

# FLUID MOVEMENT MAY PARTIALLY ACCOUNT FOR THE BEHAVIOUR OF SYMPTOMS ASSOCIATED WITH NOCICEPTION IN DISC INJURY AND DISEASE

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Gifford LS Gifford, L S 1995 Fluid movement may partially account for the behaviour of symptoms associated with nociception in disc injury and disease. In: Shacklock, M O (ed) *Moving in on Pain*. Butterworth-Heinemann, Australia.

## INTRODUCTION

It is always rather satisfying to detect predictability in the behaviour of signs and symptoms because it makes patient problems far simpler to assess and manage. Unfortunately, the difficult nature of pain, combined with the unique nature of individuals and their past make this dream of simplicity unlikely.

However, there are often common features to many disorders, especially in the more acute situations, where nociceptive pain mechanisms are predominant. A classic 'back strain' scenario is where a 30-50 year old is actively enjoying some gardening and feels an odd sensation in the back while bending or digging that is considered to be of little consequence and is passed off without further thought. On rising from a chair some time later, moderate back stiffness is noted and it is relieved by a hot bath. The next morning the full consequences of the mild twinge the day before become apparent. The patient finds it impossible or extremely difficult to get out of bed. If it is possible to get up, the sufferer can not dress easily or stand up straight for anything up to half an hour later. Slowly, spinal movement improves with gentle manoeuvres but rest presages the return of fraught disability.

This scenario has two key features, the delay in onset of symptoms after injury and post inactivity painful stiffening which frees over time with gentle and progressive movement. Painful stiffness is often at its worst first thing in the morning.

Morning stiffness has long been recognised as a key diagnostic feature of the inflammatory arthropathies and its duration an indicator of the severity of the inflammatory process (McKenna & Wright 1990). Indeed, the amelioration of morning stiffness is often used as an indicator of the effectiveness of therapeutic intervention (McKenna & Wright 1990). It would seem reasonable to infer that injury related morning and inactivity stiffness may be an expression of the severity of inflammation similar to that encountered in the inflammatory arthropathies. In practice, a spectrum of 'inflammatory' pain behaviour can be observed. At the worst end of the spectrum are the ongoing arthropathies, all of which cause quite devastating and long lasting morning pain and stiffness when the inflammatory disease process is active. At the other end of the spectrum are the more minor acute musculoskeletal sprains and strains which are often accompanied by a variable degree of painful inactivity stiffness. Here the stiffness ranges from a few moments to 30 minutes or more. As in active inflammatory disease, the duration of morning stiffness is a good marker of recovery or therapeutic progress. Unfortunately, the confusing nature of the complaint of 'morning stiffness' has led to a long line of partially unproductive research focussing on the physical properties of rheumatoid and osteoarthritic joints' through range resistance (see Gifford 1987 for review). The term 'stiffness' as used by the

patient is often very different from the interpretation used by medicine. This rather fundamental issue has been excellently addressed by Rhind et al (1987) in rheumatoid arthritis sufferers. They found that most patients who claim to be 'stiff' in the morning actually use the word to mean a combination of pain and pure physical difficulty in moving. The two issues seem to be inextricably linked. What is clear is that there are many factors operating which include changes in range of movement, fluid accumulation, weakness and perceptual and reflexogenic factors associated with nociceptive and central pain mechanisms.

### CHANGES IN RANGE OF MOVEMENT

Variations in range of movement undoubtedly occur and are generally presumed to relate to physical activity and inactivity (Adams et al 1987). Thus, ranges of movements in normals, such as forward bending and lumbar flexion, have been shown to be less first thing in the morning compared to later in the day (Gifford 1987; Wing et al 1992; Gifford 1994). Adams et al (1987) showed that measures of lumbar spine flexibility diminished after a period of activity if the subjects remained recumbent for a period equivalent to the length of time they had been up and active. In vitro load-testing of collagenous tissues tends to parallel this in vivo flexibility variation behaviour. Thus, preparations of ligament, tendon and muscle show time dependent viscoelastic properties. That is, they elongate slowly over time with an applied force (as in 'activity') and they slowly shorten again when the force is removed (as in 'inactivity') (Butler et al 1978). The implication is that collagenous tissues involved in limiting joint movement are responsible for the observed changes in range due to activity/inactivity.

Emphasis must also be given to the possibility of increased resting muscle tone and/or inhibition of movement as a result of central neural control factors as components of stiffness perception. It would certainly make sense for the central nervous system to protect musculoskeletal tissues when they are first moved after prolonged inactivity. Passive muscle factors may be important too. For instance, Gossman et al (1982) reported that length associated changes can take place from within a few hours of a muscle being immobilised. Typically, the number of sarcomeres decreases if the muscle remains in a shortened position.

Thus, inactivity leads to a loss of range of movement and gains in range are made with activity. What is of interest is that 76% of the 25 normal subjects measured two hourly over a 24 hour period by Gifford (1987), recorded a feeling of stiffness that coincided with the times of least range of movement (2400 hours to 1000 hours). In response to a postal questionnaire Badley and Tennant (1992) noted that 65% of over 50 year olds reported feelings of morning stiffness. As Bywaters (1982) says, 'Man starts as a jelly and ends as a stiff'. Morning stiffness appears to be a part of many peoples lives and may relate to simple loss of tissue compliance in association with protective central nervous system movement inhibitory mechanisms. It is tempting to speculate that in injury or disease these normal perceptions of stiffness become amplified due to sustained injury precipitated sensitivity changes in peripheral and central neural processing mechanisms. However, superimposed on this may be increased forces on sensitised afferent nerve fibre terminals due to abnormal accumulation of inflammatory fluids.

### FLUID MOVEMENT AND ACCUMULATION

The disc provides a unique example of a highly collagenous and highly fluid and movement dependent tissue whose behaviour in response to mechanical stress is worthy of our attention. The disc is reported to be the largest avascular structure

in the human body (Maroudas et al 1975) and arguably relies on continuous changes in pressure for maintenance of its health.

The fluid dependent nature of the disc is beautifully illustrated with reference to diurnal changes in body height. Apparently, we lose height through the day and regain it while recumbent overnight (see Broberg 1993 and Gifford 1994 for overviews). This is due to changes in disc height as a result of fluid exchange with the environment and viscoelastic deformations of the annulus fibres (Adams & Hutton 1983; Broberg 1993; LeBlanc et al 1994). Fluid expression is a result of not only gravitational forces, but also mechanical forces from muscular action. The degree of height variation gets less with ageing which probably reflects the changing biochemical, mechanical and hydration attributes of the ageing disc (Adams & Hutton 1983; Twomey & Taylor 1994). According to the measures of DePuky (1936) on 1,200 prison inmates, 70-80 year olds vary by about 0.5% of their body height but the general average is 1% (approximately 19 mm). He noted that his young son varied by 2%. Thornton et al (1974) recorded 3% variations in body height for astronauts in a gravitation-free environment.

Taking this overnight discal fluid accumulation into consideration one can easily see that an uninjured disc will represent a highly turgid and hence mechanically stiff structure first thing in the morning. This could account for the observed losses of lumbar flexibility at this time. Further, there appears to be a modest parallel between loss of height after rising and increasing flexibility of the lumbar spine (Gifford 1987, 1994).

### THE ACUTELY INJURED DISC AND SYMPTOM BEHAVIOUR

Delay in onset of symptoms after injury has to be viewed carefully since the central nervous system is capable of powerfully preventing the admittance of noxious impulse barrages arriving from the periphery (Blank 1994). In viewing the delay of onset of symptoms from a purely 'peripheral' stance, the disc highlights several possibilities. The disc has a relatively poor nerve supply. In the lumbar spine it is only innervated in its outer one third (Bogduk et al 1981; Groen et al 1990), thus, injury to deeper non-innervated regions may not register symptoms immediately.

Injury to well vascularised tissues normally initiates a multi-system cascade of cellular, circulatory and chemical events that lead to the formation of an inflammatory 'soup' (Levine & Taiwo 1994; Meyer et al 1994) at the injury site. The excitatory chemical constituents of the 'soup', the algogens, have three main effects on the local nerve population. Firstly, they cause most afferents, including nociceptor afferents, to either increase their normal resting impulse discharge rate or to start to spontaneously fire (Hanesch et al 1992), which probably provides the basis of the background aching/awareness we so often encounter with acute pain. Secondly, they cause nociceptors to dynamically change their sensitivity so that they begin to fire to non-noxious stimuli, for instance, the sharper pain encountered with movements and palpation. Thirdly, they arouse 'sleeping' or 'silent' afferents, that in the uninjured state never transmit a single impulse — even with extremely noxious stimuli (McMahon & Koltzenburg 1990, 1994; Schmidt et al 1994). What is of importance is that, over several hours, the afferent impulse barrage steadily increases and may account for the increasing and delayed perception of pain and injury.

It is clear that in the normal disc, there is no intrinsic circulation and that its substance, relative to most tissues, has a low cellular population and it mostly consists of fibrous and fibro-cartilage elements. Thus, classic inflammation per

se cannot occur and the disc's response to injury must be a special and relatively slow process. It is conceivable that in response to injury a unique chemical cascade and 'soup' occurs that slowly perfuses from the deeper injured site to the peripherally innervated layers, or simply, that injury to the non-innervated areas of disc are never painful. It may be that the disc nociceptive mechanisms, like that of the viscera, are relatively sluggish (McMahon & Koltzenburg 1994) so that even if injury does take place in the innervated outer annulus, there may be little immediate noxious afferent activity.

The release of fluid, or plasma extravasation, into injured or diseased tissues is one of the key components of the inflammatory response. As we have seen, the views presented so far suggest that fluid accumulation and movement may be important to range of motion variability, as well as to some consistent changes in the behaviour of symptoms. Engstrom-Laurent and Hallgren (1987) certainly think so. These investigators highlight the unique properties of the glycosaminoglycan hyaluronic acid (HA). Briefly, HA is an extracellular constituent of all connective tissues found in particularly high concentrations in loose connective tissues such as synovial membrane, but it is also found in muscle (Piehl-Aulin et al 1991), cartilage and disc (Urban & Maroudas 1980). HA is an important chemical in the homeostasis of tissue water levels as it not only attracts water but also resists water flow (Laurent 1987). The higher the concentrations of HA the more water is accumulated and the more water flow is resisted. Tissues therefore become more rigid and hence mechanically hinder movements of muscles and joints (Engstrom-Laurent & Hallgren 1987). These workers have shown that during activity HA is physically forced out of the tissues and into the lymphatics before being passed into the general circulation where its plasma concentrations can easily be measured. Thus, in the early morning prior to rising, the plasma levels are low, reflecting high interstitial tissue concentrations. After being up and active for one hour the plasma levels roughly double, reflecting a drop in HA tissue concentrations (Lindqvist et al 1988). This apparent mechanically initiated exodus of tissue HA, and hence tissue water content, appears to reflect the observed flexibility improvements noted earlier. The close association between HA levels, fluid accumulation, physical movement and the changes in flexibility outlined above, cannot be overlooked.

Production of HA appears to be stimulated during inflammation by the kinin, interleukin-1, and levels increase when inflamed tissues are at rest. Lindqvist et al (1988) have shown that in the inflamed rheumatoid arthritic state the concentrations of plasma HA are in the region of eight times that of normal. Further, after being up and active for one hour, like the normal subjects discussed above, there is a marked plasma increase which reflects the ebbing morning stiffness suffered by the patients.

The dynamic behaviour of HA biology is of special interest in the context presented here. The substance is a part of the inflammatory scenario, has been shown to be forced out of joint tissue during movement, is responsible for viscosity changes and fluid concentrations within collagenous tissues and must be considered as one of the central characters in the chronicles of morning stiffness. However, it is always unwise to pin hopes on one issue, let alone one chemical. Osmotic concentrations must change markedly during inflammation and injury due to the accumulation of the diverse 'soup' of chemicals at the injury site. All must play a part in bringing about the local fluid increases and, hence pressure changes.

As far as peripheral mechanisms of the perception of painful stiffness are concerned, the focus appears to fall on changes in tissue pressure. Thus, increased pressure within a disc following disc damage and subsequent swelling can indirectly cause discogenic pain as the increased pressure stretches the innervated annular envelope (Bogduk & Twomey 1991). Clinically, this is borne out by the observation that acute discogenic pain is often significantly worse when going from lying to sitting first thing in the morning and that this feature can be alleviated if the subject spends several minutes doing gentle lumbar mobilising exercises prior to rising. This act 'prepares' the subject; firstly in respect of the tissues by promoting some fluid exodus from the disc and hence reducing intradiscal pressure/dispersing algogens (see below) and secondly, in respect of central inhibitory mechanisms by, for instance, becoming mentally attuned to move cautiously. A further common observation that adds weight to this paradigm of fluid movement is that pain in these patients, with presumably intact discs, is made significantly worse by traction of only modest force and for a short time. Traction may powerfully accelerate the overnight fluid gain (Warden & Humphry 1964; Twomey 1985) in the disc. The most unnerving feature of traction for these patients is that they find it quite comfortable while the distracting force is applied, yet severe symptoms often occur as the traction is released (especially if a high poundage was used) or later, when they rise from the couch. This embarrassing situation can be avoided if the patient spends a few minutes performing simple crook lying lumbar rotation and pelvic rock exercises before getting up. In theory, what all this is perhaps telling us is that the hyperalgesic disc is hydrostatically intact. If this is so, this simple reaction to traction, when combined with historical and general symptom behaviour knowledge, may be used as a useful diagnostic test for disc integrity. This hypothesis, that the disc must be intact for apparent fluid dependent behaviour to occur, has some support from the work of Porter and Trailescu (1990). Their article is discussed at length in Gifford (1994).

If injury is deep and the annulus nociceptor population has been unaffected by sensitising algogenic agents, it is easy to see how rapidly symptoms may improve with gentle active or passive movements that enhance the exit of fluid from the disc. Return of fluid pressure during rest may explain the return of symptoms. However, it is highly likely that injury induced sensitivity adjustments in the peripheral afferent fibre population does occur, as most patients with the features of pure discogenic pain (if one really enquires) have ongoing background symptoms to a greater or lesser degree. It may be that the 'washing out' of algogenic chemicals as a result of movement and their accumulation with rest are further important components to the observed fluctuation of symptoms. It is certainly evident, at this level of thinking, that mechanics or 'movement' can be used to change physiological events that affect symptoms.

### **SOME ISSUES IN CHRONIC DISC DISORDER**

Symptoms that continue long after known normal healing times are often designated as chronic pain syndromes and much of the focus of attention in these states is now towards appreciating abnormalities in the function of central nervous system control mechanisms (Melzack & Wall 1988). This refreshing stance is probably set to explain many of the more stubborn pain syndromes that physiotherapy has been persistently trying to fit into a peripheral tissue model for far too long. The message is that there may be very little peripheral tissue involvement in many pain states, even though we designate a myriad of 'positive' results to implicate tissues that respond painfully to mechanical testing. An essay by

Professor Wall (1993), although confined to discussing fibromyalgia, usefully discusses some of these issues at length. What is clear is that we must always be on guard regarding assumptions about origins of pain and this should be taken into account when considering the discussion which follows.

If peripheral tissue pathobiological mechanisms do continue to maintain pain syndromes for extended periods, the implication is that some abnormal endogenous process is at work. It is relatively easy to view long term spinal pain with significant daily morning pain and stiffness that is diagnostically labelled 'ankylosing spondylitis' as having a significant peripheral inflammatory/nociceptive component maintaining the pain state. Some patients have far more benign chronic symptoms of similar character that are not 'disease' designated yet the symptoms often impose on lifestyle. The following 48 year old male patient with a five year history is not infrequently encountered. He complains of chronic morning stiffness of roughly two hours duration, he has great difficulty bending to dress and wash first thing in the morning yet with movement and simple exercise he gradually frees. Once the stiffness has subsided he is pain-free and able to work energetically at his job which involves strenuous physical activity through much of the day. In this situation, non-steroidal anti-inflammatory medication used to significantly help the stiffness but it no longer has any major effect. The sufferer is modestly helped for short periods by manual therapy and made significantly stiffer by traction. Rest, as when on holiday, used to help but more recently has little effect. Interestingly, this patient's lumbar stiffness completely cleared up on his most recent holiday when, after a long plane flight, he started to develop nasty posterior thigh pain. Although it prevented him from fully flexing due to pain, he no longer had prolonged morning stiffness. The 'sciatica' slowly cleared over a period of about 8-10 weeks and left him completely pain and stiffness free. It is very tempting to speculate that his original stiffness was being maintained by some mild internal disc 'inflammatory' mechanism which may have imposed itself via the fluid mechanisms discussed. Later, breach of the outer annulus, perhaps via a radial fissure extension, could account for the loss of the fluid mechanism and loss of the stiffness. Further, the sciatica may have been precipitated by modest leakage of antigenic disc fluid (McCarron et al 1987) causing adjacent root sleeve inflammation.

We should also acknowledge that prolonged centrally maintained sensitivity of appropriate dorsal horn cells and synapses could be responsible for ongoing stiffness due to the now pathological perception of normal fluid pressure changes and normal stiffness. It somehow seems appropriate that a previously damaged tissue that never really regains its original structural integrity should always be protected in some way against dangerous mechanical forces. Maintained central sensitivity may be a desirable homeostatic mechanism in some degenerate and post-injury scenarios.

## FINAL THOUGHTS

The tissue environment is never stable. Influences include not only mechanical effects via activity and inactivity, but also the natural fluctuations of endogenous control systems. For instance, diurnal variations in the immune system's control of inflammation (Harkness et al 1982), naturally produced inflammatory suppressive agents such as cortisol and endorphins (Dent et al 1981; Harkness et al 1982; Petraglia et al 1983) and diurnal temperature variation have been shown to relate closely to joint flexibility (Wright & Johns 1960). Tissue flexibility and our perception of stiffness and pain must consider many of these covert influences.

It is the duty of physiotherapists to attempt to make sense of pain behaviour and observed dysfunction based on understanding pathobiology, including the nociceptive concepts presented in this paper. This is hopefully a reminder that there is a huge health professional knowledge vacuum (Melzack & Wall 1988) which still has not come anywhere near being filled. Furthermore, we must report our observations of patients to the scientific and medical community (using the language of science) for the reason that physiotherapists, by virtue of their close contact with patients, have good opportunities to observe pain behaviour in great detail. It is hoped that physiotherapists may in the future contribute to the understanding of the mechanisms that produce pain and offer decision making options in a multidisciplinary pain management setting.

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