowledge and beliefs. That said, the following learning aims are likely to appear in most explanations of cervical radicular pain and radiculopathy. It is often possible to provide them in an "online commentary" to the objective assessment, to save the patient a lecture at the end.

CONCEPTS TO EMPHASISE (TABLE 3)

Since Victorian times, nerves have been thought of as a weak-point in our bodies, the slightest damage to which spells disaster (Bourke 2014). Patients may, therefore, benefit from knowing that nerves are big, tough structures with the dura mater and epineurium forming a layer of protection around them (Moseley & Butler 2017). Patients may also be pleased to hear that, like any other part of the body, nerves also like movement; a good stretch disperses inflammation and helps provide them with lots of oxygen.

The terms "trapped" and "pinched" nerves are probably nocebic, and in any event unlikely to be accurate given that the nerve root ganglion takes up just one third of the space in the outlet holes between the vertebrae, meaning that it potentially has room to move even after significant degenerative changes (Moseley & Butler 2017). Research by Schmid *et al* (2013b) has shown that mild compression, rather than tight constriction, is sufficient to induce nerve pain in rats, so "compressed" or "crowded" are more appropriate words to use. Indeed, it is neuroinflammation that is likely to be more associated with the patient's symptoms than nerve root compression (Albrecht *et al* 2018; El Barzouhi *et al* 2013), and an explanation in these terms would frame the issue accurately as a normal pathophysiological process, i.e. inflamed as opposed to an aberrant biomechanical event, i.e. trapped.

Where a patient has radicular pain with no objective loss of function, it is important to reassure them that there is no evidence that the nerve is damaged, but that it does appear to be irritated. Where there are signs of radiculopathy, i.e. loss of nerve function, it is important to be honest, i.e. advise that this implies demyelination and axonal degeneration rather than offer blind reassurance that "there is no damage", which is arguably unethical. However, the patient may benefit from hearing that this is common and normal, and that neural tissue, like any other soft tissue, is capable of healing and rebuilding. There is some evidence that loss of nerve function, as long as it is not progressive, is not particularly relevant for prognosis; indeed, it may be an indicator of a better than average outcome as it implicates a disc protrusion, which has a good chance of resolving spontaneously (Konstantinou *et al* 2017).

There is an opportunity to link nerve pain to the patient's wider health and wellbeing. Explain to the patient that nerves are connected to the brain and made of the same stuff so, if the brain is stressed or overworked, the nerve might not get a chance to calm down either. This means taking time to relax and sleep well is part of rehab. With regard to exercise, although central sensitisation is a theoretical concern when exercise is painful (Woolf 2004), there is no reason to think that even vigorous movement will cause structural damage to the nerve.

> "THE NERVES ARE CONNECTED TO THE BRAIN, SO IF THE BRAIN IS STRESSED, THE NERVE MIGHT NOT GET THE CHANCE TO CALM DOWN EITHER"

Nerve pain appears to cause patients more distress than somatic pain (Spahr *et al* 2017). Part of the reason for this may be that, for the patient, it is a strange sensation. By explaining that it is the nerve's job to send information about the body and its surroundings to the brain, and that when the nerve is irritated it can start firing off a lot of useless information that is similar to white noise, the patient can be reassured that, although they might feel all sorts of sensations from the nerve, the problem itself is likely to be minor and quite localised. Equally, the worrying symptom of night pain is easily explained; blood pressure drops at night, and when this is combined with the individual

	CONCEPTS TO EMPHASISE
NERVES	Big and tough, with a protective covering Move, bend and stretch with the rest of the body Enjoy movement. A workout gives them a good stretch, disperses any inflammation and brings them a fresh blood supply
"TRAPPED" NERVES	Even if you lose some space there is plenty spare Nerves are slidey and bendy – they are almost impossible to pinch. "It would be like picking up a lychee with chopsticks" More crowded out, or compressed, than trapped
PAIN AND DAMAGE	Zings and zaps do not mean that a nerve is injured – more likely it is sensitive Most painful nerves are sore, but safe If the nerve isn't doing its job as well as it usually does, then just like muscles and ligaments, it can repair and recover
UNUSUAL SYMPTOMS	Night pain is normal, it is because of a drop in blood pressure and often because you fall asleep in an awkward position Touchy nerves report all they possibly can to the brain – sometimes weird and wonderful things There is usually nothing wrong with the area you feel the pain

TABLE 3: Explanatory statements of nerve concepts (Moseley & Butler 2017)

> "A DROP IN BLOOD PRESSURE, COMBINED WITH AN ODD SLEEPING POSITION, CAN RESULT IN A TEMPORARY ISCHAEMIA TO THE NERVE, AND EXPLAIN NIGHT PAIN"

sleeping in an odd position, it can result in a temporary ischaemia to the nerve (Moseley & Butler 2017).

With regard to prognosis, the assumption that radicular pain has a favourable natural history is not borne out by the literature. Although some reviews are optimistic (Casey 2011; Wong 2014), a recent high-quality trial of 609 patients with back / leg pain, in primary care, found that just 55% reported a 30% or more improvement in one year (Konstantinou *et al* 2017). This study also identified factors associated with improved disability: shorter pain duration, lower leg pain intensity, fewer other symptoms associated with back and leg pain (e.g. fatigue, stiff joints, poor sleep, loss of strength), and belief that the problem will be short lived (Konstantinou *et al* 2017). Clinicians are, however, not yet in a position to be confident forecasting an outcome for an individual. Patients can be counselled that there is a good chance their pain will fully resolve, but they need to know that there is also a good chance it may not.

Conclusion

This article began by asking the question: "what are we talking about really?" when we talk about axial / limb pain. As demonstrated, there are quite clear differences between radicular pain, radiculopathy and referred pain; although the clinician should maintain a healthy scepticism of the veracity and usefulness of any mechanism-based pain diagnosis. When it comes to explaining these conditions to our patients, there are many ways to give them hope for the future and confidence that they are safe to move; although we should not, in our enthusiasm, be tempted to give explanations that are false.

To reiterate Pierre Milette's statement of 24 years ago: "If we seek to improve our understanding, it is mandatory to address this fundamental issue." I hope this article has made a small contribution to that effort.

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About the author

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Managing complexity in musculoskeletal conditions: reflections from a physiotherapist

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There are more years lived with musculoskeletal (MSK) disability than any other long-term condition. The aim of this article is to present a case study that reflects some of the challenges in translating the multitude of evidence into clinical practice and the context of the individual with multi-morbidity.

LEARNING OUTCOMES
TO SUPPORT PHYSIO FIRST QAP

1 Introduction to the concept of the intersubjective space and its potential in person-centred care.
2 Understand how to reconcile the tension between the narrative and normative using the person-centred approach.
3 Recognise how the totality of evidence is used to inform person centred decision-making.
4 Awareness that there is a way to include the use of social prescribing in the management of multi-morbidity conditions.

Introduction

Musculoskeletal (MSK) disability has an enormous impact on the quality of life of millions of people in the UK:

- One in four, or around 9.6 million UK adults, many of whom are young and of working age, are affected by MSK disability
- 30% of GP consultations in England are MSK related (Department of Health 2006)
- 10.8 million working days are lost as a consequence of MSK conditions
- A large number of co-morbidities, including diabetes, depression and obesity are associated with MSK conditions (Arthritis Research UK 2013)
- More than 25% of all surgical interventions in the NHS are MSK related and this figure is set to rise significantly over the next 10 years (Arthritis Research UK 2013)
- £4.76 billion of NHS spending each year is on MSK conditions (Department of Health 2012).

The hierarchy of evidence-based physiotherapy, with randomised controlled trials that are viewed typically as the highest and most valued source of evidence, can fall short of informing clinical decision-making due to lack of external validity, generalisability and application to the individual case. This article discusses ways in which clinicians can move towards an approach that attempts to draw on the totality of evidence from multiple sources to allow an evidence-informed process that makes sense to both the clinician and the patient, in the spirit of person-centred decision-making and compassionate care.

An opening encounter

A 58-year-old nurse with intermittent bilateral posterior thigh pain to the knee was referred to my clinic. Her main complaint was a recurrence of persistent low back pain.

She had already undergone investigation and her MRI scan showed a healthy spine, albeit with some bilateral lateral recess narrowing at L4/5 with type 1 modic changes around the vertebral end plate.

I remembered treating this patient maybe six or seven years ago. Lisa (name and details changed for anonymity) worked in a care home and was a stoical type. She had endured many difficult medical and social situations, had a history of breast cancer and, since I last saw her, she had become pre-diabetic, put on weight and was smoking, which I was sure she felt guilty about, having previously given up. We had got on really well last time and she had made a good recovery, but this time it was different.

I asked how she was doing, knowing full well that she was clearly struggling.

Her reply was “not so good”, and she lowered herself into the chair with a pained grimace. I sat opposite her and invited her to tell me what had happened since we had last met. I kept quiet and concentrated on her story.

LISA’S STORY

Over the past five or so years, the periodic back pain that Lisa had suffered had been minimal and not at all intrusive. She had felt in control of her symptoms, she understood a lot more about how it affected her and what she could do about it, particularly during flare-ups. She would take things a little

> "I AM IN CONFLICT WITH THE CURRENT MANUAL HANDLING GUIDELINES THAT ADVISE TO KEEP THE SPINE STRAIGHT AND MOVE IN WAYS THAT SEEM UNNATURAL"

easier at work, restart her home exercise programme and take some over-the-counter analgesia.

Lisa believed that the heavy manual handling she had done in her 20s had caused irreparable damage to her back. I didn't challenge this belief; I am in conflict with the current manual handling guidelines that constantly advise to keep the spine straight and move in ways that seem unnatural but, for Lisa, the identity of her back pain and her passion for her job was so deeply intertwined and I didn't want to negatively affect the rapport we had developed.

When I asked about when things started to get difficult again, Lisa explained that, about 18 months ago, while helping to turn a patient, she had felt a sharp sting in her back and a pain like she had never felt before. Her back went into spasm and, as she was unable to finish her shift, she went home to recover. The next day her back was extremely stiff and she started to experience a vague pain in both legs.

Aware of her medical history (figure 1, box 1), I asked Lisa what she was thinking at that stage. She said she had started to worry about the referred pain. She had seen patients suffering with back-related leg pain who were now in wheelchairs.

She went to see her GP who examined her and completed a neurological assessment, decided that the history indicated a back sprain, prescribed analgesia and signed her off work for a week, asking her to come back after that time. The GP had reassured Lisa about her leg symptoms and, following the visit, her pain initially eased. Feeling that she was going to recover quickly, Lisa started her home exercises the next day but for some reason they were making her symptoms worse.

When I asked what she thought might have happened, Lisa explained that she felt she must have done some serious damage and, on returning to her GP, became more concerned when the advice was that she needed more time off work and the symptoms should resolve in six weeks.

When Lisa returned to work a few weeks later she was still in pain. She was put on light duties and told that she could not return to normal duties or shift patterns until she was 100% better. Six weeks later her symptoms still had not significantly improved; she was struggling to bend to pick things up and put her shoes on and she was increasingly reliant on her family to do more things around the house. Lisa's leg pain became even more erratic with no pattern why some days were completely pain free and others were terrible. She was having trouble sleeping, was comfort eating and becoming less active. She gained weight and started to feel hopeless.

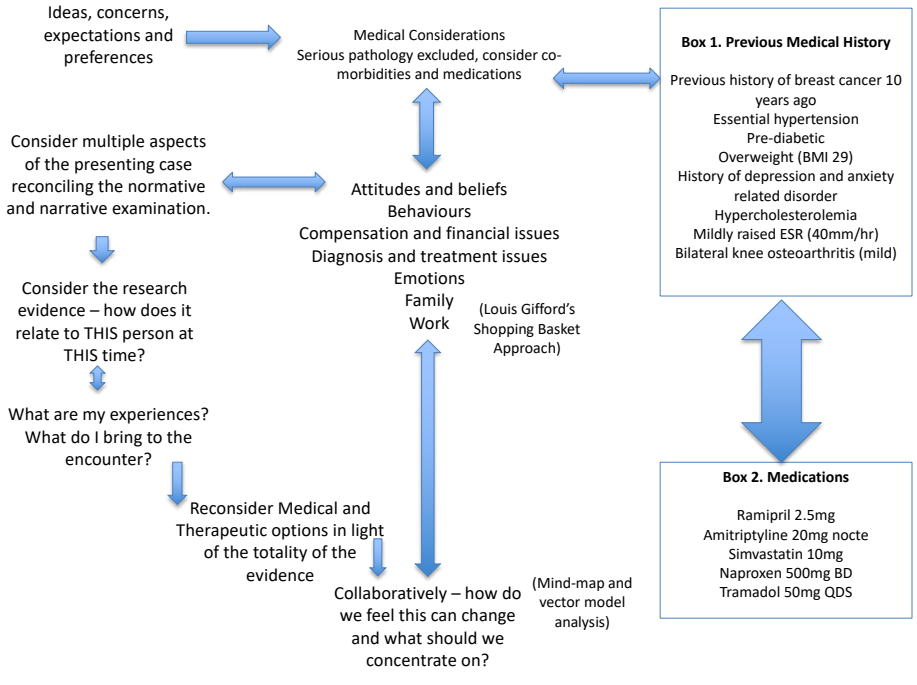

FIGURE 1: Flow chart gathering the totality of evidence

Reflections on the intersubjective space

A critical area of a narrative based approach is in the sharing of power between clinician and patient / client.

Every aspect of the sense-making process must be led so that the individual's, in this case Lisa's, own narrative is heard and isn't an entirely interpretative one that fits the clinician's bias or experience. Additionally, the concept of intersubjectivity is one that recognises that sense-making and understanding must be mutual.

A traditional biomedical approach involves dividing the patient's presentation into subjective and objective phenomena where the patient's knowledge, thoughts, perspectives and background become subjective and interpretative, while the clinician's understanding of exactly the same areas are objective and somehow closer to the "truth". This

paternalistic perspective of healthcare is not conducive to reflexivity, therapeutic rapport or balanced with respect to reasoning within this context. Recognising intersubjectivity appreciates that the patient undergoes the same sense-making process as the clinician, with exactly the same categorical areas of understanding but with different aspects of experience, knowledge and wisdom. Drawing from the individual's life experience and perspective is much more likely to facilitate understanding and knowledge translation (Launer 2018; Greenhalgh 2018) and improve the therapist – patient interaction (figure 1). For more information on this, a modification from the work of Øberg *et al* (2015) was presented in my paper (figure 2) on the clinical use of dispositions in clinical practice (Low 2017).

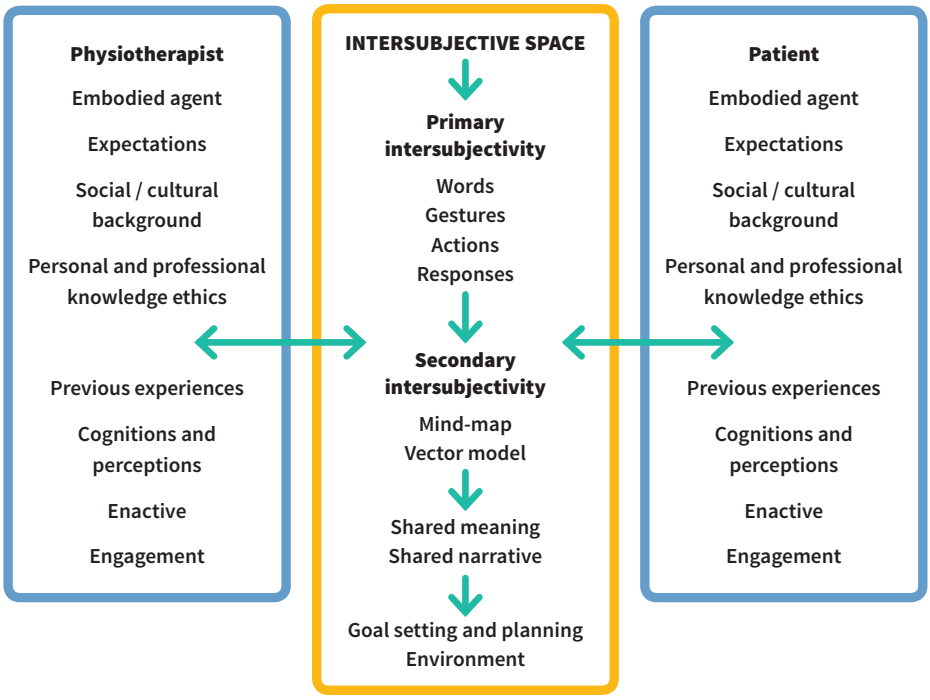

FIGURE 3: Summary of intersubjectivity interaction through intersubjective space

Continuing with Lisa's story highlights how clinicians might bring together the totality of the evidence (figure 3).

Concerns, preferences and expectations

Lisa believed that her spinal canal had narrowed due to the wear and tear that had taken place over her years of work as a nurse, and this was causing the pain in her back and her legs. She was concerned that she might be disabled in the future because of this. Her symptoms preoccupied her thoughts and she felt generally anxious. Lisa had discussed an epidural injection with colleagues but realised, following discussion with me, that the likelihood of it helping would be limited as her leg symptoms had resolved. Lisa was worried about the unpredictability of the pain and started to feel that it was becoming uncontrollable and unmanageable. She was feeling guilty that she was not fully at work and anxious about her future in her workplace.

However, despite the challenges, Lisa was open to my suggestions, comments and ideas. She recognised both the positive and negative effects that those around her might have on her pain and disability, whether by being overly protective, or providing frightening or unhelpful information, despite their good intentions.

PHYSICAL EXAMINATION

On examination, Lisa came within normal descriptions of sagittal and coronal balance, with no features of peripheral muscle atrophy or spinal deformity. Her neurological examination was normal with normal power, sensation and reflexes of the lower limb and down-going plantars. Her movements were braced and associated with grimacing and breath holding, she used her arms to steady herself during movements. She described constant central low back pain that was worse in the mornings and lasted for at least 30 minutes. Although Lisa had a mildly raised erythrocyte sedimentation rate (ESR) her C-reactive protein (CRP) was normal, as were other blood test results including full blood count. Lisa's fasting glucose was slightly elevated. Her raised ESR could be explained by the fact she now smoked and her increased body mass and may have been indicative of a mild degree of systemic inflammation.

Lisa's trunk movements were limited in all directions and her hip movements were full but causing low back pain at the end of range of movement. Repeated movements of the trunk demonstrated little insight due to pain-related distress and no clear centralisation or peripheralisation phenomenon. Neurodynamic testing of the lower limb was negative. Palpation revealed pain provocation of the local L4/5 region with

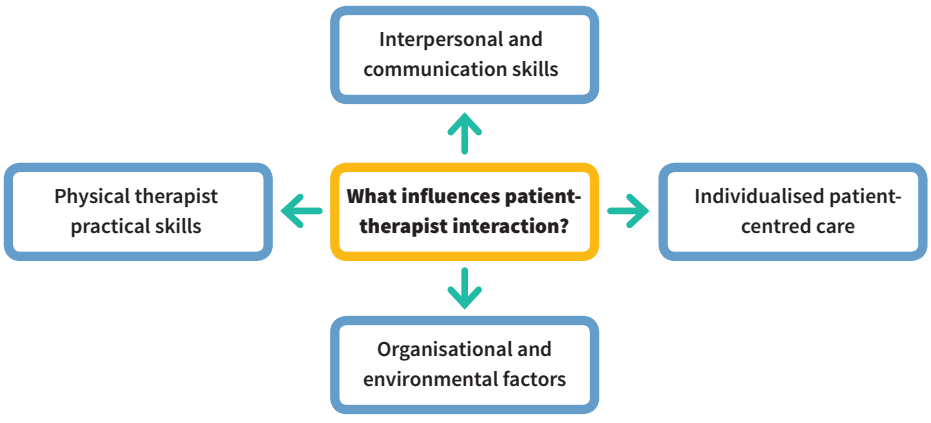

FIGURE 2: Patient / therapist interaction (O'Keefe *et al* 2016)

some reproduction of symptoms into both of her posterior thighs. Sacroiliac pain provocation testing was also negative. Light touch of the lumbar spine and surrounding areas demonstrated no clear features of allodynia. Lisa's STarT Back risk assessment tool revealed a high-risk score despite previously being a medium risk in her previous episode of care. This highlights the natural variation that the risk assessment tool has due to changes in context and circumstance.

Reflections on the tension between the normative and the narrative encounter

One of the most challenging tasks in health and social care may be in how to manage each encounter so that it continually meets both the narrative (humanistic) and normative (scientific) demands. This means recognising the equal legitimacy of the patient's need to express themselves, as well as our own need to conclude the clinical reasoning process, take action with consideration and adherence to guidelines, plan, and obtain closure. The challenge is to satisfy these needs at every point in the consultation in order to bring the normative questions and statements into the conversation at moments that fit with the natural flow of the patient's story. The tension that exists in this process grows owing to the constraints of time pressures on appointments, but when patients are given the space to talk, often with guidance, they reveal significant information that informs the normative account (Launer 2018).

My experiences and how they inform the situation

Having worked positively with Lisa in the past I was optimistic about her psychological flexibility (Kashdan 2010) in the context of her openness and in her ability to be reflective and reframe her thinking. If, during the examination, both Lisa and I could make sense of, and be able to predict and change her symptoms, it would significantly enhance Lisa's recovery. A key aspect of this was to identify what we both felt were modifiable causal factors. Looking at figure 3, the previous medical history (figure 1, box 1) includes a number of conditions amenable to exercise as a treatment (Pederson & Saltin 2015), namely hypertension, pre-diabetes, increased body weight, depression, anxiety, hypercholesterolemia, systemic inflammation (Dimitrov *et al* 2017; Woods *et al* 2012), and knee osteoarthritis (Fransen *et al* 2015). However, as much as these conditions are treated with exercise, from a social and emotional context many are barriers to exercise. Lisa had become fearful of activity because of the pain and felt little confidence in exercise, particularly in environments surrounded by healthy individuals. Exercise was clearly, therefore, a clinical goal but first we needed to establish a level of activity through methods that Lisa felt comfortable with in order to gradually build her confidence. The mind-map may provide a pathway for cognitive reassurance, but this alone was not going to be sufficient to enable change; Lisa needed to experience it.

By altering her movement behaviours using verbal and / or visual feedback, or by facilitating movement through the therapeutic sense of touch, Lisa might see that her symptoms could be altered which would give her a sense of control. It was important that Lisa should be able to do this herself as soon as possible as it would help to reduce her anxiety levels and the unpredictability of her symptoms. If we could find reproducible movements and / or postures to provide a consistency and control of her symptoms, the prognosis of her outcome was more likely to be favourable.

Lisa's workplace appeared to be very supportive and by contextualising our mutual understanding of her symptoms in relation to lifting loads we made them less threatening and less painful. Ensuring that her colleagues and family members were also aware of this change in perception would greatly enhance Lisa's outcome.

In this regard, significant amounts of time and effort were required to change Lisa's perspective on manual handling. She had lived by a rule-driven system grounded in a biomechanical context and so, at some stage, it was likely that she would benefit from behavioural experiments, mimicking lifting, carrying and twisting with loads that she might be exposed to in the reality of the workplace. She might also need to consider other components of movement strategies, including strength and capacity for load, recovery strategies including sleep, hygiene, diet, and ensuring that she could move a variety of ways.

> "WHEN PATIENTS ARE GIVEN SPACE TO TALK, THEY REVEAL SIGNIFICANT INFORMATION THAT INFORMS THE NORMATIVE ACCOUNT"

Finally, at the point when she was in a position to reduce her pain levels and gain control over her symptoms, Lisa's medication could be reduced in the long-term (figure 1, box 2). This would be an achievement not only for Lisa's health and wellbeing, but also is an often unrecognised benefit to our health economy.

Considering NICE guidelines

The NICE guidelines (2016) suggest the following for patients with LBP +/- sciatica:

- Provide information tailored to their needs and capabilities to help them self-manage, including the nature of low back pain and sciatica, and encouragement to continue with normal activities.
- Consider a group exercise programme taking specific needs, preferences and capabilities into account and consider manual therapy as a part of a treatment package (e.g. with exercise) with or without psychology.

> "WHEN EVALUATING THE EVIDENCE, ASK YOURSELF: 'HOW DOES THIS STUDY RELATE TO MY PARTICULAR PATIENT AT THIS PARTICULAR TIME?'"

- Consider psychological therapies using a cognitive behavioural approach but with exercise with or without manual therapy.

There is, however, considerable scope for more detail. There appears to be no additional guidance on what clinical information is or is not helpful, or indeed what the group exercise would entail, or what would be most effective. This is not due to the approach of NICE, but more to do with the generally questionable quality of research, and the low statistical powers required to judge confidence in treatment effects. Common sense may have already directed us to suggest that Lisa's cognitive, affective, behavioural, social and work components indicate that she will respond to a psychologically informed approach, underpinned by an exercise component, with or without manual therapy to facilitate her recovery.

Reflections on evidence-based physiotherapy

On searching and evaluating the clinical research with low back pain +/- sciatica there is a dearth of literature, and the quality of what there is in the context of evidence-based physiotherapy (EBP) has been based upon a hierarchy of evidence originating from the model created by our medical colleagues. The original evidence-based medicine (EBM) movement was devised from the field of clinical epidemiology (Guyatt 1992). Here, randomised controlled trials (RCTs) are the gold standard and, from a perspective of internal validity in comparison to other methodologies, quite rightly so. Rigorous RCTs reduce the risk of bias by controlling for confounders through the use of specific exclusion criteria. This creates a risk of poor external validity, in other words the research is difficult to generalise, or infer the results outside of the study itself.

So, when evaluating the evidence, one must ask oneself, how does this study relate to my particular patient at this particular time? With increasing multi-morbid contexts that are growing in number in the MSK patient population, the likelihood of patients who have been through RCTs being excluded or not being representative of the patient in your clinic is pretty high!

In Lisa's case, she had a number of co-morbidities (figure 1, box 1). It appears that the majority of clinical research, even in the large size RCTs where the numbers of subjects may provide a greater match to a statistical average, do not reflect in any way what Lisa may respond to. What appears clear is that population level research outcomes are no constitutive of understanding causation, but merely symptomatic of it. The real causal matter arises from the individual case. Population data may serve as a map but it does not describe the terrain.

So, here is a paradox, as Roger Kerry (2017) explains here through quoting Pearce (2015):

"RCTs are designed to reduce, as much as possible, the biases found in traditional knowledge sources, e.g. expert judgment. Thus EBP de-emphasises the role of expertise as a source of evidence of therapeutic effectiveness."

"RCTs provide the best evidence of therapeutic effectiveness in the trial sample population, but this is a different concern to treatment effect in the target population, e.g. patients outside the trial (Cartwright 2007, 2011).
Thus, additional evidence is required to inform clinical decision making in this population. This process necessitates expert subject knowledge to make judgements about external validity, as well as experience and expertise in integrating multiple sources of evidence… Thus, after de-emphasising expert judgement in its pursuit for unbiased data, such judgement is necessarily reintroduced in the social application of such data."

Reflections on Louis Gifford's shopping basket approach

Using Louis Gifford's shopping basket approach (2003) is an extremely useful way to unpack the clinician's knowledge and understanding of the presenting case. The shopping basket covers a huge amount of ground, including the biomedical, psychological, social, cultural and experiential components of the clinical encounter. Fusing both the shopping basket approach and a dispositional account (Low 2017) with the vector model may provide opportunities for greater insight, understanding and collaborative treatment.

A dispositional clinical approach is underpinned by a philosophical view of causation, by which I don't mean a simple concept of necessary and sufficient conditions that can be measured by how frequently an effect is observed. Causation makes far more sense if causal powers have a direction and strength towards, or away from an effect and that it occurs in non-linear ways.

With permission from Lisa, I drew a mind-map (figure 4) of her circumstances and contexts as I saw them and asked her to tell me if there was anything missing, or indeed, if she felt anything could be added. This then informed the vector model, which created a co-constructed account of how much each of the causal factors were felt to contribute to or counteract her symptoms.

I encourage further reading on the work of Rani Lil Anjum and Stephen Mumford (2011), both of whom engage exceptionally well with our community of healthcare professionals. Their fusion of the perspectives of the humanities with the sciences encapsulates

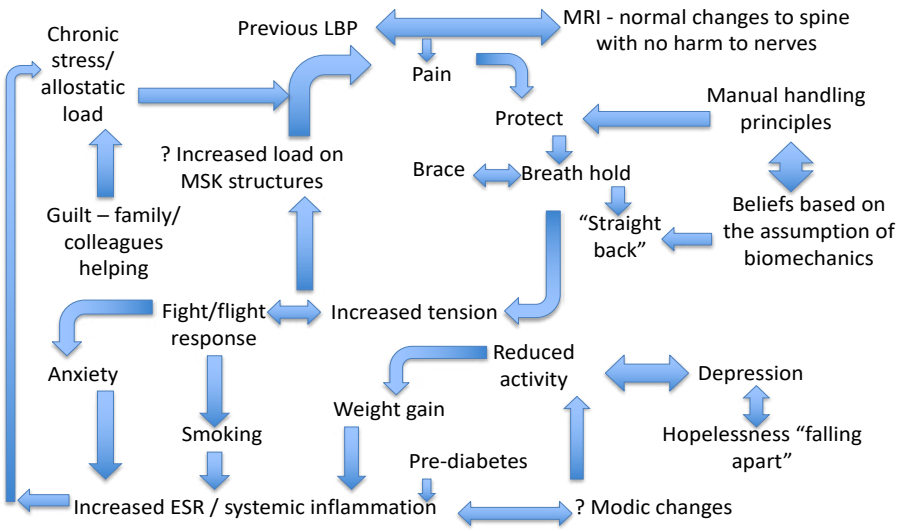

FIGURE 4: The patient mind-map

biomedical, psychological and social contexts under a canopy of integrated biology. Their vector model (figure 5) and underlying philosophy view causation as an inherently context-dependent and dynamic process that values uniqueness over the current narrow view of the observed frequency of symptomatic effects. This may provide us with a broad framework that balances the normative and narrative examinations, and methods of understanding causation in the context of person-centred care and management, that can be implemented in a clinical setting.

Treatment, management and action

By using the vector model with Lisa and producing a hypothesis on the interaction of the identified causal components through the mind-map, it allowed a discussion to emerge on what she felt to be more amenable to change.

I evaluated her movement behaviours, choosing three specific movements involved in bending, sitting to standing and walking. First of all, movements into painful directions were facilitated in offloaded positions. For example, the act of bending was practised in side lying with passive assistance and verbal feedback was given to recognise her instinctive bracing behaviours. Lisa started to recognise this and allowed herself to "sink into bending", her pain started to ease in this position. Then she practised seated bending, all the time trying to reproduce the same sensations as when side lying. Initially, with cervical and thoracic flexion, then a relaxed exhalation, allowing the movement of gravity to assist her downwards rather than resist the speed of the movement. This was initially repeated 10 times, with a short rest period and with Lisa making the effort not to brace or hold her breath. Lisa was immediately able to perform this previously painful movement with less pain, and she could see that her symptoms were modifiable. This was repeated numerous times to confront her anxiety and fear of flexion.

We used exactly the same pattern and method of cueing in standing. Firstly with the legs in abduction to increase the base of support and allow Lisa to be closer to the end position. Again, Lisa could flex repeatedly and with feedback, if she felt her back tighten up, I encouraged her to reset, breath and relax and repeat. From sit to stand, Lisa was reminded of the same movement patterns, cervical flexion, thoracic flexion and to take the trunk forward and stand up. If the patterns of cervical or thoracic extension started to occur I provided manual feedback to facilitate the movement. Initially the central key points used in neurological rehabilitation were the most helpful, and within a short period of time I could feedback with minimal use of touch and then with feedback from video footage recorded on her phone.

During walking I encouraged Lisa to swing her arms to allow her chest to drop an inch to facilitate an element of flexion and rotation in the trunk, following which I asked her to quicken her step and to increase her stride length. This exercise appeared to allow Lisa to cover more ground and be in less discomfort.

Within a week of starting these movement patterns, Lisa began to feel

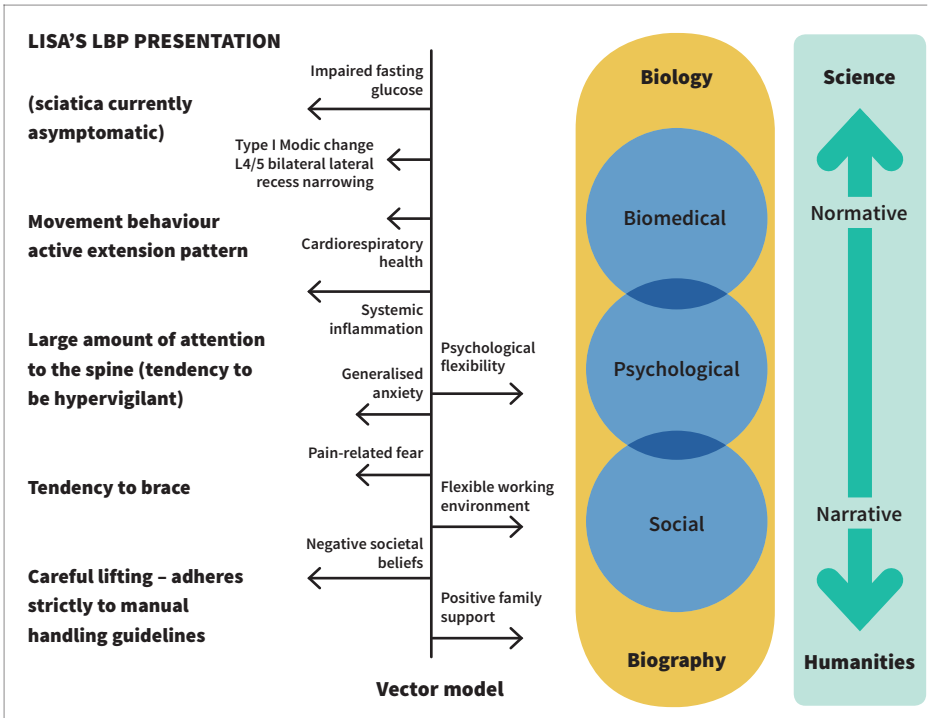

FIGURE 5: A fusion of perspectives

that progress was being made. Her pain, although still present, had lessened and she was starting to exercise with more confidence. Occasionally, her symptoms would flare up, but she was reassured to know that if she adopted the relaxed and more flexed positions, she could control them. Lisa reported that her symptoms had become more predictable which enhanced her self-efficacy.

We identified that the factors Lisa felt most empowered to change were a calorie-controlled diet and concentrate on movements modified to break the negative cycle in response to pain. I uploaded the Live Well Dorset website (**www.livewelldorset.co.uk**) which enabled me to give Lisa access to their one-off, 12-week dietary support plan with Weight Watchers or Slimming World.

The website also provides support and information on how to stop smoking and how the user can access local opportunities for becoming more physically active.

Lisa had a static bike at home that was being used as a clothes rack so I suggested that she try a static bike in the therapy department to see how she responded to the activity. We discussed cycling positions and the general concepts of seat height and resting positions on the bike. I made sure that Lisa felt that she could adjust the bike to her own comfort and that she was aware that there was no specific or absolutely "correct way". This reassured her that she could start using her static bike at home.

Access to Slimming World and using her static bike every day meant that Lisa began to lose weight and, three months on, her repeat fasting blood glucose levels had improved, as had her symptoms, and she was back to full time work.

At this stage Lisa joined a local gym and took part in Zumba and gentle body pump classes. She created a new narrative with regard to manual handling. She now believed that she should be stronger and fitter to work with manual handling rather than keep her biomechanical postures correct. She altered a few of the exercises in the classes to suit her preferences and, over a six-month period, Lisa felt that she was, on the whole, pain free. Occasionally, her symptoms would return but she felt in control of her pain and could refer to her version of the vector model and make self-adjustments. Lisa started with increasing her levels of physical activity and ended with regular planned exercise over the recommended 150 minutes a week. She felt more positive and less anxious and developed new friendship groups at her local gym.

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Final reflections and conclusion

This case resulted in a positive outcome and Lisa was a pleasure to support. Other cases may present with many causal elements that appear uncontrollable and have very challenging prognoses. Perhaps a vector model approach with a greater emphasis on the causal contexts that the patient feels that they can control, with greater dosage and perhaps greater effect or counteraction may be of benefit in such cases.

In Lisa's situation the therapeutic alliance allowed for sense-making, reflexivity and context dependent decision-making that drew on the totality of the evidence and not clinical research in isolation. The treatment plan resulted from conversations that were immersed in curiosity, engaged in the complexity of context, recognised Lisa's challenges that were treated with caution to her ideas and preferences, and with the utmost care for Lisa as a person.

About the author

Matt is a Consultant Physiotherapist at a Foundation NHS Trust on the South Coast. He qualified from Southampton University in 2003 and has worked in the NHS since. He is an accredited Clinical Educator (ACE) from the University of Brighton and has been a member of the Musculoskeletal Association of Chartered Physiotherapists (MACP) since 2010. He also works as an Extended Scope Practitioner (ESP) in back pain.

Matt has lectured and examined at local universities and teaches undergraduate and postgraduate physiotherapists on topics such as motor control, spinal manipulation and clinical reasoning skills. He completed his MSc in Neuromusculoskeletal Physiotherapy and has interests in compassionate person-centred care, the theory of causation in medically unexplained symptoms, philosophy, reflective practice and critical thinking skills.

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Useful websites

ARMA
http://arma.uk.net/musculoskeletal-networks/network-resources/

National Back Pain Pathway
https://www.england.nhs.uk/blog/charles-greenough/

NHS England
https://www.england.nhs.uk/ourwork/ltc-op-eolc/ltc-eolc/our-work-on-long-term-conditions/si-areas/musculoskeletal/

https://www.england.nhs.uk/ourwork/ltc-op-eolc/ltc-eolc/resources-for-long-term-conditions/

Reasoning exercise dosage for people with persistent pain

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It is universally accepted within society that activity, i.e. exercise that raises physical stress on the body, is important for overall health. However, the suggestion of exercise to the person living with persistent musculoskeletal pain can be a daunting one. The unpredictability and unpleasantness of musculoskeletal pain following exercise for the person living with long-term pain can be unsettling and finding the right dosage can be challenging for the practising clinician. This article aims to critically appraise the evidence with the intention of providing a clinical rationale for reasoning exercise dosage for people living with persistent musculoskeletal pain.

LEARNING OUTCOMES
TO SUPPORT PHYSIO FIRST QAP

1 Understanding the common exercise barriers faced by physiotherapists.
2 Investigating the physiotherapists' perception of the pain and exercise relationship.
3 An increased awareness of using exercise dosage in clinical settings.
4 Reviewing the clinical application of exercise dosage.

Introduction

The Exercise is Medicine (EIM) movement developed by the American College of Sports Medicine (ACSM) attempted to address the increasing rate of chronic disease worldwide. Unfortunately, the message "some is good, more is better" failed to sufficiently specify what exercise dosage is considered to be appropriate, particularly with regard to the heterogeneity of those individuals living with chronic disease (Williams *et al* 2017).

Exercise is a key component of the physiotherapist's management repertoire, and physiotherapy has, historically, neglected a broader holistic view of the patient in favour of a reductive perspective of the body, which has resulted in treatment (exercise) being focused on pathology (Nicholls 2018). While this view is the mainstay in the treatment of most acute musculoskeletal conditions, it is ineffective in the management of those patients with persistent pain where causal links to pathology cannot be substantiated (Eriksen *et al* 2013). It is also noteworthy that we cannot be nearly as specific in isolating individual muscle groups with our exercise programmes as we have commonly understood (Mannion *et al* 2012; Reinold *et al* 2004; Unsgaard-Tøndel *et al* 2010), therefore the need to be specific with regard to exercise may be less important than prescribing the appropriate dosage.

All physiotherapists have experienced the anxious expression, the dejected look, the frustrated frown of the individual living with persistent musculoskeletal pain who is asked to partake in exercise. In many cases, where physiotherapists are concerned about causing more harm with exercise, a person-centred evidence-based approach may permit opportunities that would be limited by a reductive perspective. The implementation of a biopsychosocial (BPS) approach and, to a larger extent, aiming to provide a rationale for appropriate dosage of exercise for individuals living with persistent pain is, therefore, of particular importance.

The benefits of exercise in chronic musculoskeletal conditions have been researched extensively (Ambrose & Golightly 2015; Geneen *et al* 2017; Hassett & Williams 2011; Pedersen & Saltin 2015) and, while exercise is generally advocated as an effective means to manage chronic musculoskeletal pain, it can be challenging to determine what is appropriate for the individual at the time of exercising. In addition, research has identified that physiotherapists with a more biomedical tendency may unknowingly be perpetuating the belief that pain equals damage by encouraging patients to stop exercising when they experience pain (Bunzli *et al* 2015; Simmonds *et al* 2012; Synnott *et al* 2015) Furthermore, while exercise may be linked with increased pain for individuals with persistent pain, it is unlikely to result in exercise-induced harm if dosed appropriately (Booth *et al* 2017; Geneen *et al* 2017). The International Association for the Study of Pain (IASP) has recognised that this increase in pain may be as a result of "nociplastic pain", i.e. changes in the function of the nociceptors that can occur in persistent pain states, as can "central sensitisation", i.e. an amplification of neural signaling within the central nervous system (CNS) that elicits pain hypersensitivity (Hainline *et al* 2017; Woolf 2011). This facilitates the understanding that pain

> "IT IS PARTICULARLY IMPORTANT TO PROVIDE A RATIONALE FOR APPROPRIATE DOSAGE OF EXERCISE FOR PATIENTS LIVING WITH PERSISTENT PAIN"

in a persistent state is more about sensitivity as opposed to tissue insult, which can be helpful for reinforcing the message that "hurt is not equal to harm". Therefore, clinicians should be mindful of the temptation to reduce exercise when "source searching" or attempting to identify the cause of nociception.

Pain can become pervasive in the individual's life, which unsurprisingly can lead to anxiety, decreased willingness to engage, and low self-efficacy towards exercise (Crombez *et al* 1999; Eccleston & Crombez 1999; Miles *et al* 2011; Russell *et al* 2018).

Physiotherapy, pain and exercise

The roots of physiotherapy are embedded within medicine, establishing itself as a provider of health promotion and re-enablement in three main areas of care; cardio-respiratory, neurological and musculoskeletal. Exercise and education are the foundations of the management of persistent pain in physiotherapy and, although exercise is an effective treatment for various persistent musculoskeletal pain disorders (Daenen *et al* 2015; Nijs *et al* 2012), prescribing exercise without appropriate clinical reasoning is ineffective without a broader understanding of the individual's circumstances.

Despite its misrepresentation (Pincus *et al* 2013), the biopsychosocial model (BPSM) provides the clinician with a broader framework of clinical indicators to ascertain the nature of the individual's presentation. Using a BPSM approach recognises that there are factors that require investigation beyond, but without ignoring, biological reasoning. Pain is an emergent phenomenon based on multiple factors, which can have implications on the clinical presentation at each session (Booth *et al* 2017).
In their recent paper, Tousignant-Laflamme *et al* (2017) rehabilitation research initiatives for low back pain (LBP) propose a pain and disability management model (figure 1) that triages the level of complexity and the intervention needed without reducing the BPSM into its respective parts.

In doing so, the clinician should consider the following reflective questions.
1. Why is this person presenting in this way, at this time?
2. How can we get the person going again?
3. What can be done to reduce distress and disability?

Utilising a BPSM approach and critical questioning can be helpful to determine how clinicians can:
(a) titrate exercise dosage, and
(b) make the person more comfortable when engaging in activity.

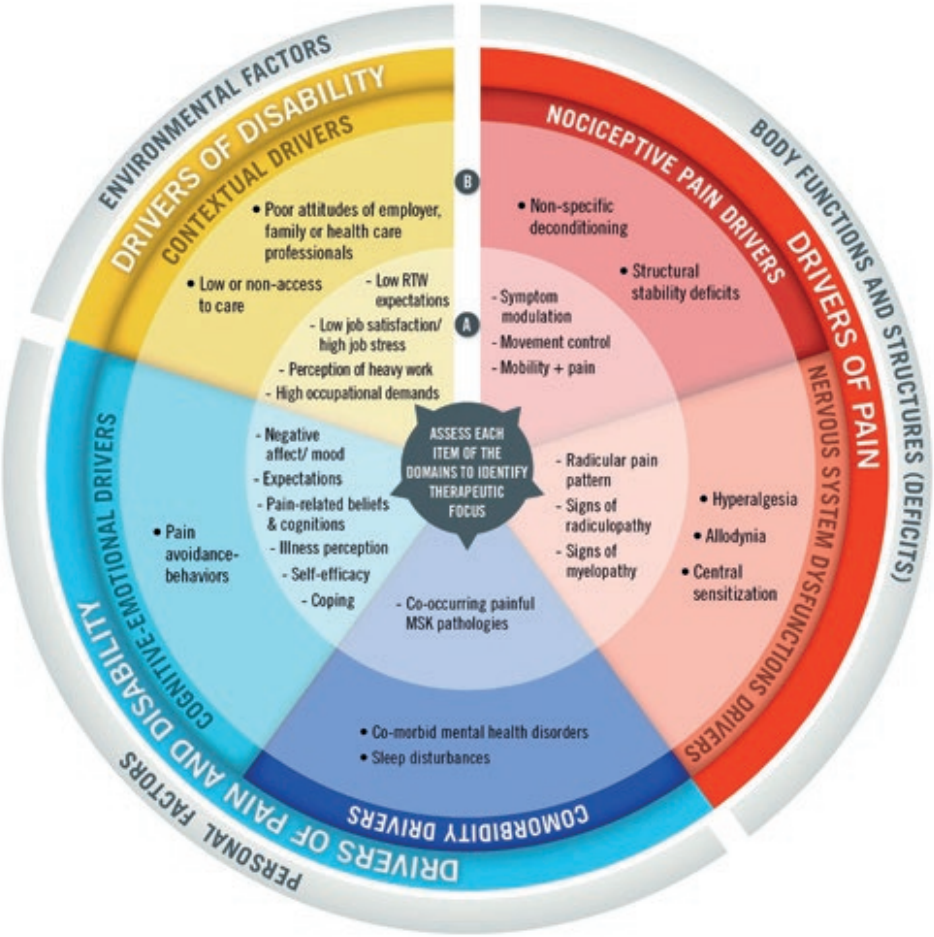

FIGURE 1: The Pain and Disability management model. Figure reproduced with kind permission (Tousignant-Laflamme *et al* 2017)

Physiotherapy and DeLorme's protocol

When it comes to exercise dosage, physiotherapy has historically been enamoured with advocating the three sets of 10 repetitions (reps) format. Early work on progressive resistance training by army physician Thomas DeLorme identified that injured US servicemen returning from the Second World War made significant improvements in function by following this exercise format and so DeLorme's protocol, understood to be the number of sets and reps

required to build strength, became the model of choice in many rehabilitation and fitness centres. It appears that the consensus behind its use was the assumption that people in pain, and / or injured, were weak (Todd *et al* 2012).

However, perhaps a more pertinent approach considers questions such as: "does this training method focus on the needs of the individual?", or "does it focus on the idea that, if the body part becomes stronger, pain will attenuate?"

Exercise induced hypoalgesia (EIH) has been researched extensively for several principles of training (Daenen *et al* 2015; Nijs *et al* 2012, Rio *et al* 2015, Smith *et al* 2017a). A recent systematic review (Babatunde *et al* 2017) identified that exercise therapy showed moderate to strong evidence for back, neck, shoulder, knee and multi-site pain, with beneficial effects in the short and long-term. Interestingly, exercise that was specifically adapted to the activities of daily living (ADLs) appeared to have greater positive effects than exercises that were not. This may provide insights into social determinants of exercise prescription.

While I would suggest that a large majority of physiotherapists attempt to integrate physical activity principles and social determinants into their practice, as Babatunde *et al* (2017) highlight, there is a paucity of evidence, with no clear guidelines for how clinicians may proceed with the delivery of exercise for people living with pain.

Further questions that arise from this are:

- What about exercising when in pain?
- What is the right dosage?
- What is the minimally effective dose to facilitate progression?

Exercise and pain

In healthy individuals subjected to experimental pain, a bout of aerobic exercise at 70% of VO2max provides up to 30 minutes of EIH (Daenen *et al* 2015; Koltyn 2002). However, several chronic pain populations, such as those with fibromyalgia, complex regional pain syndrome, chronic fatigue syndrome and chronic whiplash associated disorders, display the opposite effect, whereby exercise is pain-inducing (Daenen *et al* 2015; Daly & Bialocerkowski 2009; Hassett & Williams 2011). Recent research from Lima *et al* (2017) has indicated that for these populations, despite acute bouts of exercise resulting in an increase in their pain, EIH may, with time and appropriate titration, be facilitated by further regular exercise.

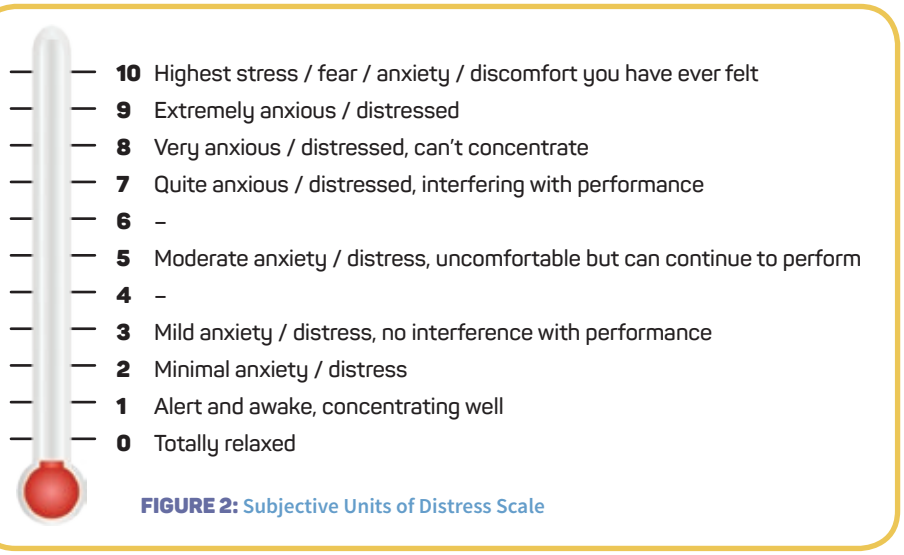

FIGURE 2: Subjective Units of Distress Scale

A systematic review and meta-analysis from Smith *et al* (2017b) indicated that painful exercise produced self-reported short-term benefits compared with pain-free exercises. It should be noted that the majority of papers reviewed specified that participants were encouraged to exercise with the understanding that pain would not exceed 5/10 on a Visual Analogue Scale (VAS), a frequently used method of measurement for pain intensity, and that symptoms would subside prior to the next bout of activity. While these studies are useful in demonstrating the utility of exercise on pain modulation, reliance on the VAS fails to capture the multidimensional nature of pain (Mannion *et al* 2007; Saragiotto *et al* 2016).

The Subjective Units of Distress Scale (SUDS) (Back *et al* 2015) is useful as it draws attention away from pain (sensory), shifting the focus instead to concerns (psychosocial) about performing a certain activity (figure 2). For example, a person may feel anxious about bending forward to pick up their keys from the floor but be happy to sit down and bend forward to put their shoes on, which is effectively the same movement but a different context. Incorporating a measure such as the SUDS exchanges the word "pain" for the word "distress", thus considering the wider psychosocial factors that so commonly accompany the postural, structural, and biomechanical aspects of persistent pain.

Smith *et al* (2017b) identified that individuals exercising with pain experienced favourable outcomes in the short term, reassuring both clinicians and patients that it is OK to exercise with pain. A caveat here, however, is that pain is subjective, and a 5/10 for one person may be a 2/10 or 8/10 for another when performing the same exercise. This may also be the case across different sessions for the same person. Using a pain scale as a means to gauge training and dosage in individuals is therefore not wholly reliable or effective, so titrating exercise dosage seems like an appropriate consideration.

One popular approach to exercise or activity in pain management is pacing. It appears in many acute injury treatment programmes where the patient is advised to gradually return to daily activity routines. Pacing is commonly implemented as one of the "3Ps", together with planning and prioritising. For some individuals with persistent pain, pairing pacing with gradual increases in activity, and close

monitoring via a time-based approach can work towards allowing them to do more activity without experiencing the dreaded pain flare-up.

Finding the right dosage

Like finding the right medication dosage, considering the exercise dosage for the individual living with pain is complex as we are still currently unsure exactly how exercise works for pain, due to its multidimensional nature.

Dosage consists of the frequency, i.e. the number of times an individual typically exercises in a week, the intensity with which the individual exercises, and the duration of the individual's exercises, recorded by time. In some cases, added to this the type of exercise performed, i.e. aerobic, weight training, etc. and the load, i.e. how much the individual lifts, can also be considered. Load is not primarily represented by weight, it can also include the influence of several factors affecting homeostasis in the individual (Hainline *et al* 2017; Soligard *et al* 2016).

Anecdotally, dosage can also influence threat perception. If an individual displays apprehension towards lifting more weight it might be worth considering a change in reps, sets or frequency, such as changing from performing the activity every day to every other day.

The versatility of using dosage means that the variables can be modified throughout the programme to accommodate any changes in the patient's presentation at each session. Through some very basic sums the clinician can also maintain a record of actual work done and this can be displayed as an arbitrary number of volume (figure 3). As the patient's attention shifts from a pain focus to a focus on volume, pain often tends to become less of a problem.

It is important to always consider, and not detract from, the goals and values of the individual when prescribing the appropriate exercise dosage, i.e. if the aim of your patient is to be able to get up from, and down to, the floor throughout the day, then it is better to prescribe low reps and gradually increasing load, as opposed to higher reps on a lower load that would be more suitable to someone who needs to repeat a movement over and over again.

EXAMPLE

An individual with persistent back pain wants to increase their cardiovascular stamina and the agreed plan is to exercise for two supervised sessions a week, for 10 minutes duration per session, on an exercise bike at a resistance of 4, resulting in a rate of perceived exertion (RPE) of 6. The total volume of this exercise dosage would be 480 (2 x 10 x 4 x 6 = 480). However, this exercise programme turns out to be unsustainable for the patient and they only manage to complete 2 x 5 mins x resistance 4 x RPE 6, resulting in a total volume of 240 (2 x 5 x 4 x 6 = 240).

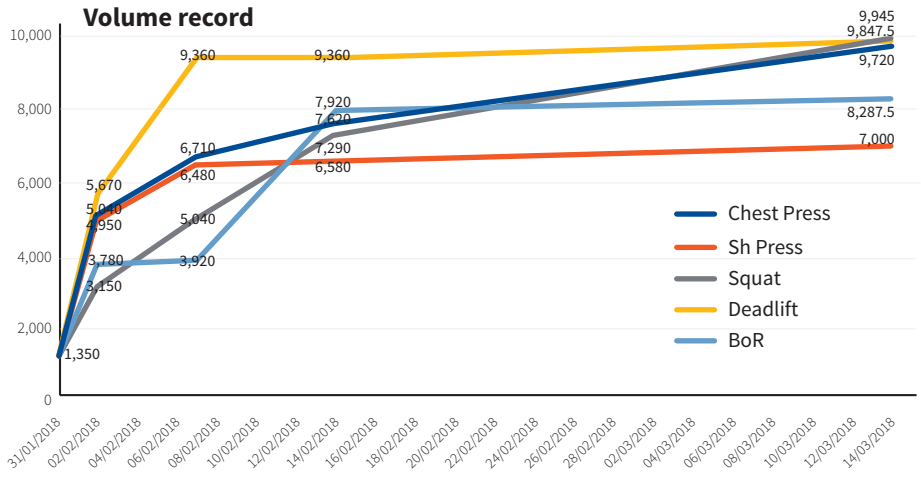

FIGURE 3: An example of tracking volume changes

> "ALWAYS CONSIDER THE GOALS AND VALUES OF THE INDIVIDUAL WHEN PRESCRIBING THE APPROPRIATE EXERCISE DOSAGE"

However, by altering the variables it would be possible to reach the desired volume. By breaking the 10 minutes into two-minute periods, the volume calculation would look like this: 2 session per week x 5 bouts of 2 minutes x resistance of 4 x RPE of 6 would be 480 (2 x 5 x 2 x 4 x 6 = 480). Giving rest periods in between each bout is likely to encourage more adherence to exercise, and hopefully lessen the patient's disruption from pain.

While it may take some time for the patient to begin to see an improvement in their self-reported pain, once they do the pain scales or SUDS may be used by the clinician less and less.

What is the minimally effective dose to facilitate progression?

For the clinician working with pain management, this question represents the challenging aspect of any exercise programme. How do you prescribe the minimally effective dose? The Goldilocks Theory, based on the famous fairytale, aims to find a level of activity that is not too much or too little, but just right.

In my clinical experience this is reliant on several factors:

1. The clinical formulation from the subjective history.
2. The established therapeutic relationship between the clinician and patient.
3. Use of psychometric screening measures.
4. Trial and error during the sessions.
5. A willingness of the patient to change or participate.

> "A PATIENT WHO HAS EXPERIENCED A FLARE-UP OF PAIN AFTER EXERCISE MAY BE RELUCTANT TO EXERCISE IN THE SAME WAY AGAIN"

6. What effects have occurred during, immediately following, and in the days after the exercise sessions.

In considering these factors, as well as the questions that were highlighted previously, we can often re-formulate what should be appropriate for the next session. A patient who has been pushed to the limit during an exercise session, and subsequently experienced a strong flare-up that has lasted for a few days, may be reluctant to exercise again in the same way. This is where collaborative skills come in; to negotiate with the patient to titrate, i.e. changing the intensity, load, reps, etc. of the dosage to a level that will still provide adaptation but reduce the risk of a further flare-up. It is important to emphasise that flare-ups will happen, but also to reiterate the message that hurt does not equal harm.

Using visual aids, technology, and recording the volume of work performed are helpful ways of representing forward progress. However, while individuals and clinicians may want to see a steady improvement in activity levels, the chaotic nature of pain may create obstacles along the way. Progress with persistent pain is never straightforward, and when it is necessary, at the start of treatment, to titrate dosage it is best that both the patient and the clinician avoid becoming hung-up on a linear progression of exercise work rate.

Finally

There is always the opportunity for the clinician to just get their patients exercising either aerobically, on a distal body part, or on the non-injured side if rehabilitating a limb as this offers neurophysiological effects (Booth *et al* 2017; Daenen *et al* 2015; Hendy *et al* 2012). Furthermore, reframing exercises as activity means that dosage could be applied to pursuits that people value such as surfing or walking. In addition, by providing tools and understanding with regard to the individual's experience during exercise or activity may help to encourage increased self-efficacy and an internal locus of control.

About the author

Paul qualified as a Physiotherapist in 2008 and immediately plunged into the world of persistent pain, initially working in a GP practice in Fulham, London before moving to a senior role in the NHS. His exposure to persistent pain grew through working alongside specialist physios in the NHS and through regular observations at the Centre of Pain Education based in South London.

Later, Paul decided to explore the world and settled in New Zealand where he now practices in a private setting working primarily in pain management.

Paul is the creator of the naked physio blog and has a strong presence on social media. He has written articles for other health professional journals and spoken at conferences in New Zealand and internationally.

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Letter to the editor

Members Benevolent Fund

The Editor – "In Touch"
Physio First,
Minerva House,
Tithe Barn Way,
Swan Valley,
Northampton, NN4 9BA

Dear Editor,

I am writing on behalf of the Trustees of the CSP Members Benevolent Fund to ask if the following could be printed in the next edition of "In Touch" in response to the fabulous donation recently made to the MBF please.

"The Trustees of the CSP Members Benevolent Fund would like to once again thank members of "Physio First" for the collection of over £1,000 for the MBF at the April conference.

The money will be used to assist the beneficiaries that little bit more and give additional assistance to those really in need. The numbers of beneficiaries continues to rise and it is always good to hear when someone can manage again without assistance. Many beneficiaries have sad and tragic tales, and the average age is in the 30's to 40's with many due to illness and family breakdown. We continue to hear from those in private practice who have not insured themselves against loss of earnings – usually in the early days of setting up their private work, and who have students loans and other debts which catch up in unforeseen circumstances, whatever the cause.

It is good to be able to assist our colleagues in this way and the donation from Physio Fist will really assist where it is needed most. One never knows what is around the corner especially but this will assist our colleagues to get back on their feet when things have taken a turn for the worse. Thank you all so much for your generosity.

For further information about the MBF please visit the CSP Website at www.csp.org.uk/MBF.

Dorothy MF Toyn
Chair, CSP Members Benevolent Fund."

Many thanks for your assistance in this matter.

Yours Sincerely

Dorothy Toyn

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Chair, CSP Members Benevolent Fund.
Dorothy.toyn@ntlworld.com

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We will be trialling joint funding of a grant application made to the CSP Charitable Trust and the results will be closely monitored to ascertain its feasibility to continue.

We, as PPEF Trustees, look forward to working with the CSP and we are hopeful of the mutual benefits.

We will of course continue to fund grants on applications made directly to the PPEF and continue our special relationship with Physio First.

Details about the PPEF can be found at **www.ppef.org.uk**

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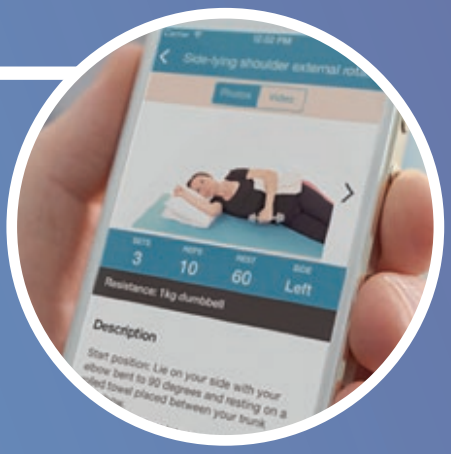

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It is a UK-based company committed to creating innovative evidence-based rehabilitation solutions that are user-friendly for physiotherapists and their patients.

The company was founded by HCPC-registered physiotherapist and strength and conditioning expert Ian Prangley. He has master's degrees in both Sports Physiotherapy and Exercise Science and has worked in private physiotherapy clinics, the Australian Institute of Sport physiotherapy department, as the Australian Davis Cup team physiotherapist, and for the last four years has guided the physical development of Kyle Edmund, Britain's no.1 tennis player. He is supported by an experienced team of technology professionals.

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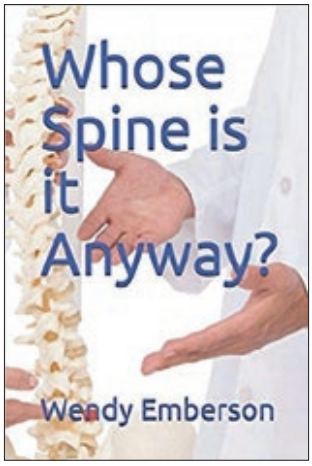
To learn more about TrackActive, please visit our commercial partner page by navigating to **www.physiofirst.org.uk/resources/commercial/trackactive.html** where you can access your discount code and sign up for a free 30-day trial. If you have questions for the TrackActive team, please email them at **support@trackactive.co**

Respiratory muscles and back pain



Whose Spine is it Anyway?

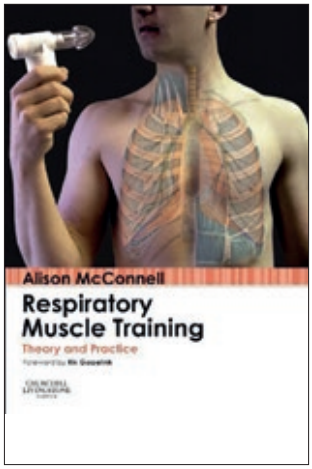
Wendy Emberson

Publisher: Independently published | ISBN: 978-1521359778
RRP: £15
Paperback: 149 pages

This is written with passion and from the heart! The semi-biographical content takes on "back pain" from the perspective of a seasoned physiotherapist and long-time Physio First member. In it, Wendy explores the reasons for the increased incidence of back pain, what physiotherapy can offer in terms of assessment, treatment and self-management, and the areas where the medical system fails patients.

In addition to its clinical aspect, this book summarises a lifetime of work, knowledge and experience, and what's more, I found myself nodding in agreement with Wendy with the turn of each page.

Tobias Bremer



Respiratory Muscle Training: Theory and Practice

Alison McConnell

Publisher: Elsevier | ISBN: 978-0702050206
RRP: £52.99 (hard cover) £34.22 (Kindle)

This is a very methodical approach to the why, when and how to approach respiratory training. It starts off with the usual introduction to the anatomy and physiology of the airways, including blood supply and skeletal components. I particularly enjoyed the explanation of how breathing is controlled, and the mystery that still surrounds its function.

The well-written chapter on exercise physiology and training principles critically addresses all the facets associated with this subject, such as why do muscles fatigue? Are we measuring this correctly? And this is followed by the equally as good chapter on the effects of various pathologies on the respiratory muscles, but I feel the book starts to lose its way after this point as it fails to translate all this theoretical know-how into a practical setting.

I think the content would benefit greatly from details highlighting assessment and outcome measurement tools that enable the clinician to work more specifically on, and benchmark a patient's deficits. The exercises suggested serve as a good starting point on which to base a training programme, but again I feel that without the assessment skills and use of outcome measures these are just a stab in the dark as the efficiency of the programmes is unmeasurable.

My comments may be a little hyper-critical as I did enjoy this book but, as we move towards the prospect of needing to prove our quality assured status as private practitioners, it is exactly these subtle differences in being able to assess, treat and measure our efficacy that will set us apart as providers in our changing healthcare marketplace.

Tobias Bremer

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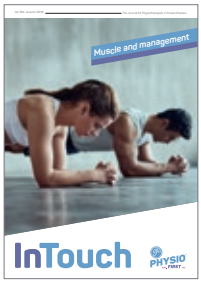

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For further information, please call Simon Lively at M&L Healthcare Solutions:-

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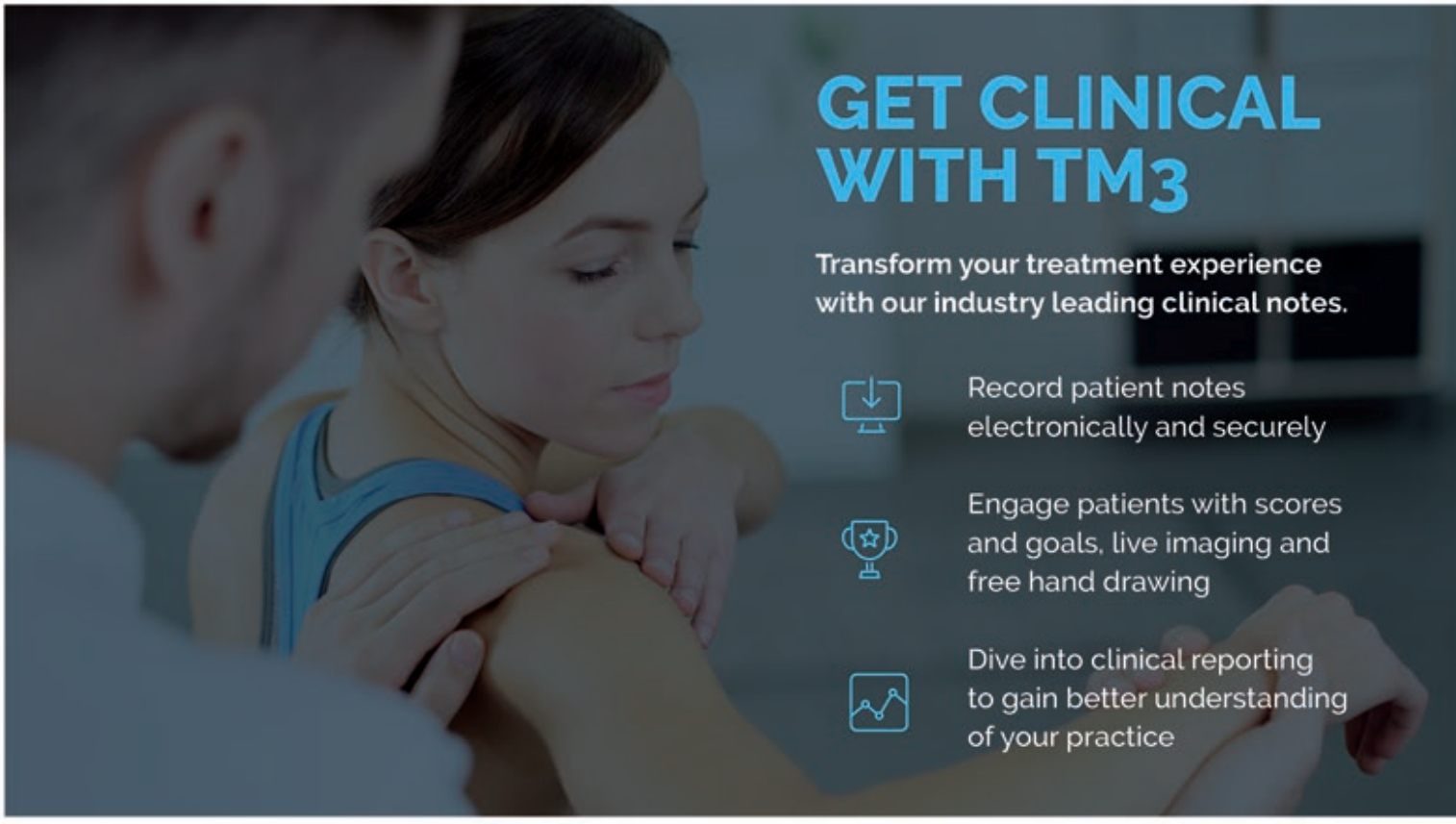

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