Extracorporeal shockwave therapy (ESWT) an option for chronic tendinopathy management: a clinical perspective

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Abstract.

Aim To highlight extracorporeal shock wave therapy (ESWT) as an option available to clinicians when faced with chronic unresponsive tendinopaties, and to clarify some of the conflicts that arise in medical literature as to its efficacy, based on evidence and clinical experience.

Background Overuse tendinopathies are a commonly seen condition in sports and occupational medicine that presents a challenge to clinicians and therapists alike. The exact etiology and the nature of tendionpathic disability and pain have not been fully elucidated. However, the disruption of the natural healing process with the associated pathological neurobiochemical activities leading to tendon degeneration, are seen to be some of the causative factors involved.

Treatment options Despite the abundance of therapeutic options available to treat tendinopaties, there seems to be several inconsistencies, irregularities and the lack of evidence based guidelines for the management and treatment of this condition.

Conclusion When used by an experienced operator with the appropriate energy flux density levels and treatment protocols ESWT proves to be an effective treatment modality. ESWT should not be used as a primary treatment option for any condition, but may be considered when at least two or three other conservative treatment options have failed to resolve tendinopathic issues.

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Introduction

The tendon is a viscoelastic structure with plastic properties and is an integral part of the musculotendinous unit facilitating joint motions via the transmission of contractile forces from muscle to rigid bone levers by generally concentrating their pull and direction of a muscle at a small region (Benjamin et al 1973; 1995; Jarvinen et al 2004; Constantinou et al 2005; O’Brien, 2005). The strength of a tendon is dependent on the size, collagen fiber orientation, thickness and internal fibrillar organization (Oxlund, 1986; O’Brien, 2005). Tendons are subject to high tensile and compressive loads acting on them as they aid with postural maintenance, energy storage, and shock attenuation (O’Brien, 2005). Hence tendons are at an increased risk of injury and repetitive stress syndromes especially at the myotendinous junctions and osteotendinous insertion regions. Tendon injuries may occur by either acute trauma or by repetitive loading (tendinopathy), where the management of the latter is the focus of this article. The commonly seen overuse tendinopathies involve the Achilles tendon, rotator cuff, medial and lateral elbow epicondyles, and the patellar tendon; where patients may suffer considerable disability ranging from several months up to years despite what is deemed to be appropriate management (Almekinders & Almekinders, 1994; Sharma & Maffulli, 2005). When considering treatment options for overuse tendinopathies, a thorough appreciation of the properties of a healthy tendon, the functional interplay and mechanical properties of the musculotendon complex, and emotional status of the patient is necessary. Despite the abundance of therapeutic options available to treat tendinopathies, there seem to be inconsistencies, irregularities and the lack of evidence based guidelines for its treatment (Frohm, 2006; Crossley et al 2007; Maffulli & Longo, 2008).

Figure 1. Tendon structure and tight packing of collagen fibers in healthy tendons

Tendon structure and physiology.

Some tendons possess a true synovial sheath while others have their epitendon surrounded by a paratendon which aid in the lubrication, friction prevention and protection of their structures. There are marked variations in a tendons form and structure: they may be rounded cords, flattened ribbons, or strap like bands, and when in a healthy state, present as brilliant white with a fibroelastic
texture. In contrast, a tendinopathic tendon presents as grey or a yellow-brown amorphous structure (Khan et al 1999; Fredberg & Stensaard-Pederson, 2008). Tendons are primarily made up of 55 – 70% water and a substantial portion of this is associated with proteoglycans at the extracellular matrix (Kjear, 2004). The basic extracellular elements of a tendon are: cells, collagen and ground substances. The extracellular matrix (ECM) of tendons comprise of approximately 90 – 95% elements of tenoblasts and tenocytes that lie between the collagen fibers along the long axis of the tendon, and the remainder of the 5 – 10% of the cellular elements comprise of chondrocytes, vascular and synovial cells (O’Brien, 1997; Kannus, 2000; Kannus et al 2000; Kjear, 2004; Sharma, 2005). Tenocytes are responsible for collagen synthesis which makes up 60 – 80% of the dry mass of human tendons, with elastin making up 2% of this matter, which is embedded in the proteoglycan-water matrix (O’Brien, 1997; Kannus, 2000; Kjear, 2004; Sharma, 2005). **Blood Supply.** Blood supply of tendons arrive from the intrinsic system at the myotendinous and osteotendinous junctions, and extrinsically via either the synovial sheath or from the paratendon. The ratio of blood supply from the intrinsic and extrinsic systems vary from tendon to tendon (Naito & Ogata, 1983; Hooper et al 1984; Carr & Norris, 1989). In regions where tendons are enveloped by friction reducing sheaths, branches from major vessels penetrate the mesotendon reaching the visceral synovial sheath and form a plexus supplying the superficial portion of the tendon while some vessels penetrate the epitendon (Jozsa & Kannus, 1997). In the absence of a synovial sheath the paratendon provides the extrinsic component of the vascular supply. The vascularity of tendons are compromised at junction regions and at sites of friction, torsion and compression, where generally, vascularity is seen to decrease with mechanical loading and age (Sharma, 2005). **Metabolism.** Being predominantly an extracellular tissue, tendons have a lower blood supply and metabolic requirement compared to most other tissues (ie muscles), where oxygen consumption in tendons have been observed as being over seven times lower compared to skeletal muscles (Vailas et al 1978; Jozsa, 1997; Sharma, 2005). This low metabolic rate and the well developed anaerobic energy generation allow for the prolonged and increased tension states to continue in tendons without the risk of ischemia and subsequent necrosis. However this very characteristic of tendons is a primary factor associated with their slow recovery rates and degeneration when fatigued or injured (Williams 1986; Kannus & Jozsa, 1991; Sharma, 2005). Fatigued tendon recovery times are slower when compared to muscles ranging between 6 – 14 weeks (Valias et al 1978; Sharma, 2005).
**Innervation.** Tendon innervation is supplied by the sensory nerves from the overlying superficial nerves and / or from the close deep nerves, which are largely afferent (Jozsa & Kannus, 1997; O’Brien, 2005). There are four types of receptors inside tendons: Ruffini corpuscles, Vater-Pacini corpuscles, Golgi tendon organs, and free nerve endings (Jozsa & Kannus, 1997; O’Brien, 1997; O’Brien, 2005; Sharma, 2005). These receptors respectively detect pressure, stretch reflex, sensory perception, along with reactive motion changes to acceleration and deceleration, tension and pain (Jozsa & Kannus, 1997; O’Brien, 2005).

**Mechanics.** Tendons are viscoelastic tissue that exhibit high mechanical strength and flexibility, with a unique ability to transmit force and absorb shock to limit muscle damage while performing their role in human locomotion and function (Oxlund, 1986; O’Brien, 1992; Sharma, 2005). Tendon thickness and collagen content contribute to the tensile strength of a tendon but vary in size, wavelength, and crimp angle from tendon to tendon, with the thickest fibers found in the Achilles tendon, and the thinnest fibers found in the extensor policies tendon (Jarvinen et al 2004). During the lengthening phase of a tendon a degree of extensibility allows for elastic energy to be temporarily stored enabling elastic recoil (Sadwick, 1990; Alexander, 2002). Tendons may be broadly divided into two groups; those that undergo low stress and those that undergo high stress. The highest stress forces acting on tendons occur during strenuous eccentric muscle contraction such as, jumping, weight lifting, and landing from a jump (Ishikawa et al. 2005). Both in-vivo and in-virto experiments conducted by Barfred 2001 and Jarvinen et al 2004; demonstrated that a tendon may be at its highest risk of rupture when forces are rapidly and obliquely applied during eccentric muscle contractions (Sharma, 2005). **Crimp.** The crimp represents the resting unloaded state of a tendon (also a feature in ligaments). The crimp (Figure 2) provides a buffer where tendon fiber elongation may occur without damage while simultaneously acting as shock-absorbers (Fratzl et al 1998; Jarvinen et al 2004; Freed & Doehring.

![Figure 2. Demonstrating the linear properties of a tendon vs stress force.](image-url)
Tendons are capable of responding to repeated loads, and when these loads exceed the physiological threshold of the tendon, they react by either the inflammation of their sheath or the degeneration of their main body or both (Jarvinen, 2004; Sharma, 2005).

**Tendon injury and tendinopathy.**

Tendon injury arise from a combination of intrinsic and extrinsic factors; acute tendon injuries may be predominantly caused by extrinsic factors, whereas in overuse syndromes as in the case of tendinopathy it may be caused by multifactorial combinations of both intrinsic and extrinsic factors. An example of an intrinsic factor for tendinopathies are: poor biomechanics such as limb malalignments and hyperpronation that may cause increased traction loads acting on the foot and ankle that may increase the incidence of Achilles, flexor hallucis longus and posterior tibialis tendinopathies (Clement et al 1984; Nigg, 2001; Oster, 2009; Simpson et al 2009). The most commonly accepted pathological factor associated with tendinopathies is the failed healing response due to repetitive overload arising from sports and activities of daily living, that result in disability both in sports and at the workplace (Maffulli et al, 2003; Kjaer, 2004; Ames et al 2008; Maffulli et al, 2010). Inflammation of their sheath, degeneration of the tendon body or ethesis can occur singularly or in combination as a general response to repetitive loads beyond the physiological threshold (Benazzo & Maffulli, 2000; Maffulli et al 2003; Sharma, 2005). The exact etiology of tendinopathies has not been fully elucidated and different stresses may induce varying responses. There are multifactorial theories such as tensile overload, tenocyte related collagen synthesis disruption, tendon load induced ischemia, neural sprouting, and histological adaptive compressive responses seen as some of the causative factors that give rise to activity disruption and disability due to tendinopathies (Cook, 2000; Purdam, 2003; Warden, 2003; Hamilton, 2004; Cook et al 2005; Lian, 2005; Frohm, 2006; Crossley et al 2007; Maffulli, 2008; Maffulli et al 2010). The most commonly accepted cause for this condition however is seen to be an overuse syndrome in combination with intrinsic and extrinsic factors leading to what may be seen as a progressive interference or the failing of the innate healing response (Cook, 2000; Witvrouw et al 2001; Richards et al 2002; Purdam, 2003; Warden, 2003; Cook et al 2005; Crossley et al 2007; Frohm, 2006; Hamilton, 2004; Lian, 2005 & 2006; Woodley et al 2007; Maffulli, 2008; Maffulli et al 2010).
Effects of aging and abnormal biomechanics in tendinopathies.

Tendinopathies are commonly seen in both the primary care setting as well as in sports clinics as aging, occupation, activities of daily living, and sports contribute to this condition (Sharma, 2005; Rees et al 2009; Simpson, 2009; Maffulli, et al 2010). Interference of the collagen synthesis and collagen degeneration due to age is a common underlying factor for tendinopathies, and has been detected as early as the third decade of life. The mechanical strength of individual collagen molecules are dependent upon the extracellular formation of the triple helix molecule that assemble into a collagen fibril, and as 60 – 80% of the dry weight of a tendon in composed of collagen (mainly type 1), the physiological and mechanical attributes of the collagen composition ultimately dictates tendon strength (Josza & Kannus, 1997; O’Brien, 1997; Jarvinen et al 2004).

The natural aging process and the associated biochemical and cellular changes are seen to disrupt tenocyte activity which impacts collagen integrity and regenerative properties (Kannus, 2005). Age simultaneously impacts both the micro and macroscopic aspects of tendons, and result in age related physiological changes making the aged-tendon weaker to its younger counterpart resulting in an increased risk of overuse syndromes especially if the tendon already suffers from pathological degenerative changes (Langberg et al 2001; Kannus, 2005; Sharma, 2005; Kettunen et al 2006). Biomechanical and musculature induced anatomical variances such as Q-angles, limb length discrepancies, planus and cavus foot orientation, muscle weakness and imbalances, impingement syndromes, and the associated ambulatory pathologies such as lateral heel strike with compensatory hyper-pronation, all increase the force and load moments occurring in tendons, and are seen as possible contributory factors for tendinopathies (Kannus, 1997; Nigg, 2001; Williams et al 2001; Wityrow et al 2001; Schmidt, 2002; Mahieu et al 2006).
**Exercise and overuse tendinopathies**

Overuse tendinopathies caused by continuous repetitive loads resulting from sports, occupation and activities of daily living may be attributed to the inability for the tendon to cope with the consistent loads and forces acting on its structure and ultimately succumbs to these stresses triggering the tendinopathic process of an impaired or failed healing response, and the eventual degeneration of the tendon (Maffulli et al 2003; Kannus, 2005; Sharma, 2005; Alfredson, 2006; Rees et al 2009; Simpson, 2009; Maffulli et al 2010). Exercise under normal conditions enhance tendon strength but when the regenerative threshold is crossed, damage may occur in varying intensity. If adequate rest along with ambient nutritional and blood flow is afforded to the tendon, the restorative process may prevail and result in complete tissue restoration and injury resolution. However inadequate recovery time and an untimely return to strenuous activity may lead to what is seen as the first phase of tendon damage, where micro-damage occurs within the tendon. If unarrested a cascade of biochemical degenerative events occur. The sequence from first stage to second stage often occurs insidiously and is highly variable due to the complexity of individual factors acting on the tendon (Kjear, 2004; Rees et al 2009).

**Clinical presentations.**

The clinical presentation of tendinopathies are often insidious with impaired joint range of motion which may worsen over time especially with increased activity. There may simultaneously be swelling or muscle atrophy, and tenderness along the course of the tendon (Cook, 2000; Pfirrmann et al 2008). In the early stages of tendinopathy, stiffness and decreases in joint range of motion is usually experienced mainly in the morning which is relieved after a period of warming up. Pain at this stage may be described as sharp during activity and dull during rest. As the condition progresses pain may be experienced even during periods of rest and inactivity and often interrupts sporting activities and in some cases cause severe limitations and disability presenting a clinical challenge (Simpson, 2009; Maffulli, 2010). Chronic tendionpathies may often have more than just a physiological impact on and individual, as persistent pain and disability are known to impact an individual’s self worth, resilience and may increase emotional stress (Craig, 2010). This is an important point to consider as it may ultimately impact upon treatment outcomes (Craig, 2010), but may be often overlooked when clinically assessing and treating chronic conditions such as tendinopathies.
**Histology**

Haphazard tenocyte proliferation and cellular composition, collagen degeneration and irregular fiber orientation and structure, increase in neovascularisation, hypoxic mucoid degeneration, fibrinoid necrosis, microtears, calcific deposits, granulation tissue and a combination of entities are considered as expressions of a failed healing response associated with tendinopathies (Kannus & Jozsa, 1991; Khan, 1996; Cook et al 1997; Paavola et al 2002; Cetti et al 2003; Klear, 2004; Maffulli et al 2010). Despite the prolific use of the historic term tendinitis when describing chronic overuse tendon injuries, histopathologic findings indicate that chronic tendinopathy is devoid of an overt prostaglandin mediated inflammatory process (Alfredson et al 2001, Magra, 2006; Maffulli et al 2010), although inflammation may be seen in the initial stages, it is not seen as a propagator of the disease process (Alfredson et al 2001; Sharma, 2005; Magra, 2006; Rees, 2009; Maffulli, et al 2010). This suggests the term tendinitis may be a misnomer and some of the traditional treatment modalities aimed at inflammatory control such as non-steroidal anti-inflammatory drugs (NSAID’s) and cortisone injections may not be the most suitable options when treating chronic tendinopathies (Khan et al 2002; Wilson 2005; Magra, 2006; Andres & Murrell, 2008; Maffulli et al 2010). However consideration must be given to the fact that although prostaglandin mediated inflammation may not be the cause of disease progression, neurogenic inflammation due to an increased expressions of neurotransmitters have the potential in initiate peritendinitis, which in turn may lead to degenerative changes in the tendon (Hart et al 1998; Sullo et al 2001; Fredberg & Stengaard-Pederson, 2008) not totally eliminating the role of inflammation in tendinopathies emphasizing the complexity of this condition.

**The psychophysiology of pain in chronic tendinopathies.**

With the exception of those with a congenial insensitivity to pain, the experience of pain is something that humans are familiar with (Ebrahimí-Nejad, 2007; Quniter, 2008), yet this familiarity with pain has not allowed us to fully grasp the very nature and causative factors of pain in chronic states (Craig, 2010). The psychophysiology of the pain experience resulting from injury or tissue damage encompasses a combination of unpleasant sensory and emotional qualities that is highly individualistic (Malzack, 2005; Ebrahimí-Nejad, 2007). In the acute phase, pain serves as a primary adaptive function as it signals that the body has undergone an insult or injury, and triggers the avoidance of the causative agent or situation to prevent further
injury (Ebrahimi-Nejad, 2007; Craig, 2010). However in the chronic phase of injury, pain becomes maladaptive, and has ceased to have a useful function and now, in addition to the physical injury; emotions of fear, frustration, anger, and even depression may be experienced by sufferers (Ebrahimi-Nejad, 2007; Craig, 2010), making the management of chronic injuries such as tendinopathies a multifaceted challenge for clinicians. Traditionally it was thought that chronic inflammation was the cause of pain in chronic overuse tendinopathies however histological findings indicate the absence or the lack of inflammatory mediated markers in these conditions (Kannus & Jozsa, 1991; Astrom & Rausing, 1995; Alfredson et al 1999; Alfredson & Lorentson, 2002; Kjear, 2004; Fredberg & Stengaard-Pederson, 2008) and as such pain in chronic tendinopathies may originate from a combination of neurobiochemical and mechanical factors (Khan et al 1999; Khan et al 2002; Sharma, 2005). Pain in tendinopathy may be multifactorial and is seen to be caused by an increased presence of neurotransmitter substances as studies on patients with chronic Achilles tendinopathies show an increase in levels of lactate and glutamate compared to control (Alfredson, 1999; Alfredson et al 2002; Sharma, 2005; Alfredson, 2006). Neuromodulators and transmitters such as substance P and calcitonin gene related peptide (CGRP) are commonly found in unmyelinated sensory fibers, and are seen in increased levels in tendinopathic Achilles and patellar tendons, and elbow epicondyles, along with the increased presence of neural in-growth (Ackermann et al 2002; Ljung, 2004; Schubert et al 2005; Sharma, 2005; Alfredson, 2006; Lian et al 2006). As sensory nerves transmit pain to the spinal cord, the increased levels of these excitatory and nociceptive substances may play a role in the pain modulation experienced by patients with chronic tendinopathies (Alfredson et al 2001; Ljung, 2004; Schubert et al 2005). When treating and rehabilitating chronic injury, attention to the emotional state of the patient is of clinical significance when one recognizes that emotions although subjective and intangible is what motivates human behavior and interactions, which includes; movement, or the avoidance of it (Foa & Kozak, 1986; Heilmann, 1997; Leonard, 1998). Appreciation of the intricate and complex association of the spinal cord and brain stem will further provide the premise that pain, disability, emotions, and the individual reactions and responses are closely interrelated. For example; a light touch in a dark room will elicit a completely different response to the same stimulus in a bright ballroom, hence a change in the reactionary movement pattern based on emotion given the difference in settings. The reticular formation which receives inputs from a wide area of the cerebral cortex including inputs from
the; spinothalamic tract (pain), hypothalamus (emotion, motivation, and endocrine function), limbic system (emotion and endocrine function), is simultaneously responsible for directing motor output (movement). It has been noted that motivated excitement may enhance motor performance, while fear and anxiety can interrupt movement patterns, impede performance, and even destroy athletic careers (Leonard, 1998; Powell, 2004; Alfermann et al 2005). The motor-emotional triggering mechanisms of the reticular formation may be one of the triggering mechanisms for conditions known as fear avoidance and cognitive inattention, the two opposite extremes of injury. Where in one individual, the fear and anxiety of injury triggers emotions of panic and movement avoidance due to the imbedding of a neurosignature, and in another, beliefs such as; no guts no glory or toughen-up, may cause the individual to ignore the cognitive episode and continue on despite the risk of injury aggravation (Bandura, 1977; Nelson, 2009). This intimate relationship between disability, pain, motor activity and emotion is not an imaginary concept but one which is very real and may be experienced by the general public as well as high performance athletes. Hence a biopsychosocial approach when treating chronic conditions such as tendinopathies may prove effective when assessing patients, providing education and the development of their rehabilitation programmes.

Healing process in tendons.

There are three phases involved in the healing process in tendons: the initial inflammation phase (at injury), the repair phase, and the remodeling phase. During these phases phagocytosis of necrotic material occur, and chemotactic and vasoactive factors are released initiating angiogenesis and tenocyte proliferation among others (Murphy et al, 1994; Oakes, 2003). Nitric oxide a free radical with several biolocigal functions including bactericidal properties is released at the repair phase inducing angiogenesis and vasodialative action (Ziche et al 1994; Murrell et al 1997; Lin et al 2001; Fox & Murrell, 2007). Tendon healing may occur either by epitenon and endotenon tenocyte proliferation, known as the intrinsic healing process, or extrinsically by cell invasion from the surrounding sheath and synovium; at these times a cascade of biochemical factors occur and a phasic increase in collagen type I and III syntheses is seen (Murphy et al 1994; Oakes, 2003; Sharma, 2005). Due to the slow metabolic nature of tendons, and the turnover time of the tenocyte-collagen process, healing of the tendon complex after fatigue and injury may take a longer period before the tendon may be able to cope with excessive stresses.
Tenocyte dysfunction and apoptosis may occur due to excessive strains placed on tendons due to an inappropriate return to strenuous activity. It may be prudent to recommend an adequate timeframe of rest and structured rehabilitation before an injured tendon is expected to withstand load conditions prior to its injury.

**Current treatment options for tendinopathies.**

Current conservative and non-surgical treatment options available for the management and treatment of tendinopathy include: rest, ice, massage therapy, eccentric exercise, NSAID’s, cortisone injection, ultrasound therapy, LIPUS, acupuncture, electrotherapy, taping, sclerosing injections, blood injection, glyceryl trinitrate patches, and (ESWT) extracorporeal shockwave therapy (Warden, 2003; Wilson, 2005; Visnes et al 2005; Frohm, 2006; Warden et al 2006; Crossley et al 2007; Murrell, 2007; Visnes & Bahr, 2007; Wang et al 2007; Maffulli, 2008; Paoloni et al 2009; Maffulli et al 2010). Despite the abundance of therapeutic options available to treat tendinopaties, the inconsistencies, irregularities and the lack of evidence based guidelines for treating this condition (Frohm, 2006; Crossley et al 2007; Maffulli & Longo, 2008;) warrants the exploration of treatment modalities and methods that may help address these issues, especially for chronic conditions.

**Extracorporeal shockwave therapy (ESWT).**

Shockwaves were investigated for biomedical application which eventuated into the development of the kidney stone lithotripter, the world’s first minimally invasive surgery and to date, the gold standard for the eradication of kidney stones (Herr, 2008). Investigations into the side effects of medical shockwaves revealed that patients who had undergone lithotripsy reported reduced back pain, increased bone density and tissue growth in the localized treatment area (Ogden, 2004; Wang, 2007; Endes et al 2008; Caccihio, 2009).

![Figure 4. Physical properties of medical shockwaves: Positive pressure amplitude is followed by a tensile wave (pressure vs time).](image-url)
Since the 90’s extracorporeal shock wave therapy (ESWT) have been applied in orthopaedics, trauma and musculoskeletal medicine with successful results and its application has expanded into; sports medicine, pain management (CRPS1), arthropathy, chronic wound and ulcer management, limb dystonia, neurologic disorders, cosmetic medicine, and cardiology (Schaden, 2001; Ogden, 2002; Weil et al, 2002; Ogden et al., 2004; Furia, 2006; Amelio & Manganotti, 2010; Angehrn, 2008; Cacchio, et al, 2009; Furia, 2008; Moretti et al 2008; Moretti et al, 2009; Notarnicola et al., 2010; Schaden et al, 2007; Taki et al., 2007; Wang, 2007; Trompetto et al., 2009; Vasyuk, et al, 2010; Zelle, 2010). ESW are acoustic waves that transmit energy from a generator source, presented as a single positive peak pressure pulse with a rapid peak rise time of up to 100MPa, followed by a pressure decrease within nanoseconds Figure 4 (Chen et al 2004; Mariotto et al 2004; Angehrn, 2008;)

**Mechanism of action.**

Although the exact mechanism of ESWT is not completely understood, research have shown that dose dependent ESWT increases cell-membrane permeability, induces progenitor cell expression, stimulation of mesenchymal stem cell recruitment and regeneration stimulus, endothelial nitric oxide syntheses (eNOS), neural nitric oxide syntheses (nNOS), increased regional blood circulation, transforming growth factor beta1 mediation, type I & III collagenogenesis, and a cascade of endogenous biochemical responses including the down regulation of neurogenic inflammatory substances such as substance P; promoting physiological healing in tendon, bone and wounds (Wang, 2002; Chen et al 2004; Mariotto et al 2005; Siems et al, 2005; Koshiyama, 2006; Schaden et al 2007; Saggini et al, 2008; Moretti et al 2009; Notranicola et al 2010). Use of ESWT in musculoskeletal injuries have shown to be effective for Achilles tendon, patella tendon injuries, plantar heel pain, pseudo arthrosis, and calcerea shoulder tendinitis among other indications (Ogden, 2002; Carter et al 2004; Ogden et al 2004;
There are four different methods of producing medical shockwaves (Figure 5). Electrohydraulic devices are generally high energy devices, peizo-electric and electro-magnetic devices are generally medium to low energy devices, and radialwaves are pressure pulses generated via pneumatic or electro-magnetic generation. ESWT offers a non-invasive, drug free and fairly rapid pathway to recovery, while demonstrating a safety profile that is possibly unequaled in modern rehabilitative medicine. Utilizing the correct energy flux density levels (EFDL) is essential in order to obtain the desired results, as therapeutic outcomes are dose dependent.

Evidence
There has been a vast amount of research conducted using ESWT for various medical applications over the past twenty years, with over 400 studies and trials published in a variety of peer reviewed journals on chronic musculoskeletal applications mainly showing positive results. There has been much debate about the efficacy of ESWT in literature. Most of this confusion was the result of two earlier studies conducted by Buchbinder et al and Haake et al. Although well designed studies, they utilized an EFDL that was too low to produce a therapeutic response for the indication under investigation resulting in what may be considered as a type II reporting error (Rothman, 2010). This prompts for caution when reviewing literature of unfamiliar modalities and treatment techniques. Many clinicians who did not have a working familiarity with ESWT accepted the conclusions of these studies not understanding the dose dependant nature of the treatment. ESWT outcomes are dose dependant, and as demonstrated by Theodore and colleagues in 2004, when they employed the same device utilized in the Buchbinder 2002 trial, but utilizing appropriate energy levels, produced positive treatment outcomes and the investigators concluded that ESWT was an effective and viable option. Similarly Haake and colleagues when utilizing higher energy levels compared to their other trial, concluded that ESWT was an effective treatment option for calcific shoulder tendinopathy. A report compiled by the Australian Safety and Efficacy Register of New Interventional Procedures – Surgical (ASERNIP – S), confirmed that ESWT for calcific shoulder tendinopathy is a well established procedure in Europe, Australia and Japan, and the only reason why very few specialists and clinicians are conducting this procedure is due to the fact that the initial cost outlay for the technology is beyond the funding capability of most medical centers. An evidence based review
conducted by the Accident Compensation Corporation (ACC), 2004 concluded that high energy ESWT was effective for the treatment of chronic plantar heel pain (Carter, 2004), and a recent report compiled for the ACC demonstrated that ESWT delivered good treatment outcomes with long term survival curves (Craig, 2010). ESWT has demonstrated both a good safety record and outcomes for several chronic musculoskeletal conditions, including that of tendinopathies (Ogden, 2002; Weil et al, 2002; Carter et al, 2004; Furia, 2006; Rompe et al 2007; Wang, 2007). Given the clinical frequency of which tendinopathic conditions present in medical practices globally there still remains a lack of evidence based guidelines for treating these conditions especially in chronic states (Frohm, 2006; Crossley et al 2007; Maffulli & Longo, 2008). Reconsideration of the continuous use of traditional pharmacogenics to control inflammation may be necessary as they may not be the most appropriate treatment option especially when considering the risks associated with these agents, and where numerous dated and current evidence suggests that there is an absence of an overt inflammatory process present in chronic tendinopathies (Alfredson et al 2001; Bresalier et al 2005; Sharma, 2005; Wilson 2005; Caldwell et al, 2006; Kearney et al 2006; Magra, 2006; Andres & Murrell, 2008; Kane et al 2008; Kongsgraad et al 2009; Rees, 2009; de Vos et al, 2010; Fosbøl et al 2010; Maffulli et al 2010). The mechanism of action of ESWT has shown to induce the down regulation of inflammatory markers and neurotransmitters, while promoting tissue healing and increased collagen Type I synthesis, and other growth factors, reflecting the innate healing process of tendons. This infers that ESWT may be considered as an option when treating chronic tendinopathies that have failed to respond to at least two or three other non-surgical methods.

**Discussion and conclusion**

There were many aspects of the biochemical responses, pain, healing nature and the neuromechanotransduction with regard to overuse tendinopathies which was beyond the scope of this article to address in detail. The exact etiology of tendinopathy is not fully understood, and while there are several theories as to the pathophysiology of this condition, the commonly accepted cause is seen to be a failed or incomplete healing response devoid of an overt inflammatory expression. However, it may be plausible to suggest that there may be a mutual and synergistic occurrence of a pathogenic inflammatory and degenerative cascade leading to tendon failure, pain and disability commonly seen in tendinopathies. Whatever the causative
Factors may be the fact remains that chronic tendinopathies impact the psychophysiosocial aspect of the individual and presents a clinical challenge. Meaningful interventions will help restore an individual’s general health and productivity which should be the management focus (Bandura, 1995; Cartoni, 2005; Galambos, 2005; Waddell, 2006; Mann, 2007; Craig, 2010). When considering options for managing tendinopathies, it may be useful to consider the regenerative and degenerative processes occurring in tendons as described in literature. The mode of action of ESWT on the human organism infers that it is a viable non-pharmacogenic and non-invasive treatment option when faced with chronic unresponsive musculoskeletal conditions. Patients are returned to work and sedentary duties immediately after ESWT, but rest from undertaking strenuous physical activity is recommended for up to 6 weeks for optimal results. From clinical experience ESWT works very well with gym based physiotherapy 4 weeks after treatment, even on patients who have had previously failed to respond to this method prior to ESWT. This article aims to highlight ESWT as a viable treatment option available to clinicians when faced with chronic unresponsive tendinopathies, and clarify some of the earlier disparities in medical literature as to its efficacy. It is pertinent to note that therapeutic results are dose dependent and therefore selecting the right treatment energy flux density level is essential when treating various musculoskeletal and tendinopathic conditions. When used by an experienced operator with the appropriate treatment protocols ESWT proves to be an effective treatment modality. ESWT should not be used as a primary treatment option for any condition, but may be considered when at least two or three other conservative treatment options have failed to resolve tendinopathic issues.

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Conflict of interest
KC is a director of Kompass Health Associates in private practice and provides ESWT as part of their practice.

This article was undertaken for the purpose of academic discussion. There were neither financial exchange, grants, funding consultancies, stock ownership, stock options, royalties, employment affiliations, nor the promotion of any particular device(s) or their manufacture(s) associated with this article.
Reference


