

Case Report

Chronic Pain after Reported Whiplash Injury – A Patient Case Report

Gunnel Berry *

Private Practitioner, Hunter's Moon Cottage Preston Candover, Basingstoke, UK; E-Mail: gunnel.berry1@gmail.com

* **Correspondence:** Gunnel Berry; E-Mail: gunnel.berry1@gmail.com**Academic Editor:** James D. Adams**Special Issue:** [Complementary and Alternative Medicine in Nervous System Conditions](#)*OBM Integrative and Complementary Medicine*
2019, volume 4, issue 1
doi:10.21926/obm.icm.1901012**Received:** April 13, 2018
Accepted: February 21, 2019
Published: February 25, 2019**Abstract**

Chronic pain is notoriously multifactorial, multifaceted and difficult to manage. Twenty per cent of reported whiplash-injured persons go on to develop a Whiplash Associated Disorder (WAD) where persistent pain becomes chronic with no prospect of change [1]. Paucity of effective therapies to address the effect of WAD enforces new approaches. This case report, with a patient's perspective, illustrates a novel therapy, Adapted Reflextherapy (AdRx), which is akin to reflexology and has been used, developed and revised by the author for two decades, to address the physical and psychological aspects of chronic WAD. While there is no reported single therapy recommended to treat chronic WAD there is evidence that cognitive processes [2] and patient centredness [3] are strongly related to positive outcomes in the treatment of patients with chronic low back pain. Similarly, exploratory [4] and experimental [5] studies suggest that reflexology has an anti-nociceptive effect and may reduce anxiety and stress in conjunction with reducing hyperalgesia. The patient in this case report developed chronic symptoms after a double, opposite-directional car crash 3.5 years prior to commencing AdRx treatment. The patient was encouraged to write a self-reporting pain diary where she describes her predicament and experience of receiving treatment. Her symptoms shifted from a 'stalemate' position to one of improved quality of life (QoL) and reduced pain levels. This case report offers a hypothesis that ongoing pain may be a 'paper



© 2019 by the author. This is an open access article distributed under the conditions of the [Creative Commons by Attribution License](#), which permits unrestricted use, distribution, and reproduction in any medium or format, provided the original work is correctly cited.

trail' of compromised neural plasticity as an effect from not only one injury but that of an array of incidents from which pain-producing peptides have accumulated in the nervous system. As AdRx is applied on nerve endings at the periphery, it is suggested that it acts as a 'counter-irritant' in a neural sense producing an array of descending (inhibitory) signals at spinal and central levels. The case report aspires to illustrate changes in a chronic WAD patient and perhaps offer an opportunity for change in others.

Keywords

Whiplash injury; whiplash associated disorder; chronic pain; quality of life; pain diary; adapted reflextherapy; reflexology; neural plasticity.

1. Introduction

'Whiplash injury' is a controversial diagnosis in today's medical and insurance policy climate. Existence, or non-existence, of whiplash injury after a road traffic accident (RTA) has been reported and discussed in literature with various opinions. Dishonest claims of a whiplash injury affect insurance premiums and call into question the plausibility of those who really do suffer after a motor vehicle accident. Some sceptics consider the diagnosis to be untrue or at least doubtful [6].

Whiplash injury arises from an 'acceleration/deceleration mechanism of transfer of energy to the neck arising from a motor vehicle accident or other setting such as work or sport' [7-8].

Whiplash Associated Disorder (WAD) arises and describes people with ongoing symptoms affecting the whole body as a sequela from the whiplash injury [9]. WAD is associated with chronic pain, anxiety, depression, catastrophising and comorbid health issues [10]. In the UK, 300,000 people were diagnosed with 'whiplash injury' in 2003 [11] with a falling trend for light reported injuries up to 2016. Although there is still no known treatment recommended for WAD or chronic pain perse [12], clinical practices still receive referrals for WAD [9]. There is an abundance of literature supporting biopsychosocial approaches to treat WAD (Sterner Gerdle 2004; Soderlund Lindberg 1999; Jull Vicenzino et al. 2005) and physical therapy has its role to play whereby 'stabilising exercises, low velocity mobilising techniques and ergonomic advice was proven to be superior over a self-management programme' [13]. In addition, while Bogduk [14] supports the notion of zygapophysial joint injury as source for chronic neck pain after a whiplash advocating radiofrequency neurotomy to relieve symptoms, Michaleff, Maher, Christine Lin et al. [15], conclude from their meta-analysis data that radiofrequency neurotomy is a complex procedure and may only be effective in selected patients. They advocate that 'simple advice' is as effective as comprehensive physiotherapy programmes. Concomitantly, AdRx emerged as a treatment for chronic persistent pain after whiplash and WAD [16-18].

This case report presents a patient who, having sustained a double, opposite-directional whiplash injury in a motor vehicle collision gained appreciative, self-reported, enduring improvement after AdRx intervention. It is suggested that by touching the skin, an action potential arises which initiates a neural response which has an effect on central and spinal mechanisms

(ascending) as well as peripheral structures (descending) to change a persistent pain state. It is hypothesised that alterations in neural plasticity play an active role in the measurable changes.

2. Adapted Reflextherapy (AdRx)

Adapted Reflextherapy (AdRx), akin to reflexology, is a therapeutic intervention applied to the feet (hands may also be used) in musculo-skeletal pain patients as a primary assessment and treatment tool. The therapy has been used for over two decades by the author and has been found to facilitate change in patients with chronic pain conditions associated with WAD [18] and persistent pain [18-19].

Reflexology [20-27] states that the medial arches of the foot relate in anatomical terms to the spine (see Figure 1 and 2), and, metaphorically, the anatomical body is 'superimposed' on the foot like a 'pedal homunculus' (Figure 3). AdRx evolved from this theory but was adapted as a therapeutic treatment technique with a supporting working hypothesis during a clinical research episode in a GP practice [28]. It was found to be effective in patients with severe whiplash injury from motor vehicle accidents as well as those who suffered injuries in a serious train crash in London in 1999 [29]. A teaching programme was set up to share the AdRx hypothesis and treatment practice in 2002.

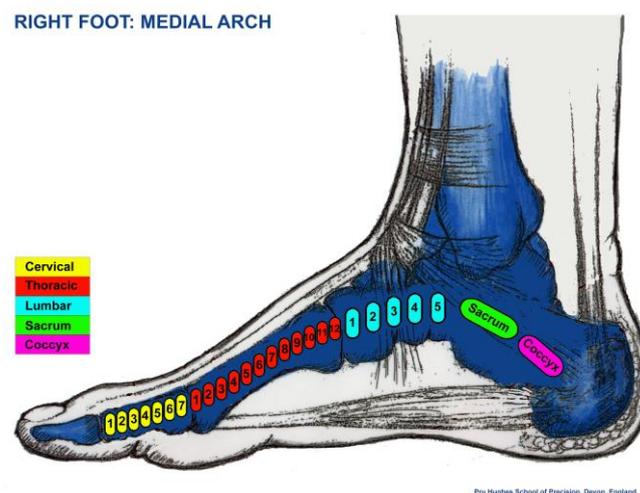


Figure 1 Medial arch right foot.

Spinal segments: Cervical, thoracic, lumbar, sacral, coccygeal (Reprinted with permission: Pru Hughes School of Reflexology).

Colour key: Yellow = cervical, 1-7; Red = thoracic, 1-12; Blue = lumbar, 1-5; Green = sacral, 1-4 fused; Pink = coccygeal fused.

Albeit empirical, the AdRx hypothesis is based on retrospective observations of patients in the clinic which include clinical reasoning in relation to the patient presenting symptoms, past history, anatomy, neurophysiology, neuroplasticity and effect of injury in terms of hypersensitivity, chronicity and centralisation. In addition, AdRx originates from a convergence of theory of reflexology, neurophysiology, chronic pain theories, concept of 'memory of pain', and effect of trauma, and coinciding with two colleagues, one physiotherapist and one reflexologist,

independently demonstrating pressure on one spot on the foot to relieve pain in a corresponding somatic area. For instance, in accordance with Saab & Haynes [30], by touching the skin, an action potential arises within the axon which initiates a neural response at spinal and higher centres (thalamus) which initiates a counteractive response affecting central and peripheral neural mechanisms supported by the notion that ‘massage has an hypoalgesic effect on experimental pain’.

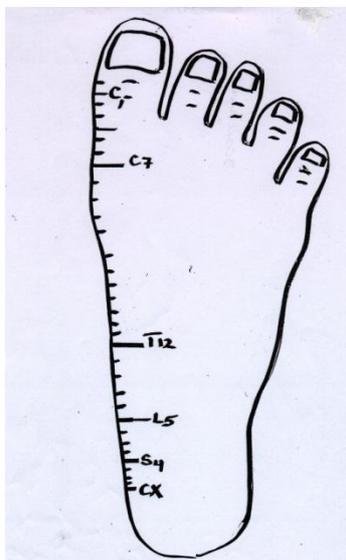


Figure 2 Dorsal aspect - right foot.

Vertebral segments: Cervical (C1-7), Thoracic (T12), Lumbar (L5), Sacral (S4) and Coccygeal (Cx).

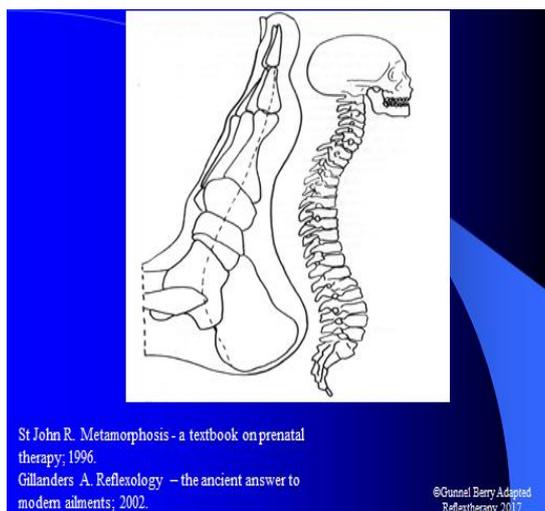


Figure 3 ‘Pedal homunculus’.

Spine superimposed on left foot according to Robert St John. (1996) [44] and Ann Gillanders (2002) [45]. Note anatomical position, i.e. plantar aspect of foot represents anterior aspect of body.

Reflexology has earned recognition to reduce pain in cancer [31-32] in lower limb amputees and phantom limb pains [33]. It has been reported as helpful in conditions including multiple sclerosis, anxiety [34], and childbirth [35]. However, Poole, et al. [36], found insufficient evidence for using reflexology in low back pain sufferers and White AR (2000) and White AR, Williamson, and Ernst (2000) disputed claims for health promotion [37-38] considering 'diagnoses' no better than chance in identifying medical conditions in one blinded study [39].

Nevertheless, using a pseudo-medical phenomenology, AdRx has been found to reduce pain and restore function in patients suffering from persistent pain after injuries. The physiological effects from an acceleration/deceleration mechanism during a car accident, or a fall while skiing or skating or falling off a ladder and slipping on ice or wet floor, however long ago, should be considered as a source of chronic pain. AdRx hypothesises that past injuries could be responsible, or at least, play a role in the present pain patterns. Clinical findings of hypersensitive areas and reduced mobility in the feet, introduces possibilities of understanding the origin of a painful state. It is also hypothesised that in addition to the mechanical compromises after injury, the neural plasticity has also altered to an extent beyond the normal which we call a *compromised neural plasticity* (CNP) state. It describes a state of the neural plasticity which is not quite normal yet functional and without apparent severe damage. It is a term used for patients who present with 'non-specific', persistent pain patterns interpreted as a 'legacy' of a disturbed neural plasticity rather than a response to disease or inflammatory processes. Based on documentation that pain-producing peptides remain in the neural system even after the main pain issues have gone [40], Guez, Hildingsson, Rosengren et al., found nerve tissue markers indicating damage after whiplash trauma [41]. AdRx hypothesis assumes a 'legacy' of peptidal change in the axonal flow after a whiplash injury which increases after each repeated acceleration/deceleration episode. A simple stumble may set the pain-producing peptides in motion and increase adverse neural plasticity activity. This peptidal 'echo' becomes part of the neurophysiological response in the next injury resulting in a CNP, presenting as hypersensitivity, centralisation and continued pain and dysfunction [42]. Although it is not simple to identify nociceptive receptor fields without ambiguity [43], AdRx hypothesises that by tactile exploration of the feet, a reasoned suggestion can be drawn to the causes of a painful state. The great advantage of AdRx is its simplicity and easy access to assess and treat the patient. Areas that are painful during touch are not necessarily painful during fully weight bearing.

2.1 AdRx Examination

As part of an otherwise orthodox physiotherapy assessment, the feet of the patient are examined in supine in the context of a possible spinal injury and sensitivity reflecting the neural elements of pain patterns. The examination detects, reflects and interprets the foot/joint stiffness as stiffness and sensitivity at spinal joint levels including costo-vertebral junctions, symphysis pubis and sacro-iliac joints. Comparisons are made with anatomical pain sites regarding the relevant pain and the anatomical assessment already carried out. The patient is asked questions such as: 'Does this reasoning make sense to you? Does the explanation relate to your symptoms? How do the symptoms not relate to your previous understanding and belief systems you may have learnt from other health professional?' Anatomical models are used to explain clinical reasoning to the patient.

As part of the assessment and treatment, a 'review' process and 'goal-setting' is included at each visit. This highlights signs and symptoms to be assessed at the next visit. A goal may be as simple as walking upstairs without aid, or just being able to stand and sit on a chair without pain. The ultimate aim may be to live a life without taking painkillers. In cases where people do not tolerate having their feet touched the hand can be used. Complete refusal to have the feet touched has occurred on 3 occasions, based on religious grounds.

The feet are examined to detect painful areas with particular focus on the medial arches and crural joints with the patient lying supine (Figure 1 and 2). Degrees of inversion and eversion mobility at the medial arches are noted as well as dorsi- and plantarflexion at the crural joint which are interpreted in relation to the patient's pain presentation. Reduced inversion/eversion mobility indicates reduced thoracic spine mobility. Reduced dorsi/plantarflexion at the crural joint is interpreted as compromised mobility of the symphysis pubis joint indicating reduced pelvic tilt mechanism. Reduced mobility at the 1st metatarsal-phalangeal joint indicates reduced cervical spine mobility, etc.

The depth of manual pressure on the dermis varies depending on the sensitivity of the skin in relevant areas. Areas of chronic nature are frequently found to be less sensitive. Acute areas are often highly sensitive to such a degree that the skin is too sensitive to touch at all in which case the therapist merely 'hovers' over the intended area for a second or two to obtain information, in which case it may be advisable to address the hand instead. It has been found that, as treatment progresses the skin accommodates and reduces sensitivity to allow direct contact. This is interpreted as reduced hyperalgesia and sensitivity.

2.2 AdRx Treatment

The AdRx treatment has high specificity and task purpose in its application, meaning that only areas which are deemed interconnected with the patient's symptoms are treated. Treatment consists of direct pressure on the dermis at 90° direction as much as possible (see Figure 4).

Depth of pressure varies depending on the severity of the patient's symptoms. In acute cases, including high sensitivity on touch, 'light touch' (1-3mm) is used. Deep pressure (3-6mm) is applied in chronic and less sensitive cases.

Duration of continued pressure on one single area depends on chronicity. 'Short' duration in acute cases (30 secs) and 'long' duration in chronic situations (60 secs – 120 secs).

Foot therapy application like AdRx frequently increases pain issues for 24 hours post-treatment. This can be interpreted as exacerbated symptoms and hence a disadvantage to the patient. Conversely, it could be seen as an 'effect' from the treatment, hence, an advantage. Anecdotally, the first treatment causes most 'effect'. Subsequent treatments may increase pain but with reduced intensity. Improvements are not always linear especially in patients with a history of decades of pain. Improvement in these cases is usually slow, interrupted by episodes of pain exacerbation, yet with improvement in mobility, function, moods and quality of life. Even those with complex pain issues are known to achieve change. While a reflexology treatment may require one hour's treatment using reflex zones [46-47], on the whole foot, AdRx is task specific and selects one or two relevant areas for treatment each time. As effective treatment time is approximately 10–20 minutes at each visit, AdRx procedure fits in well in the physiotherapy department.



Figure 4 'Four-point' pressure on Symphysis Pubis, Sacro-Iliac, Hip and Sacral areas.

2.3 Whiplash Injury and Chronicity

A whiplash injury may occur at any time that the body is subjected to a sudden acceleration/deceleration mechanism. Whiplash injuries are graded 0 – IV depending on severity of symptoms at time of impact.

Briefly, WAD Grades include [9]:

- 0 No neck complaint
No physical sign (s)
- 1 Neck complaint of pain, stiffness or merely tenderness
No physical sign (s)
- 2 Neck complaint and musculoskeletal sign (s)
- 3 Neck complaint and neurological sign (s)
- 4 Neck complaint and fracture or dislocation

The effect on the human body at time of deceleration can only be estimated but even small injuries to nerves may set off a cascade of compromises in the neural plasticity which ultimately leads to pain [48]. 20% of whiplash injured patients go on to develop chronic symptoms [49] and women are more affected than men suffering more headaches in the case of vehicle impacts [50, 51]. The ultimate effect of the deceleration forces may depend partially on the individual's anatomical build and strength to resist kinetic energies during the impact. Hypersensitivity, hyperalgesia, catastrophising, depression and bizarre pain patterns are commonly considered to be symptoms of 'central sensitisation' which are the effect from a whiplash injury and commonly called Whiplash Associated Disorders (WAD), to describe the overall involvement of psyche and soma simultaneously. Widespread hypersensitivity is associated with poor recovery [52] and caused by continuous bombardment of adverse action potentials from the periphery to brain and central structures. It is suggested that patients should be assessed for 'intensity of pain, depression and catastrophizing when planning a rehabilitation programme' [10]. In terms of AdRx hypothesis, applying touch and topical pressure facilitates an action potential which appears to affect the whole of the nervous system including the limbic system. Full recovery is not always achievable, and in cases of chronic pain it may take years to fulfil its full potential. Dutiful care should be taken during the history taking to establish whether a patient has ever had an injury or mishap which could be held 'responsible' for the ultimate symptoms as it may have compromised

neural plasticity. The incident may have happened as a child, may seem irrelevant, happened a long time ago and may simply have been forgotten. However, the physical impact on the body may set in motion the adverse neurophysiological responses, as discussed, resulting in a compromised neural plasticity process. This process affects the somatosensory system as well as the limbic system involving the autonomic nervous system resulting in symptoms which are bizarre and may be misinterpreted by patient and health professional alike.

3. Case Report

A 32-year-old female postgraduate researcher, Tina (not her real name), had a motor vehicle accident 3.5 years earlier resulting in Grade 2 I whiplash injuries [9]. At the time of impact, Tina's car was hit at the rear (1st whiplash) during a stationary holdup on the motorway pushing her forward into a crash barrier (2nd whiplash). She felt no pain at time of accident (-s). However, neck pain commenced 3 hours later (Grade I). Tina claimed that the car accident 3.5 years ago still affected her daily life with a variety of symptoms such as headaches, peripheral numbness, shoulder pain, upper and lower back pain, tinnitus, tension in the leg and knee muscles, achy feet and hands, sciatica, carpal tunnel syndrome and, finally, an underactive thyroid which was diagnosed 4 months post-accident. Whether this was coincidental or a product of injury is not known. She described her symptoms as 'achy limbs, felt heavy and painful all over from general use with pain radiating around the body even into nails and teeth'. Joints felt 'sticky' and she had to keep moving around continuously to 'free up'. Some of the original symptoms eased one year after the injury but had now returned.

Working at a university establishment, Tina followed up an invitation to pursue a research project in the usefulness of AdRx for chronic neck pain sufferers [53] carried out by a physiotherapy MSc student. The research project included four weekly treatments @ 10 minutes each session, and aimed to assess efficacy to reduce pain in chronic neck pain patients using AdRx as a method of treatment. Tina was, by chance, allocated to the sham group and received a 'pretend' treatment which included a mild foot massage on the lateral aspect of the foot (away from the 'spinal' areas). The sham treatment had nevertheless a mild positive effect and reduced Tina's neck pain a little. She became curious to possible further effects of treatment and wished to continue with the AdRx treatment after completion of the research project. The 10 volunteers included in the project were invited to continue with further treatment offered by the therapist at a reduced commercial rate. Tina self-funded her treatment.

3.1 Previous Therapies and Present Status

By the time Tina started her AdRx treatments 3.5 years after the original injury, she had received, and benefitted from, 43 treatments of physiotherapy and chiropractor treatments. A rheumatologist offered advice and analgesic medication, such as amitriptyline, and magnesium (Mg).

Tina suffered from daily symptoms of persistent pain and reduced 'quality of life' since the car accident. Her pain was under control but 'quality of life' issues were still 30% away from being fully recovered. She had daily severe headaches, neck pain, low back pain, all-body sensory unexplained pains increasing with sudden fast movements and any kind of lifting even light bags. Sleeping was partially disturbed depending on positioning, work increased backache from

prolonged sitting at a desk working on the computer with muscle tension and joint stiffness to protect her from the pain which in turn increased tiredness and affected mental concentration. To relieve symptoms, Tina stood up every 20 minutes to do gentle stretching exercises. Her daily routine included 1.5 hours of yoga, cycling, Pilates and swimming in various combinations to keep the worst symptoms away.

Previous Medical History: Fractured wrist age 7, under-active thyroid developed post-injury

Social: Tina lived with her parents at the time of the accident and travelled by car to work. She was a normal young lady enjoying her research work which took her abroad from time to time. She enjoyed outdoor life and would do much more in the form of exercise and outdoor activities if only the persistent pains would ease.

3.2 Assessment

3.2.1 In Standing

Posture

Tina was tall, slim with an erect posture.

3.2.2 Mobility

Spinal mobility:

Hypermobility tendency [54].

Lumbar Spine: Forward flexion – touch floor

Extension - beyond 15° with degree of discomfort

Side-flexion - unremarkable

Thoracic spine: Unremarkable

Cervical spine: Unremarkable

Shoulder mobility: Full range

3.2.3 In High Sitting

SLUMP [55] tested positive (+ve) on both legs increasing tension posterior-knee and down the leg at full knee extension. Posterior leg symptoms were reduced with neck extension.

3.2.4 In Lying

SLR (Straight Leg Raising) was equal at 85°, limited by posterior knee stiffness but no pain.

Hip and knee mobility: No abnormality detected

3.2.5 Pain

Self-reported pain areas and quality of pain 3.5 years post-injury as described in Figure 5.

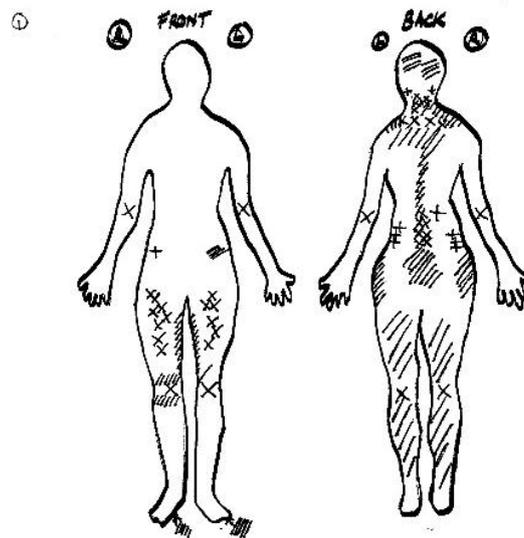


Figure 5 Pain September 2013 (1st Assessment) (revised for printing purposes).

Pain Chart Legend: **Front** (+ = 'stabbing' pain (R) hip; x = 'aching' pain elbows, thighs, knees; //// = 'other symptoms' medial thighs, anterior (L) hip, anterior (R) knee.), **Back** (+ = 'stabbing' pain back neck, lower back; x = 'aching' pain neck, shoulders, spine and posterior knees; //// = 'other symptoms' back of head, (R) shoulder, thoracic & lumbar spine, sacrum, bilateral posterior hips and full legs including soles of feet.).

3.3 Foot Examination as Per AdRx

General impression of feet: flexible and mobile.

Skin condition of feet: unremarkable.

3.3.1 Tenderness on Palpation

Bilateral areas: Symphysis Pubis joints (SPJ), Lumbar segments 1-5 (L) > (R), and Cervical C6/7 level.

Mobility of medial arches was restricted at Lumbar areas and rotation of 1st phalanx at metatarsal/phalangeal joint.

Outcome Considerations

- Anatomical build
- Gender
- Hypermobility
- Chronicity since time of accident
- Personal profile
- Motivation
- Diagnostic challenges and reasoning

Anatomical Build: 'The slender female neck is more vulnerable to injury than the average male neck' [56] supporting the opinion that females are more likely to develop chronic neck symptoms associated with an acceleration-deceleration mechanism to the neck [56].

Gender: Tina is vulnerable due to her slender build and gender, following a trend that women have more persistent neck pain and headaches than men do by a ratio of 7:3 [50]

Hypermobility: Hypermobility is a generic ability of soft tissues to extend beyond normal joint flexibility resulting in poor joint stability. In severe cases, the heart muscle may be affected. Anecdotally, the author has found that patients have persistent pain in conjunction with hypermobile joints. Even small degrees of excessive joint flexibility have been found to cause excessive pain during joint movement. The question arises, is it possible that hyperflexibility is, in part, responsible for ongoing pain states? The patient has to continuously keep on stretching tissues and joints to achieve comfort and pain-free movements as observed in Tina's case. No literature has been noted to date to support this observation in cases of whiplash injury and chronicity.

Chronicity: 3.5 years post injury, chronicity is well established. Prognosis and predicted outcomes can only be speculative. The general consensus is that biopsychological approaches may offer coping strategies but no specific treatment approach has proved superior to another. From this rather bleak baseline, no prognosis was made.

Personal Profile: Tina was a highly intelligent young lady with a good job at a higher education establishment. She worked conscientiously with her daily diary to produce a clear picture of her reactions after each treatment and attainment targets she had set herself. Her prediction of constant pain put her under strain and made her understandably mildly anxious. After 3.5 years of continuous symptoms her endurance waned from time to time.

Motivation: Tina was highly motivated to improve her quality of life situation. She wanted to increase her outdoor activities and do more travelling.

Diagnostic Challenges and Reasoning: Central hypersensitivity to peripheral stimulation was found in whiplash patients by Curatolo, Petersen-Felix, Arendt-Nielsen et al. [57], and is associated with poor recovery [58] which in this case was prevalent and demonstrated by the self-reported diagrams. Experience using AdRx in hypersensitive patients has determined that caution has to be observed in response to the magnitude of reactions which may occur in patients having suffered a neck injury. The treatment has to commence in areas relating to the base of the spine at the first visit and finally, on the 3rd visit possibly, include the neck area. Adverse reactions have been observed in two female patients aged 22 and 25, by applying too much pressure on the feet resulting in, temporarily, syncope and fainting. The outcomes from the treatments are always discussed and negotiated between therapist and patient.

3.4 Outcome Measures

3.4.1 Principle of Outcomes

Tina had achieved good progress from physiotherapy and osteopathic interventions during the 1st year post-injury. She had adopted a self-managing approach using exercises, stretching and swimming to cope and manage her ongoing symptoms. Starting AdRx as a method of treatment, it was agreed to use a 'phenomenological' approach to assess progress by using a reflective pain diary [59] recording variations in quality and quantity of pains in different part of her body by drawing body charts and writing verbal annotations. The body charts were compared weekly comparing changes in pain patterns over time. Straight leg raise (SLR) is a neurodynamic test. It

has reasonable validity and Charnely stated in 1951 that ‘the straight leg raise was more important than all of the other clinical and radiological signs put together’ (Butler 2000) [60].

- JOINT MOBILITY
- SLR [60] (Straight Leg Raise)
- SLUMP – (a SLR test in sitting) [60].
- PAIN DIARY

Joint Mobility: Spinal mobility was never an issue due to hyperflexible joint mechanisms compared to normal joint mobility. However, in relation to hypermobility, Tina had reduced hip flexion with 90° flexion at 1st assessment. The expectations of her joint mobility were adjusted to accommodate this anomaly.

SLR 85° bilateral

SLUMP [61] + ve bilateral

Pain Diary: The aim of Tina’s treatment was to reduce persistent pain and improve quality of life (QoL), both of which are ‘individual’ interpretations. Huskinson states that ‘pain is a personal psychological experience’ [62]. Using a pain diary Tina had an opportunity to identify, interpret and express her own moods and variations in the variations of pain patterns. She drew ‘quality’ and ‘quantity’ pain changes on the body chart and also narrated the effects from the treatment. Comparisons between drawings and writing week by week helped to identify changes. This approach worked well.

She described her symptoms in Figure 5, 6 & 7:

‘achy pain’ (x)

‘stabbing pain’ (+) on two occasions in description under figures

‘other pains or symptoms’ (///)

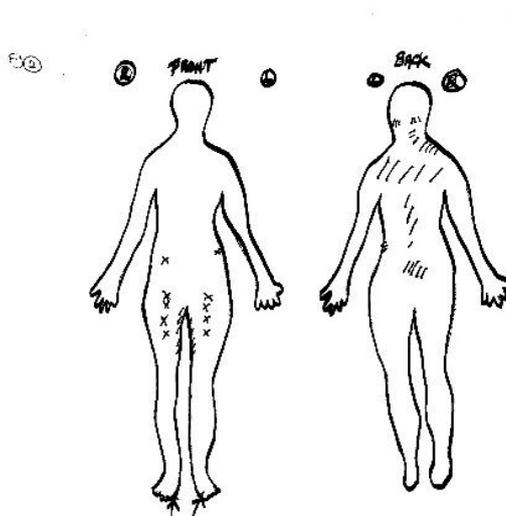


Figure 6 Pain February 2014 – 6 months after starting treatment (revised for printing purposes).

Front (x = (achy pain) at anterior (R) hip and both thighs; /// = (other pains or symptoms) medial upper thighs.), **Back** (/// = (other pains or symptoms) at base of skull, thoracic and lumbar spine, soles of feet; ‘achy pain’ (x); ‘stabbing pain’ (+); ‘other pains or symptoms’ (///).).

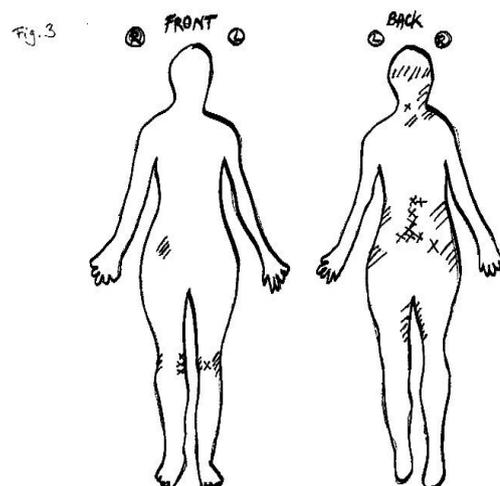


Figure 7 Pain December 2014 - 16 months after starting treatment (revised for printing purposes).

Front (x = Medial and lateral knees.), **Back (R)** (/// = 'other symptoms' at back of head, (R)side of neck, T1 area, bilateral hips and medial aspect of top of thighs.).

3.5 Summary from the 1st Assessment

A rear shunt (injury 1) forced a frontal crash (injury 2) resulting in a double, opposite-directional, blow to spinal structures 3.5 years ago resulting in a grade 2 WAD. Ongoing symptoms are considered to be CNP-producing severe hyperalgesia, allodynia and maladaptive changes affecting mood and quality of life in accordance with 'central sensitisation'. Mild to moderate arthrogenic involvement in association with hypermobility was noted.

3.6 Prognosis at 1st Assessment

A prognosis was not made in this case because of the unpredictability of the case in terms of chronicity, uncertainty of widespread symptoms in conjunction with hypermobile joints. A question mark was raised whether the thyroid gland had responded adversely due to the incident or may have been an 'in the making' already prior to the road traffic incident.

3.6.1 Goal Setting

It was decided to carry out 4 initial, consecutive treatments and review overall outcomes in view of satisfaction and changes. It was decided to complete another 6 treatments. Minor goal setting was completed at each treatment episode.

3.6.2 Timeline

Tina received 10 AdRx treatments spaced over 6 months. After finishing all treatments, she got in touch after 10 months and submitted a 'body-chart', Figure 7, finalising the overall outcome.

3.6.3 Treatment

Each visit lasted 30 minutes including 10 minutes assessment with a review, 10 minutes active treatment and 10 minutes to organise a follow-up with review to reflect on quality of life changes and pain patterns. Treatment was carried out in supine with two pillows under the knees to create a mild flexion position of the lumbar spine. The treatment focused on the symphysis pubis/sacroiliac joint areas including the plantar and dorsiflexion at the crural joint of each foot but included the whole of the lumbar spine with emphasis on 'stretching' and 'linking' upper and lower ends of the autonomic chain. Treatment outcomes were noted after each treatment. Except for a sensation of dizziness, she did not experience any other adverse effect from the treatment(s). All treatments were carried out by the author in her private rooms.

3.6.4 Results

Joint Mobility: Spinal mobility was not an issue but did nevertheless increase mildly beyond normal range of movement in accordance with a tendency to hypermobility.

SLR: 100°+ on the 9th treatment

Slump: 7th visit showed full knee extension with full dorsiflexion of the ankle on both sides. Asymptomatic progress was maintained at the 10th visit

Pain: Self-reported pain charts and conscientious and expressive narratives of her experiences after AdRx treatments demonstrated the changes in quality and quantity of pain in the whole body. Tina was able to differentiate the outcomes as she became increasingly aware of changes in her symptoms. This qualitative approach offered a chance to measure changes in pain using 'size of pain areas' to describe quantity rather than a more orthodox Visual Analogue Scale (VAS) [63], which in terms of chronic pain is unsatisfactory [64-65].

3.7 Conclusion at End of Treatment (6 Months)

Figure 5, 6 & 7 show changes in both *quality* and *quantity* of pain on the body charts. The +ve outcomes in both QoL and pain, interspersed with a few short-lived ups and downs of pain, were maintained 1 year after stopping the treatment. Tina was able to manage her 'downs' by self-management over time.

Tina was able to endure and enjoy more outdoor activities, travel widely and was able to sit for longer periods without fear of complete exhaustion and increased pain. She was satisfied that the AdRx treatment made a big difference to facilitate improvement in her condition and felt a strong sense of empowerment overcoming her symptoms. Although there were still episodes of discomfort and low back pain from time to time, they were of shorter duration and easier to overcome. No other treatment intervention was used. After 10 treatments it was decided that sufficient progress had been achieved for Tina to 'go solo'. The overall impression of using AdRx in WAD patients with chronic pain is that even with persistent pain and associated symptoms it is possible to achieve altered somatosensory conditions.

3.7.1 +ve Aspects of Tina's Treatment

- ✓ Long-lasting benefits from treatment compared with other treatments.

- ✓ Despite chronic symptoms, lasting improvement was achieved.
- ✓ Activity, exercise endurance, quality of life improved.
- ✓ Hope at the end of the tunnel.
- ✓ Progressive improvement.
- ✓ Tina pointed out to the therapist to 'target most effective areas' as far as she was concerned. This helped to make relevant choices in relation to the painful spots on the feet (which in turn would affect the anatomical areas).
- ✓ Tina thought this was the most positive and effective treatment out of all treatments she had received.
- ✓ After a few treatments, Tina suggested that she was less sensitive to touch – this was interpreted as change in the hyperalgesic condition of nerve endings.
- ✓ Tina gained confidence to be more active and has now the duration to fulfil those ambitions.
- ✓ Tina had increased strength to carry out household tasks.
- ✓ Felt she had more energy in general.

3.7.2 -ve Aspects of the Treatment

- ✓ Increase of pain after treatment; worst after 1st session; interesting that "I felt like my worse symptoms from several years before had returned".
- ✓ Persistent pain still there albeit much reduced.
- ✓ Dizziness: a new symptom.

3.8 Patient Perspective

3.8.1 Experience of Adapted Reflextherapy Described by the Patient

18th February 2018:

Three and a half years before I started Adapted Reflextherapy, I was involved in a car accident having been hit at an angle from the rear and pushed into a crash barrier on a motorway. The classic symptoms of whiplash started after four hours, with new symptoms emerging up to two years post-accident. These included pains or aches in my head, shoulders, upper back, lower back, spine, legs, knees, feet, hands, plus widespread stiffness and achiness, sciatica, tinnitus, hypothyroidism and concentration problems. Most severe was the pain in my lower back and neck. Some of these I still have today but are now very much reduced.

Before I had Adapted Reflextherapy, the effects of whiplash were reduced through five months of physio, three years of chiropractic treatment, six months of magnesium, a treatment of amitriptyline, aromatherapy, Pilates and yoga. The most effective of these was magnesium, but Adapted Reflextherapy changed the game again.

Prior to the treatment I was in constant pain. Each morning I would wake up extremely stiff and needed to stretch or undertake gentle exercise for 45 minutes each morning to relieve the pain. I could sit for about 1 hour at a time in the morning, maybe two hours in the afternoon before the stiffness and pain became too much. Being active was important. I had built up my walking and cycling to about 2 hours (with lots of rests), but this was at times

challenging and often resulted in achiness, stiffness, and tiredness, all of which could only be relieved by stretching. Resting made the symptoms worse. My neck and back pain meant housework, in particular bending, was challenging and had to be broken down into smaller chunks. I struggled to cook a simple meal after work. Lifting heavy objects was impossible, I struggled to carry a handbag and could not live an independent life. In addition to my morning exercise, I exercised 45 minutes each evening, plus as I spent much of my day working on a computer, hourly short walks and/or stretches were essential to reduce pain.

In the days after my first Adapted Reflextherapy treatment, my legs felt tight, I had heightened pain in my neck and head, was dizzy and felt very achy and heavy. I was tense and often jittery with tingling nerves all over my body. It felt like my worse symptoms from several years before had returned, in addition to the new symptom of dizziness. After a few days these subsided, and the pain in my back was reduced.

Over 5.5 months, I had ten treatments. Each targeted a different part of my body so that the extent and type of pain was substantially reduced, particularly in my head, neck, lower back and legs. As with other treatments or exercise programmes, some treatments were more effective than others. I talked with Gunnel to target the most effective areas of pain, and this helped.

Overall, this treatment was the most positive and effective one I received: I could reduce the amount of my daily exercises and stretches and became less reliant on them. I can now do a greater range of exercises and undertake exercise by choice rather than as a means to reduce pain. Immediately after my final treatment, I was able to sit for several hours at a time and I really enjoyed watching my first film which I didn't need to exercise through! Three months after finishing the treatment I was able to walk for 5 hours carrying a small handbag, with less problems than before. Generally, I had more energy and also was less sensitive to touch. I was also able to do housework and cook for longer. Gradually as I gained more strength and confidence, the range of activities I could do increased in number, difficulty and duration. Importantly, unlike other treatments, apart from the occasion wobble, the benefits have been long-lasting, and this was a step-change in my prognosis. The only long-lasting negative effects of the treatment I received was dizziness on lifting heavy objects which subsided after a couple of years and an involuntary jitter of my limbs a few times a day which still persists. Although I still have some pain, this is very much less than before, is less widespread and is variable in intensity, and I have much more control of it. At times, I have hours now which I am pain free and importantly have achieved activities that I never thought I would be able to do again if it was not for the Adapted Reflextherapy treatment.

4. Discussion

WAD is the result of ongoing symptoms from a whiplash injury including 'central sensitisation' where pain is modulated and controlled by higher centres which also control moods [66]. There is no single therapeutic intervention which is recommended in chronic and persistent pain states apart from pain management programmes, tailored to the individual [13], and emphasising psychological aspects of pain [67]. Here, we give a case report of a lady described as suffering from WAD for 3.5 years. She was selected to represent a novel approach in treating WAD, whereby it is hypothesized that sensory input from the periphery produces a neural activation which

encompasses higher centres thereby including the psyche as well as the soma. The patient was advised to express her treatment experience using a self-reporting (narrative) diary including drawing body chart images to describe areas of pain and adverse sensations. This was considered a suitable tool to describe physical and mental changes in the patient. The role of the therapist was to focus on measurable somatic alterations as has been praxis of the author throughout her clinical practice. The importance of incorporating teaching of pain mechanisms during a course of treatment is acknowledged. In this case, the patient had had plenty of opportunity to learn about pain management and causes of pain from a series of other therapists. It was not necessary to repeat these psychosocial aspects of pain. The research methodology chosen in this presentation aims to combine the quantitative changes and qualitative impressions to give the patient a voice in order to encourage improved clinical practice. Ong and Coady write that ‘... qualitative approaches ... provide insights into the way people live with pain and disability, how patients and clinicians interact, and how professional education can be improved’ [68].

Pain is an enigma with many variables and there is a call for further insight into chronic pain [69]. It is nevertheless my opinion, based on observations and clinical experience, that chronic pain conditions are frequently associated with a past history of sudden deceleration mechanisms of the body whereby minor injuries occur, invisible to the eye or examination, leaving a trail of compromised neural activity. It is as if the nervous system has accommodated itself to a ‘siege’ mentality via reactions in its neural plasticity and cannot re-set itself to normal workings. It reacts like a diesel engine filled with petrol – it coughs and splutters and stops. Similarly, the chemistry does not gel and the body aches and hurts and finally gives up to pain. Patient choices are limited in terms of how they can help themselves, let alone understand ‘why’ they have pain. In orthodox musculoskeletal pain management, ‘pain’ is considered an inflammatory, or disease, process yet seldom as an ongoing neurophysiological reaction per se after a deceleration mechanism and blow to the soma. Non-steroidal anti-inflammatory medication is the ‘first aid’ intervention to combat the pain, but, it does not solve ongoing persistent pain. It is of interest to note that Olausson et al. [70] propose a particular hypothesis including CT (connective tissue) afferent coding property and pathways in gentle skin-to-skin contact which they name ‘the social touch hypothesis’. This hypothesis applies to unmyelinated tactile afferents in non-glabrous skin (in rats). Maybe there is an explanation waiting to be discovered (in humans) which could justify and explain the sometimes drastic and enigmatic changes observed during and after a foot treatment? The AdRx hypothesis states that touch on the feet is able to moderate acute and chronic pain states acting as a neuro-plasticity counter irritant.

With regards to the overall importance of the central mechanisms involved in pain production, AdRx offers a hypothesis that ‘touch’ initiates a sensory afferent impulse signalling and affecting ongoing neural plasticity. This in turn will affect pre-existing neural compromises from previously injured areas. Ongoing pain may potentially originate from the content of the axonal flow from another injury, including *quality* and *quantity* peptides, in the autonomic as well as the somatosensory nervous systems. Peptidal changes vary depending on demand in normal and abnormal circumstance, such as after injury [48, 71] and will affect, and are dependent on, synaptic integrity as well as the axonal transport [30]. Peptidal changes vary depending on demand in normal and abnormal circumstance, such as after injury [48, 71]. It is not possible to determine nor differentiate the origin of pain due to, as discussed, its transient behaviour and intermittent occurrence. See more in Khalsa (2004) [72]. Hypothetically, the spinal cord area,

medulla, autonomic chain and peripheral connections are all implicated to a greater or lesser degree in their role to conduct information and may be a source of pain. There is not one source of pain but a widespread effect and reaction in psychoneuroimmune (PNI) responses throughout, including in the autonomic nervous system chain which links closely to the limbic system [73]. Treede, Jensen, Campbell et al. call for more integrative research into the spinal segments that are part of pain-producing structures [74]. AdRx is known to have facilitated change in hyperalgesic and hypersensitive persons with persistent pain. It has been a first choice of treatment to reduce hypersensitivity and hyperalgesia in acute and chronic pain after whiplash including WAD. Once 'hypersensitivity' in tissues has been reduced, orthodox methods of physical therapies may be applied as necessary which hitherto have been impossible to implement due to the hyperalgesia of the tissue matrix [19].

AdRx intervention appears to have had a favourable effect on hypersensitive symptoms in the context of injury and WAD. Although Tina still suffers episodes of pain, they are less frequent and less severe. Pain no longer dominates her life and QoL has much improved. To date, the improvements have persisted beyond 18 months. Tina had tried various coping strategies during 3.5 years to overcome her dysfunctional existence. As per her body charts, she found that the AdRx treatment assisted in reducing her uncomfortable symptoms. Six months after her first treatment, hypersensitive areas had reduced in size and intensity. Ten months later, during which no treatment was carried out, minor low backache symptoms seem to have recurred but without interference to daily activities.

Tina experienced reduced neck pain after having had a sham treatment in the research project. This may be suggestive thinking like a placebo effect, i.e. a positive effect without a real neurological link. Placebo is a powerful usage of suggestive thinking in the treatment of pain. In a postal questionnaire of 100 members of the Association of Chartered Physiotherapists in Reflex Therapy (ACPIRT), it was considered by the majority of the respondents that 'placebo' played a 25% part in the outcome of the patient. In other words, the effect of the treatment played a 75% part in changes that could be observed and self-reported by the patient [75]. Does this support or negate the treatment philosophy, i.e. by stopping treatment, you stop incoming action potentials into the nervous system hence symptoms have a tendency to return albeit with lessened power and strength.

Hypermobility was a comorbidity in Tina's case. This has been found to be a common feature in individuals who do not recover fully from injury. The reason for this is not known but should be considered in cases of 'non-recovery'.

5. Takeaway Message

While AdRx remains an enigma, in terms of chronic pain and unresolved WAD, patients have responded well to a foot application regardless of the origin of their pain and chronicity of symptoms. After 20 years of clinical experience in using AdRx, it is not considered a fanciful notion but aims to convey the capacity for the neural plasticity to self-regulate. Chronicity leads to comorbidities and unfortunately, in some cases, to suicide [76]. We have to dare to rethink how pain is maintained with concomitant physiological responses in order to overcome the challenge of persistent pain. The world is desperate to find a way to combat chronic pain. This case report

aims to raise awareness of an alternative approach to chronic pain in WAD. It suggests a coherent interconnective neural signalling system which can be utilised to change pain behaviour.

Informed Consent

Patient provided informed consent – yes.

Author Contributions

Gunnel Berry did all the research work of this study.

Competing Interests

The author runs courses in AdRx. A book is for sale on the AdRx website.

References

1. Malanga G, Nadler S. Whiplash. Philadelphia: Hanley & Belfus; 2002.
2. Woby SR, Roach NK, Urmston M, Watson PJ. Outcome following a physiotherapist-led intervention for chronic back pain: The important role of cognitive processes. *Physiotherapy*. 2008; 94: 115-124.
3. Cooper K, Smith BH, Hancock E. Patient-centredness in physiotherapy from the perspective of chronic low back pain patient. *Physiotherapy*. 2008; 94: 244-252.
4. Samuel CA, Ebenezer IS. Exploratory study on the efficacy of reflexology for pain threshold and tolerance using an ice-pain experiment and sham TENS-control. *Complement Ther Clin Pract*. 2013; 19: 57-62.
5. Kiriknakriengkrai S, Komar S, Hughes CM, McDonough SM. An experimental study on the effect of reflexology on the nervous system in healthy adults. *Altern Ther Health Med*. 2011; 17: 32-37.
6. Todman D. Whiplash injuries: A historical review. *Int J Neur*. 2017; 8: 1-6.
7. Spitzer WO, Skovron ML, Salmi LR, Cassidy JD, Duranceau J, Suissa S, et al. Scientific monograph of the quebec task force on whiplash-associated disorders: Redefining “whiplash” and its management. *Spine*. 1995; 20: 1S-73S.
8. Holm LW, Carroll LJ, Cassidy JD, Hogg-Johnson S, Côté P, Guzman J, et al. The burden and determinants of neck pain in whiplash-associated disorders after traffic collisions: Results of the bone and joint decade 2000-2010 task force on neck pain and its associated disorders. *Spine*. 2008; 33: S52-S59.
9. Moore A, Jackson A, Jordan J, Hammersley S, Hill J, Mercer C, et al. Clinical guidelines for physiotherapy management of whiplash associated disorder. *Chartered Society of Physiotherapy*; 2005.
10. Börsbo B, Peolsson M, Gerdle B. Catastrophizing, depression, and pain: Correlation with and influence on quality of life and health – A study of chronic whiplash-associated disorders. *J Rehabil Med*. 2008; 40: 562-569.
11. Crouch R, Whitewick R, Clancy M, Wright P, Thomas P. Whiplash associated disorder: Incidence and natural history over the first month for patients presenting to a UK emergency department. *Emerg Med J*. 2006; 23: 114-118.

12. Thacker M. Physiotherapy management of whiplash injuries: A review. In: Gifford L, editor. *Topical Issues in Pain 1: Whiplash: Science and Management*; 2013; 93-104.
13. Kidd B. The contribution of neurogenic plasticity to persistent neck pain: Implications for diagnosis and therapy. In *Touch*. 2010; 131.
14. Bogduk N. On cervical zygapophysial joint pain. *Spine*. 2011; 36: S194-199.
15. Michaleff ZA, Maher CG, Lin CW, Rebeck T, Jull G, Latimer J, et al. Comprehensive physiotherapy exercise programme or advice for chronic whiplash (PROMISE): A pragmatic randomised controlled trial. *Lancet*. 2014; 384: 133-141.
16. Berry G. Adapted reflextherapy – A treatment for spinal pain and whiplash injury. *Focus Altern Complement Ther*. 2007; 12: 7.
17. Berry G. Adapted reflextherapy in whiplash associated disorder. *Congress Proceedings; Birmingham, UK; Chartered Society of Physiotherapy (CSP)*; 2006.
18. Berry G. Adapted reflextherapy in treatment of whiplash related injuries – A case for adoption? 14th International World Physical Therapy Congress; 2003; Barcelona, Spain.
19. Berry G. *Adapted reflextherapy: An approach to pain*. Dorset: Honeybee Books; 2017.
20. Bayly D. *Reflexology today*. London: Thorsons Publishing; 1982.
21. Byers D. *Better health with foot reflexology*. 3rd ed. St Petersburg (FL): Ingham Publishing; 2006.
22. Ingham E. *Stories the feet can tell thru' reflexology and Stories the feet have told thru' reflexology*. 10th reprint. St Petersburg (FL): Ingham Publishing; 2005.
23. Issel C. *Reflexology: Art, science and history*. Sacramento (CA): New Frontier Publishing; 1996.
24. Lett A. *Reflex zone therapy for health professionals*. London: Churchill Livingstone; 2000.
25. Marquardt H. *Reflexotherapy of the feet*. Stuttgart-New York: Thieme Verlag; 2000.
26. O'Hara C. *Core curriculum for reflexology*. London: Douglas Barry Publications; 2006.
27. Stormer C. *Reflexology*. London: Hodder & Stoughton; 2004.
28. Hall H, Cook F. *Audit of a GP practice based physiotherapy service, wiltshire and swindon healthcare NHS trust and Swindon & Marlborough NHS trust*. Executive Summary; 2000.
29. Berry G. Adapted reflextherapy; Theory and practice to treat Musculo-Skeletal pain. In: Berhardt LV, editor, *Adv Med Biol*; 2018; 129.
30. Saab CY, Haynes BC. Remote neuroimmune signaling: A long-range mechanism of nociceptive network plasticity. *Trends Neurosci*. 2009; 32: 110-117.
31. Stephenson N, Dalton J. Using reflexology for pain management. *J Holist Nurs*. 2003; 21: 179-191.
32. Mackereth P, Tiran D. *Clinical reflexology – A guide for health professionals*. London: Churchill Livingstone; 2002.
33. Brown C, Lido C. Reflexology treatment for patients with lower limb amputation and phantom limb pain: An exploratory pilot study. *Compl Ther Clin Pract*. 2008; 14: 124-131.
34. McVicar AJ, Greenwood CR, Fewell F, D'Arcy V, Chandrasekharan S, Alldridge LC. Evaluation of anxiety, salivary cortisol and melatonin secretion following reflexology treatment: A pilot study in healthy individuals. *Complement Ther Clin Pract*. 2007; 13: 137-145.
35. Tiran D, Mackereth P. *Clinical reflexology – A guide for integrated practice*. 2nd ed. Edinburgh: Churchill Livingstone Elsevier; 2011.
36. Poole H, Murphy P, Glenn S. Evaluating the efficacy of reflexology for the management of chronic low back pain. *Focus Altern Complement Ther*. 2010; Suppl1: 47.

37. White AR. Reflexology: Unchartered waters. *Complement Ther Med*. 2000; 8: 149.
38. White AR, Williamson J, Hart A, Ernst E. A blinded investigation into the accuracy of reflexology charts. *Compl Thera Med*. 2000; 8: 166-172.
39. Ernst E, Pittler M, Wider B. *The desktop guide to complementary and alternative medicine. An evidence-based approach*. 2nd ed. Philadelphia (PA): Mosby Elsevier; 2006.
40. Butler D, Moseley L. *Explain Pain*. Adelaide, Australia: NOI Group publications; 2003.
41. Guez M, Hildingson C, Rosengren L, Karlsson K, Toolanen G. Nervous tissue damage markers in cerebrospinal fluid after cervical spine injuries and whiplash trauma. *J Neurotrauma*. 2003; 20: 853-858.
42. Berry G. Whiplash: Risk factors, management and long-term effects. In: Berhardt LV, editor. *Advances in Medicine and Biology*. NOVA publication; 2017; 122.
43. Khalsa PS, Zhang C, Qin YX. Encoding of location and intensity of noxious indentation into rat skin by spatial populations of cutaneous mechano-nociceptors. *J Neurophysiol*. 2000; 83: 3049-3061.
44. St John R. *Metamorphosis – A textbook on prenatal therapy*. Winchester: Robert St John Publisher; 1996.
45. Gillanders A. *The ancient answers to modern ailments*, 6th ed. United Kingdom: J.W. Arrowsmith Ltd; 2002.
46. Dougans I. *Reflexology and the 5 elements and their 12 meridians – A unique approach*. London: Harper Collins; 2005.
47. Fitzgerald WF, Bowers EF. *Zone therapy or relieving pain at Home*. Columbus, Ohio; 1917.
48. Greening J. Minor nerve injury: An under-estimated source of pain. In *Touch*. 2001; 96: 7-12.
49. Treves KF. Understanding people with chronic pain following whiplash: A psychological perspective. *Topical Issues in Pain*, eds. Louis Gifford. Falmouth, Adelaide: NOI-press; 1999.
50. McCrory PR. Whiplash-related headache. *Whiplash*, eds Gerard Malanga & Scott Nadler, Chap 8. Philadelphia: Hanley & Belfus; 2002.
51. Stemper BD, Corner BD. Morphology and whiplash injuries. *Whiplash Injury*, eds Helge Kasch, Dennis Turk, Troels Jensen, Wolters Kluwer. Philadelphia; 2016.
52. Sterling M. Differential development of sensory hypersensitivity and a measure of spinal cord hyperexcitability following whiplash. *Pain*. 2010; 150: 501-506.
53. Paget L, White P. An exploration study in to Adapted Reflexotherapy as a treatment for chronic neck pain: A randomised controlled pilot study. Southampton University; 2014.
54. *Hypermobility Syndromes Association*. [Internet] April, 2018.
55. Petty N, Moore A. *Neuromusculoskeletal examination and assessment. A Handbook for Therapists*. London: Churchill Livingstone; 2002.
56. Taylor J, Twomey L. Whiplash injury and neck sprain: A review of their prevalence, mechanism, risk factors and pathology. *Crit Rev Phys Rehabil Med*. 2005; 17: 285-299.
57. Curatolo M, Petersen-Felix S, Arendt-Nielsen L, Giani C, Zbinden A, Radanov B. Central hypersensitivity in chronic pain after whiplash. *Clin J Pain*. 2001; 17: 306-315.
58. Sterling M, Jull G, Vicenzino B, Kenardy J. Sensory hypersensitivity occurs soon after whiplash injury and is associated with poor recovery. *Pain*. 2003; 104: 509-517.
59. Wall C, Glenn S, Mitchinson S, Poole H. Using a reflective diary to develop bracketing skills during a phenomenological investigation. *Nurse Res*. 2004; 11: 20-29.
60. Butler D. *The sensitive nervous system*. Adelaide: NoiGroup Publications; 2000.

61. Yeung E, Jones M, Hall B. The response to the slump test in a group of female patients. *Aust Physiother.* 1997; 43: 245-252.
62. Huskisson EC. Measurement of pain. *Lancet.* 1974; 2: 1127-1131.
63. Kahl C, Cleland JA. Visual analogue scale, numeric pain rating scale and the McGill Pain Questionnaire: An overview of psychometric properties. *Phys Ther Rev.* 2005; 10: 123-128.
64. Carlsson AM. Assessment of chronic pain. Aspects of the reliability and validity of the visual analogue scale. *Pain.* 1983; 1: 87-101.
65. Boobstra AM, Schiphorst Preuper HR, Reneman MF, Posthumus JB, Stewart RE. Reliability and validity of the visual analogue scale for disability in patients with chronic musculoskeletal pain. *Int J Rehabil Res.* 2008; 31: 165-169.
66. Nijs J, Torres-Cueco R, van Wilgen P, Girbes E, Struyf F, Roussel N, et al. Applying modern pain neuroscience in clinical practice: Criteria for the classification of central sensitization pain. *Pain Physician.* 2014; 17:447-457.
67. Soderlund A, Lindberg P. Long-term functional and psychological problems in whiplash associated disorders. *Int J Rehabil Res.* 1999; 22: 77-84.
68. Ong BN, Coady DA. Qualitative Research: Its relevance and use in musculo-skeletal medicine. *Topic Rev.* 2006; 9: 1-8.
69. Costigan M, Scholz J, Woolf CJ. Neuropathic pain: A maladaptive response of the nervous system to damage. *Annu Rev Neurosci.* 2009; 32: 1-32.
70. Olausson H, Wessberg J, Morrison I, McGlone F, Vallbo A. The neurophysiology of the unmyelinated tactile afferents. *Neurosci Biobehav Res.* 2010; 34: 185-191.
71. Eliav E, Herzberg U, Ruda M, Bennett G. Neuropathic pain from an experimental neuritis of the rat sciatic nerve. *Pain.* 1999; 83: 169-182.
72. Khalsa P. Biomechanics of musculoskeletal pain: Dynamics of the neuromatrix. *J Electromyogr Kinesiol.* 2004; 14: 109-120.
73. Alford L. Psychoneuroimmunology for physiotherapists. *Physiotherapy.* 2006; 92: 187–191.
74. Treede RD, Jensen T, Campbell JN, Cruccu G, Dostrovsky JO, Griffin JW, et al. Neuropathic pain – Re-definition and a grading system for clinical research purposes. *Neurol.* 2008; 29: 1630-1635.
75. Berry G, Svarovska B. Report on a membership audit of the Association of Chartered Physiotherapists in Reflex Therapy (ACPIRT). *Complement Ther Clin Pract.* 2014; 20: 172-177.
76. Kindy K, Keating D. A handy, and deadly, combination of medications. *The Washington Post*; Thursday September 1, 2016.



Enjoy *OBM Integrative and Complementary Medicine* by:

1. [Submitting a manuscript](#)
2. [Joining in volunteer reviewer bank](#)
3. [Joining Editorial Board](#)
4. [Guest editing a special issue](#)

For more details, please visit:

<http://www.lidsen.com/journals/icm>