

RAPID RECOVERY FROM ALL SYMPTOMS OF SEVERE ME/CFS BY TREATING PROPRIOCEPTION DYSFUNCTION SYNDROME: A CASE STUDY

ORLANDO ALVES DA SILVA

ABSTRACT. *Background.* Fatigue with no apparent organic cause is one of the characterising symptoms of ME/CFS, and fatigue is one of many possible symptoms of Proprioception Dysfunction Syndrome (PDS). The aim of this study was to investigate whether a patient with ME/CFS also had PDS, and to treat the patient following the non-invasive and inexpensive protocol for PDS from [13]. *Methods.* A patient with severe ME/CFS was diagnosed with PDS using simple biomechanical tests that investigate body and visual-spatial perception. The key elements of the treatment involve the use of glasses with ‘Active Prism’ lenses, combined with postural reprogramming. We subsequently followed four further patients with the characterising symptoms of ME/CFS through diagnosis and treatment for PDS. *Results.* The results show a rapid recovery of the clinical state of the primary patient during the course of the treatment as measured on the Bell Fatigue Scale, allowing him to return quickly to professional life and to re-establish his pre-disease quality of life. Of the four additional patients, one regained pre-disease levels of function, while the clinical state of the remaining three patients improved measurably. *Conclusions.* Research into the prevalence of PDS among ME/CFS patients should be carried out, and those ME/CFS patients with PDS should undertake the treatment that we describe (beginning with postural reprogramming, see Appendix B).

1. BACKGROUND

1.1. **ME/CFS.** Myalgic Encephalomyelitis / Chronic Fatigue Syndrome (ME/CFS) is a debilitating illness of unknown aetiology that is characterised by often profound fatigue and impairment that lasts for at least six months, by post-exertional malaise, by unrefreshing sleep and by at least one of either cognitive impairment or orthostatic intolerance [17]. Additional symptoms are common, including disequilibrium and problems with balance, muscle pain, gastro-intestinal problems and flu-like feelings suggesting abnormalities in immune system function [4]. Symptoms can persist for years, and most patients never regain their pre-disease level of health [17]. The World Health Organization classifies ‘Postviral fatigue syndrome’, which incorporates both myalgic encephalomyelitis and chronic fatigue syndrome, as a disorder of the nervous system [18].

There is no definitive test for ME/CFS, there is no known cure, and the efficacy of therapies to manage symptoms is not well understood [17]. The diagnosis is one of exclusion, where patients who present with all of the characterising symptoms listed above are said to have ME/CFS if no other underlying physiological or psychological cause can be identified. In particular, diagnosis can be made no sooner than six months from the onset of symptoms. Even for those diagnosed with ME/CFS, there is an ongoing debate among stakeholders about how best to manage fatigue in patients; for example, the National Institute for Health and Care Excellence (NICE) in the U.K. is currently undertaking a major review of its guidance on the diagnosis and treatment of ME/CFS [16].

1.2. **The proprioceptive system and PDS.** Proprioception is the sense that allows us to know our body position, movement and spatial orientation, including the relative position of different parts of our body. The proprioceptive system, first considered by Sherrington [24, 25] is a global physiological system that provides us with our sense of proprioception. The afferent sensory receptors that contribute to the sense of proprioception are diverse and are located throughout the body. Such receptors include: muscle spindles; receptors in the fascia; the Golgi tendon organ; joints; the skin; the mouth and mucous membrane; and palisade endings in the extraocular muscles [8, 10, 19].

As with any system in the body, the proprioceptive system can malfunction. *Proprioception Dysfunction Syndrome* (PDS) is a set of signs and symptoms arising from unconscious shifts from the ideal biomechanics of the human body [11]. Introduced originally in studies by Martins da Cunha that continued in subsequent joint work with the first author [13, 15], PDS was initially known as Postural Deficiency Syndrome due to the postural signs of the patients, and the working hypothesis was that the aetiology was postural. Indeed, patients presented with both stereotypical, asymmetric body position and an incorrect perception of body position: for those with predominant left foot support, the head, trunk and right foot were rotated clockwise, the left hand was rotated internally, and the direction of sight was oriented predominantly to the left; those with predominant right foot support were a mirror image of this. However, while postural signs are always present in patients with PDS, later studies indicated that PDS is a dysfunction of the proprioceptive system [3, 12, 14]. A patient with PDS

can present with a wide range of symptoms relevant to our case study, including but not limited to: pain of the back, neck, chest or limbs, as well as migraines; a lack of balance, including vertigo, dizziness or body disequilibrium; cognitive dysfunction, including lack of attention and hypersensitivity; and fatigue, including difficulties in sleeping.

The symptom of PDS that is most relevant to the present paper is fatigue. When an individual has a proprioceptive dysfunction, the brain perceives incorrectly the tonus of muscle fibres throughout the body and, as a result, many muscle fibres may become and remain hypertonic even at rest. In particular, hypertonicity in both muscles of an antagonistic muscle pair may result in no movement, but would nevertheless require an ongoing supply of energy. One potential consequence of this is profound, long-lasting fatigue that is not substantially alleviated by rest, because rest alone may not correct the perception of the tonus of muscular fibres throughout the body nor the resulting hypertonicity. The clinical experience of the first author shows that when the dysfunction of the proprioceptive system can be addressed, the brain starts to perceive correctly the tonicity of the muscle fibres, and the brain sends a new signal for the hypertonic muscle fibres to relax. As a result, the unexplained fatigue may disappear.

1.3. Extraocular muscles and Active Prisms. We learn by experimenting as children that vision plays an important role in maintaining postural stability. More subtle, but nevertheless much studied in the literature [27, 26, 9, 21], is the impact on the proprioceptive system of information obtained through extraocular muscles.

The important work of Roll et al. [20, 21] demonstrated in a lab setting that a proprioceptive relationship exists between skeletal muscles and extraocular muscles in humans. More precisely, the experiments produced the same illusory motion effect of an image by stimulating the anterior tibial muscle as by stimulating the inferior rectus muscle; a similar experiment achieved the same illusory effect by stimulating the Achilles tendon as by stimulating the superior rectus muscle and the intermediary muscles at the neck (including the trapezius and splenic). It was subsequently shown by Bérard and Silva [7] that tonic relaxation of selected extraocular muscles in turn relaxes all skeletal muscles of the same muscle family. For example, the tonic relaxation of an external rotation muscle, such as the inferior oblique, induces tonic relaxation of all the external rotators that are physiologically related to the inferior oblique by the proprioception system, including the external rotators of the contralateral lower limb. These links between the eye and the muscles in the leg demonstrated experimentally that the proprioceptive system is a global system in the body.

Active Prisms are low-powered prism lenses developed by the first author in collaboration with Martins da Cunha in the course of their work on PDS [15]. It has been known since Baron's work on fish [5] that altering the extraocular muscles can induce a response from the proprioception system when the degree of deviation is less than or equal to four degrees. In the case of humans, prism lenses of more than four diopters, including classical prisms used to compensate for ocular deviation, do not induce a response from the proprioception system. By contrast, prism lenses of less than or equal to four diopters can relax selected extraocular muscles when aligned correctly, and hence relax the skeletal muscles of the same muscle family; this leads in turn to a normalisation of the proprioceptive system. The clinical evidence of the first author shows that PDS results from a dominance of the external rotator muscles, and one can induce a positive clinical reaction in patients with PDS by aligning Active Prism lenses to relax either the inferior oblique muscles or the external rectii (the superior oblique and the superior rectus muscles are internal rotators, so one should not position prism lenses to relax those muscles when treating PDS). Note also that the prism lenses must be of different power, because PDS corresponds to body asymmetry, be it lateral, rotatory or anteroposterior. A table listing the possible outcomes with appropriate prism refraction in each case can be found at the first author's website [1].

Whatever the list of symptoms suffered by a patient, there is a single diagnostic protocol and a single treatment protocol for patients with PDS because all symptoms derive from a dysfunction of the proprioceptive system [14]. Given the overlap between symptoms of ME/CFS and PDS, the initial aim of this study was to investigate whether a patient who had been previously diagnosed with ME/CFS also had PDS, and, if so, to treat and record the outcome of treatment following the non-invasive, pain-free and inexpensive protocol for PDS developed by Martins da Cunha and Alves da Silva [3, 12, 13, 14]. We later expanded the case study to include several additional patients with the characterising symptoms of ME/CFS.

2. METHODS

Each patient in this case study underwent a diagnosis for PDS (see section 2.1), and their rating on the Bell Fatigue Scale (see Appendix A) was recorded for the three months preceding diagnosis. The treatment programme (see section 2.2 and Appendix B) was explained, and communication was maintained with each patient throughout the four month study. The rating of each patient on the Bell Fatigue Scale was recorded one month after diagnosis, and again four months later.

¹Alternatively, the clinician can perform a diagnosis using the directional scotometry technique on the Clemon Clarke Synotophore as described in [14].

2.1. Performing the diagnosis. To diagnose a patient with PDS, the clinician initially carries out two simple tests¹ as follows:

- (1) *Head extension.* The patient is asked to stand upright (unless the patient cannot stand) and, while keeping their shoulders still, is asked to tilt their head backwards to look up to the ceiling. The indicator for PDS is to observe whether the neck extension muscles (including the trapezius and intervertebral muscles of the posterior chain) on both sides present similar levels of contraction while maintaining full neck extension. This can be recognised² by measuring the distance between the shoulder and the ear lobe on a vertical line. The clinician places their palms flat along either side of the patient's neck with the first articulation of the little finger of each hand touching the top horizontal edge of the trapezius muscle. The distance from the clinician's thumbs to each earlobe is observed and provides the information on the symmetry of the head in full extension.
- (2) *Head rotation.* The patient stands upright (unless the patient cannot stand) and, while keeping their shoulders still, turns their head slowly to one side as far as possible, then slowly turns the head around to the other side as far as possible. The clinician assesses whether the patient presents with right-left asymmetry in the amplitude of head rotation; such an asymmetry is one of the signs of PDS. This test highlights the relative tonic state of the extensor muscles of the head on each side of the body.

If the patient demonstrates asymmetry on both tests (1) and (2), then the experienced clinician can select appropriate prism lenses. The type of PDS and the corresponding Active Prism lenses to be used follows a well-established protocol (see [1]). In more than 90% of cases diagnosed by the first author, the side of the neck that is shorter under test (1) coincides with the side where head rotation is hardest under test (2). In such cases, the prisms should be positioned at upper temporal base (125 OD 55 OE) and the prism strength is selected as follows: the eye on the side of the body where contraction is shorter and harder in tests (1) and (2) receives the stronger prism (either 2.5 or 3 diopter, depending on the result of the Maddox test), while the other eye receives a 2 diopter lens. See Appendix C for the Maddox test and information on additional tests.

Before a diagnosis of PDS can be made, the appropriate pair of glasses with Active Prism lenses is selected for the patient, the glasses are adjusted so that the lenses are aligned precisely for the patient's eye-position, and the above tests are repeated. If, on repeating the head extension test with the selected glasses, the patient is able to maintain a level

head while tilting their head back, and if on repeating the head rotation test with the selected glasses, the patient is able to rotate their head equally far to both sides, then the clinician is able to diagnose the patient with PDS. Note that both tests must be repeated in this way before a diagnosis can be made.

2.2. The treatment protocol. The treatment protocol involves three main corrective components:

- (1) *Provision of new visual input.* Following diagnosis, the glasses with appropriate Active Prism lenses are prescribed, with added refraction as required. The frames of the glasses must be aligned carefully, and the glasses must be worn all day long.
- (2) *Provision of new plantar input.* Special insoles containing a reflective material insert comprising two polarised films should be worn all day long in direct contact with the skin. These films reflect the infrared radiation emitted by the foot in order to stimulate the plantar skin to produce a reflex movement of the toes, and especially of the big toe.
- (3) *Provision of new musculoskeletal input.* The postural reprogramming protocol of Martins da Cunha should be introduced. There are several aspects: postural correction; diaphragmatic breathing; ergonomics; and patients are required to sleep only a thin foam mattress on a wooden board. See Appendix B for details.

The elements of the treatment programme are not simply a collection of local mechanical modifications. Rather, by consciously repositioning body segments globally into what is considered to be the ideal biomechanics, the patient is providing new proprioceptive input for their body. Clinical experience shows that this protocol contributes to improving the function of the proprioceptive system. Lack of compliance with any aspect of the treatment may lead to failure of the treatment.

3. RESULTS

Initially, only one patient who had been previously diagnosed with ME/CFS was considered, and he was diagnosed with PDS. The rapid recovery of this patient, referred to as Patient 1 below, from all symptoms of ME/CFS while undergoing the treatment programme described above led us to test for possible PDS in five other patients with chronic fatigue. One of these additional patients had a nickel allergy and had to drop out of the study following an allergic reaction to the metal frames of the glasses, so only four additional patients were treated for PDS. No other patients were studied, i.e. we describe below the outcome of the diagnosis and treatment for PDS of *all* patients in the case study.

²This process requires clinical training, because such patients normally present with a rotation of the trunk that can lead to an incorrect diagnosis.

3.1. Case report: Patient 1. Patient 1 is a 44 year old male. He fell ill in October 2014 after a long period of badly disrupted sleep and persistent stress, and was largely housebound over the next six months. In January 2015 he was referred to a fatigue management clinic run by the National Health Service (NHS), where he was instructed briefly in meditation techniques by a psychologist; he exhibited no signs of mental illness or depression. Following many blood tests (one of which indicated that he had the Epstein Barr virus), he was formally diagnosed with ME/CFS in April 2015. At that stage, his treatment by the NHS ended.

By that time he had already introduced many self-management strategies to relieve symptoms and assist recovery. He adopted various dietary restrictions (avoid alcohol, caffeine, dairy and gluten); took a range of nutritional supplements (including Acetyl-L-Carnitine, D-ribose, Vitamins B, C and D, and Ubiquinol); took regular magnesium salt baths; practised daily meditation and, when strong enough, some light yoga. By December 2015 he had made a near-complete recovery, but it wasn't until June 2016 that he was able to travel with confidence and work without restriction. However, at no point did he ever regain his pre-disease level of energy or function, especially when multi-tasking; in particular, he was unable to look after both of his children simultaneously, even during this period of recovery.

He suffered a relapse in June 2017 following a demanding period at work; doctors ascribed this relapse to a virus. Despite maintaining all of his self-management strategies, the symptoms were worse this time around: the fatigue was more severe, and he suffered from thigh pain (typically but not exclusively on the left side), repeated and prolonged nausea, erratic temperature regulation in his feet, reduced ability to concentrate and hypersensitivity to more than one stimulus. His mobility deteriorated to the point where he required a wheelchair to move around at home. His doctor was unable to provide any support after his diagnosis in 2015, and he had to quit the telephone treatment programme run by a private fatigue clinic after consecutive sessions each led to a prolonged period of additional pain and fatigue. The situation deteriorated further in February 2018, after which he was unable to stand, sit, hold a conversation with one person for more than a few moments, or watch TV without suffering increased pain and heightened fatigue lasting a week or more. He was nevertheless able to crawl to the bathroom once each day. Throughout the period June 2017-April 2018, he was cared for by his wife, and he tried to assist his own recovery by meditating for at least an hour each day (normally 1 hour 45 minutes) while maintaining a positive outlook.

On 24th April 2018, Patient 1 was diagnosed with PDS and was prescribed Active Prisms: 2 at 125 OD and 3 at 55 OE (upper temporal base). He was very reluctant to stand

during the demonstration of the treatment programme, but nevertheless, while wearing the glasses for the first time, he was lifted from bed and given help to stand against a wall. While he did not feel any immediate improvement, neither did he suffer additional pain or fatigue the following day. He later acknowledged that this was surprising, especially given that he, and to a greater extent his wife, interacted with the doctor and a physiotherapist for more than an hour over two sessions during the diagnosis and explanation of the treatment programme.

Despite initial scepticism, he began the treatment programme described above and adhered very strictly to the protocol. In addition to wearing the glasses, he was given the insoles upon diagnosis, he received the bed boards and thin foam mattress three days later and was given shoes with fitted reflective insoles nine days later. Relief from all symptoms of ME/CFS occurred rapidly:

- after 2 days he could sit in a chair for short periods (for the first time in 3 months);
- after 4 days he had a night of refreshing sleep (for the first time in 5 months) and was able to interact with his children for short periods (for the first time in 3 months);
- after 6 days he ate all meals at a table and began to take steps around his bedroom (for the first time in 3 months);
- after 8 days he could walk around the house, including up and down stairs, and he ate two meals with his family (for the first time in 6 months);
- after 10 days he could walk outside for half a mile (for the first time in 6 months) and ate all meals with the family (for the first time in 3 months);
- after 12 days he had lunch in a restaurant, walked for a mile and managed to cope while both of his children were shouting to get his attention (for the first time in 3.5 years);
- after 14 days he woke early to practice yoga, and enjoyed a full, active day (for the first time in 11 months).

Patient 1 was back at work full time within one month of beginning the treatment. At no point during the first week after being diagnosed with PDS did Patient 1 accept that the treatment programme could be the catalyst for the improvements to his health. While this alone does not rule out the placebo effect, we can say that the rapid improvement in his symptoms did not simply result from his belief that the treatment would work.

In the final week of June 2018, Patient 1 felt the return of minor fatigue. He was retested for PDS and an alternative choice of (weaker) prism lenses was prescribed, namely 2.5 at 125 OD and 2 at 55 OE (upper temporal base). This gave immediate relief from fatigue, and the improvements to his overall well-being and gut health were such that he set aside completely the dietary restrictions that he had maintained

for several years. Again, minor fatigue returned in late July 2018, but Patient 1 was nevertheless able to work full time and go on a holiday abroad. He stopped wearing the glasses with prism lenses in mid-August and subsequently noticed a brief respite from his fatigue, but this lasted only a week. Finally, he was retested for PDS at the end of September 2018, and he required a third prism prescription, namely 2 at 125 OD and 2.5 at 55 OE (upper temporal base). This time the recovery from fatigue was immediate, robust and long-lasting: at the time of writing, more than 11 months after his diagnosis with PDS, Patient 1 continues to be free of all symptoms of ME/CFS while he maintains the lifestyle changes described in section 2.2 above. Adopting a poor posture, e.g. while playing games on the floor with his children, is enough to bring back symptoms of minor fatigue for 24 hours, so he continues to adhere strictly to the protocol.

Patient 1 Fatigue Rating: At the time of diagnosis with PDS and in the preceding months, the rating of Patient 1 on the Bell fatigue scale (see Appendix A) was 0, and one month later it was 70 (he had returned to full-time work but was resting frequently as a precaution, so the phrase ‘working full time with difficulty’ is appropriate). As the second month since his PDS diagnosis began, his rating was 100 and, setting aside both periods when Patient 1 was wearing glasses with the wrong prismatic prescription, he has remained at 100 on the Bell fatigue scale ever since.

Additional remarks on the clinical history of Patient 1: After the link between PDS and the symptoms of ME/CFS suffered by Patient 1 was established, several additional aspects of his clinical history prior to 2014 became relevant to his case. He had repeated ankle ligament problems in his late teens and early twenties, he suffered severe achilles tendonitis for two years in his mid-thirties (resolved only with orthopaedic insoles), and he suffered from a painful neck with restricted movement of his left trapezius muscle throughout his early forties. In addition, Patient 1’s right foot has pointed roughly 20° to the right for more than twenty years, and he was under the impression that his right leg was longer than his left leg (he was of predominant left foot support, see section 1.2). In fact, his foot placement and leg-length imbalance were both resolved during the course of the treatment for PDS (the apparent leg-length imbalance was therefore the result of involuntary, prolonged muscle contraction), and he no longer wears orthopaedic insoles. Patient 1 also noted that immediately prior to falling ill originally in October 2014, and again before his relapse in June 2017, he was putting considerable strain on his body through deadlifting and similar gym exercises.

3.2. Case report: Patient 2. Patient 2 is a 43 year old female. She began to suffer from fatigue at the age of 16 and has struggled ever since: she was found to have the Epstein

Barr virus while at University; she was hospitalised with exhaustion and possible viral infection aged 26; and she was diagnosed with ‘third stage adrenal fatigue’ at the age of 40 by clinicians at a private medical clinic.

While fatigue, post-exertional malaise, unrefreshing sleep and shoulder tension have been persistent symptoms of Patient 2 for more than two decades, these symptoms intensified in early 2016 and in addition, she suffered with noticeable cognitive impairment, an ever-present dull headache, the inability to run for more than two minutes at a time, muscle pain, restricted movement, stiffness and inflexibility. By this stage, Patient 2 had all the symptoms of moderate ME/CFS, and a further deterioration occurred in early 2018, with increased shoulder tension and more serious headaches, together with occasional paresthesia in fingers when sleeping. Throughout this period, she maintained a healthy lifestyle to facilitate recovery: adopting a restricted organic diet; nutritional supplements, daily walks, weekly pilates, regular meditation and magnesium salt baths, ion cleanse foot spa protocol, periods of sports massage, acupuncture and sauna use. In addition, she maintained a fairly low profile socially, minimising commitments where possible.

On 20th June 2018, Patient 2 was diagnosed with PDS and was prescribed Active Prisms: 3 at 125 OD and 2 at 55 OE (upper temporal base). She received the insoles within 7 days, and she started wearing glasses with prism lenses on 4th July 2018. Initially, allergies prevented her from finding a suitable thin mattress, so she slept on the floor. In the weeks that followed, Patient 2 underwent a partial relief from the symptoms of ME/CFS:

- after 2 days she woke feeling refreshed and without a headache after sleeping on the floor (for the first time in 1.5 years);
- after 4 days she was able to sustain longer and more active days without suffering post-exertional malaise (for the first time in 1.5 years);
- after 6 days she slept on a soft mattress and suffered general muscle pain, feeling unrefreshed on waking for the first time in several days. Nevertheless, comments she received from others suggested that her treatment was having a noticeable effect on her posture;
- after 8 days she was once again sleeping significantly better having returned to sleeping on the floor, and waking with more energy;
- after 10 days she had no widespread muscle soreness after pilates (for the first time in 2 years), though she was still unable to run for more than a couple of minutes.
- after 14 days she felt energised and had no brain fog (for the first time in 1.5 years), generally achieving much more each day than before the treatment.

Within one month of beginning the treatment, the symptoms of ME/CFS for Patient 2 were less severe across the board: she was achieving more each day and nevertheless feeling less fatigued than before; her post-exertional malaise was receding, noticeably so after pilates classes; on most days she felt refreshed after waking; and her cognitive impairment had decreased.

By early November 2018, four months after being diagnosed with PDS, Patient 2 had enjoyed continued incremental relief from the characterising symptoms of ME/CFS. Perhaps the most significant improvement in her quality of life came from a decrease in the level of pain she was suffering: there was a noticeable relief from muscle soreness, and from the headache that she had struggled with for the better part of two years. She had also started running periodically for up to 30 minutes.

Patient 2 Fatigue Rating: At the time of diagnosis with PDS and in the preceding months, the rating of Patient 2 on the Bell fatigue scale was 50, after one month it was 70-80, and four months after diagnosis it had climbed to 90.

Additional remarks on background of Patient 2: In light of the improvements to her health after beginning the treatment for PDS, Patient 2 noted that in 2015-16 (roughly when her health deteriorated markedly), she had replaced the very firm mattress on her bed by a softer mattress, and she had also replaced her supportive sofa with softer seating. Patient 2 felt that the change to sleeping on a thin mattress on wooden boards had been a crucial element in her recovery.

3.3. Case report: Patient 3. Patient 3 is a 47 year old female. She began to suffer from fatigue at the age of 24, and within a year was diagnosed as having ME/CFS by clinicians at Havering Hospital in London. She was subsequently referred to a behavioural therapist at a specialist ME/CFS service run in King's College London, and she underwent cognitive behavioural therapy at the same time as working towards a degree in psychology. From the age of 30, she no longer had contact with clinicians about her symptoms of ME/CFS. Throughout this period and in the years that followed, she was able to function relatively well while working as a clinical psychologist if she managed to lie down to rest in a quiet room twice a day.

In January 2014, her son fell severely ill and required a great deal of care. This took a toll on the health of Patient 3 and her symptoms of ME/CFS worsened, to the extent that by September 2016 she was largely house-bound, and fatigue limited greatly her ability to function, especially post-exertion. In addition to the characterising symptoms of ME/CFS, she suffered from back pain and, from the start of 2017, a frozen left shoulder. In September 2017 she sought

advice from her doctor, and was referred to a fatigue management clinic run by the NHS. From March 2018, she had to deal with significant, additional stress which led to a further deterioration in her health.

On 21st June 2018, Patient 3 was diagnosed with PDS and was prescribed Active Prisms: 2 at 125 OD and 3 at 55 OE (upper temporal base). During the course of the diagnosis, and while wearing her glasses with Active Prism lenses, her frozen shoulder relented and she was able to freely raise her arms above her head for the first time in 18 months. She received immediately the glasses with the prism lenses, and received wooden boards for her bed and insoles within a week. She also purchased a suitable mattress at the same time. The glasses with prism lenses did not have any vision refraction, so Patient 3 simultaneously wore varifocal contact lenses and used a magnifying glass for reading.

After initial improvements in her energy levels upon wearing the glasses, she began to struggle again within ten days. She was retested and an alternative choice of prism lenses was prescribed, namely 2.5 at 125 OD and 2 at 55 OE (upper temporal base). By the end of the first week, she reported a noticeable increase in her energy levels and a lessening of pain in her back. Within a month she was no longer housebound and estimated that her overall health had improved by 15%; indeed, she began to use her electric bicycle daily for up to 15 minutes. She continued to maintain all aspects of the treatment programme, but at this stage she became worried that her glasses might be misaligned, and in the months that followed she did not see any further improvements to her health. Four months after diagnosis with PDS, she complained of headaches and was recommended to stop wearing the glasses with Active Prisms.

Patient 3 Fatigue Rating: At the time of diagnosis with PDS and in the preceding months, the rating of Patient 3 on the Bell fatigue scale was 30, after one month it was 40, and her rating has remained at 40 ever since³.

3.4. Case report: Patient 4. Patient 4 is a 60 year old female. In December 2013 she caught the H1N1 virus. She subsequently caught pneumonia and was diagnosed with type I and type II respiratory failure, and was put in a medically-induced coma for over a week. In the years that followed, Patient 4 struggled with weakness and fatigue, post-exertional malaise, unrefreshing sleep, sciatica in her left hip, headaches, pain in her right leg, and she had minor problems with brain fog. She was finally diagnosed with ME/CFS in July 2016, and was referred to a fatigue management clinic run by the NHS where she attended a regular 'Help group'. She continued to work only rarely for short

³Six months after the conclusion of this case study, Patient 3 was retested for PDS and required a third prism prescription: 2 at 125 OD and 2.5 at 55 OE (upper temporal base). The pattern of prism changes required by Patient 3 follows that of Patient 1.

periods, and required at least a day of bed rest after any organised activity out of the house.

On 25th June 2018, Patient 4 was diagnosed with PDS and was prescribed Active Prisms: 3 at 125 OD and 2 at 55 OE (upper temporal base). On 6th July 2018 she received the glasses, the insoles, a desk slope to aid writing, and foot blocks. Against advice, she chose not to replace her soft mattress for fear of aggravating her long-standing sciatica, but she did follow the postural reprogramming (though her sciatica allowed her to perform the diaphragmatic breathing exercises only once or twice per week). There was an additional complication in that she required reading glasses, and yet the Active Prism lenses that she was given did not have any vision prescription. This meant that she had to remove the glasses with prism lenses more frequently than is recommended.

Despite this, Patient 4 initially made excellent progress. Within the first month, she had noted a considerable improvement in all of her symptoms. The increase in energy levels and the reduction in post-exertional malaise were notable: during the final week of July 2018, she was on holiday abroad and did not require a single day in bed to recover from her increased activity, in marked contrast to her situation prior to beginning the PDS treatment programme. Unfortunately, she suffered from a severe sciatica flare in mid-August 2018, and this became a limiting factor in her recovery in the months that followed. Indeed, by November 2018, she described the major reason for her disability as being sciatica. At this time, she continued to experience a reduction in her post-exertional malaise and unrefreshing sleep when compared to the period prior to her diagnosis with PDS, and she experienced fewer headaches and less pain in the right side of her body.

Patient 4 Fatigue Rating: At the time of diagnosis with PDS and in the preceding months, the rating of Patient 4 on the Bell fatigue scale was 20, after one month it was 60-70, but it had dropped to 50 after four months following a sciatica flare. Since Patient 4 chose not to replace her soft mattress by a firm mattress on wooden boards, a complete recovery was not expected in her case.

3.5. Case report: Patient 5. Patient 5 is a 22 year old transgender male, currently undergoing gender reassignment. In September 2017 he began to suffer from severe pain in the back and legs (including sharp, stabbing pains), reduced mobility, plus fatigue despite no prior exertion. The pain frequently left him bed-bound and sensitive to both light and sound. He often became exhausted, having to rest multiple times a day and sleeping for more than 12 hours at night, yet waking unrefreshed; at other times he had difficulty falling asleep. His other symptoms included post-exertional malaise, gastro-intestinal problems, a heightened

sensitivity to cold, and cognitive dysfunction including poor concentration, hypersensitivity to multiple stimuli and anxiety.

Prior to suffering from these symptoms, Patient 5 had been very active. He had competed internationally in elite level triathlon for five years as a teenager, so his inability to walk far, and at times his inability to stand at all, was a dramatic change in circumstances. Multiple visits to his doctor in late 2017 led to blood tests and an MRI scan of his back that detected no organic cause for the sudden onset of health problems. He was prescribed painkillers (daily dosage: 4 × 50mg Tramadol and 3 × 250mg Naproxen) in February 2018. By March 2018, he satisfied all of the characterising symptoms of ME/CFS, though he received no formal diagnosis.

On 6th July 2018, Patient 5 was diagnosed with PDS and was prescribed Active Prisms: 3 at 125 OD and 2 at 55 OE (upper temporal base). He received immediately the glasses with Active Prism lenses. He did not receive insoles, but he did replace his mattress by a thin mattress on a wooden board a few days later. Within the first week, he saw some improvement to his endurance levels, and a reduction in muscle pain and post-exertional malaise. Within one month of diagnosis, Patient 5 completed a march at a Pride event and spent the evening dancing at the after party without having to take breaks, and he no longer suffered from post-exertional malaise. At this stage he tried to reduce his intake of painkillers, but his pain levels became unbearable very quickly so he continued with the same dosage (daily dosage: 4 × 50mg Tramadol and 3 × 250mg Naproxen).

At the four month follow-up, Patient 5 had enjoyed relief from fatigue and post-exertional malaise, with only a little more muscle soreness than he would expect. He no longer experienced unrefreshing sleep, and his cognitive impairment had improved as his pain levels and his pain medication dosage had decreased (daily dosage: 1 × 50mg Tramadol and 1 × 250mg Naproxen).

Patient 5 Fatigue Rating: At the time of diagnosis with PDS and in the preceding months, the rating of Patient 5 on the Bell fatigue scale depended on the level of pain-killers he was taking: it was 30 without medication, rising to 50 with medication (daily dosage: 4 × 50mg Tramadol and 3 × 250mg Naproxen) After one month, his rating was 80 with the same level of medication, and after four months, his rating was 100 'most of the time' with reduced medication (daily dosage: 1 × 50mg Tramadol and 1 × 250mg Naproxen). Given that Patient 5 was still taking painkillers after four months, we also followed-up after six months and at that time his fatigue rating was 100 *without* painkillers.

Additional remarks on background of Patient 5: He has an accessory navicular in both feet, his left leg is one inch longer than his right (he wears orthotic insoles to correct for

this), he has an extra floating rib and half an extra vertebra in his spine. In his late teens, a psychologist's assessment suggested that Patient 5 has ADHD, though he was never formally diagnosed with this condition. He suffered from severe bulimia for nine years, but he had recovered fully from this by early 2018.

3.6. Numerical data for all five patients. For convenience we plot in Figure 1 the numerical data measuring the disability experienced by each patient as measured on the Bell fatigue scale (see Appendix A) in the months leading up to diagnosis with PDS, at the time of diagnosis with PDS, and in the months that followed.

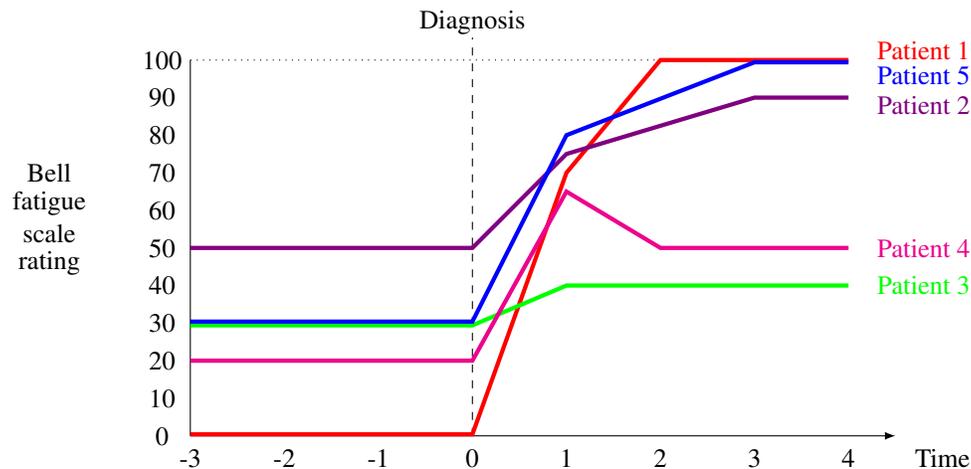


FIGURE 1. Bell fatigue scale rating plotted against time in months. Notes: (i) Patient 3 was retested for PDS after the conclusion of this case study and at that time she required a new prism prescription; (ii) Patient 4 chose not to follow all aspects of the treatment protocol, so a complete recovery was not expected in her case; (iii) Patient 5 no longer required painkillers (and still rated as 100 on this scale) six months after diagnosis.

4. DISCUSSION

In this case study, all five patients suffered from the characterising symptoms of ME/CFS, all five were diagnosed with PDS, and all five patients saw improvements to their symptoms of ME/CFS during their subsequent treatment for PDS. Two of these patients, namely Patients 1 and 5, made a complete recovery from the characterising symptoms of ME/CFS, Patients 2 and 4 made significant strides (at least initially) towards a strong recovery, while Patient 3 improved to a lesser degree.

We consider first the cases of Patients 1 and 5. The symptoms of Patient 1 at the time of diagnosis with PDS were severe, while many of the symptoms of Patient 5 were severe, especially the pain. There are a number of similarities between the cases of these patients: both engaged frequently in high-intensity exercise for years before falling ill; both committed to applying rigorously the PDS treatment protocol throughout the course of the case study and beyond; and both made a complete recovery from the symptoms of ME/CFS (at the time of writing, both patients continue to adhere to the PDS treatment protocol). It would be interesting to consider a larger cohort of patients who had been committed to exercise before falling ill with symptoms of

ME/CFS, and to understand the extent to which diagnosis and treatment for PDS could assist their recovery.

Turning attention to Patients 2 and 4, the treatment for PDS led both patients to make a partial recovery from the characterising symptoms of ME/CFS that had a significant, positive effect on their level of function. The level of fatigue and post-exertional malaise that both patients suffered was reduced rapidly during treatment, though it was not eliminated. Both patients also noted that sleep was more refreshing, and that there had been a reduction in the level of pain, including headaches, they were experiencing. This led to a step-change in the quality of life for each patient (though in the latter period of the study, Patient 4 was contending with the disabling effects of sciatica).

Finally, the severity of the symptoms of ME/CFS in Patient 3 decreased during the course of her treatment for PDS to the extent that in the months leading up to July 2018 she was largely confined to her house, whereas after undergoing the treatment for PDS she was no longer housebound; indeed she cycled outside using an electric bicycle regularly at this time. However, following the immediate relief from the frozen shoulder that Patient 3 experienced during the diagnosis for PDS, we had anticipated a stronger recovery in her case. Given that her prism prescription changed during the

first ten days of the case study (clinical experience suggests that this happens in less than 2% of patients with PDS), it is possible that she was not wearing the correct Active Prism lens prescription for much of the case study⁴. It is worth noting that several months after removing the glasses as advised, Patient 3 was keen to repeat the diagnosis for PDS because she had felt more robust and had suffered much less pain while wearing the glasses. We also note that there may also have been a further medical complication that does not result from a dysfunction of the proprioceptive system.

This case study also illustrates through Patient 1 that a patient with ME/CFS who is being treated for PDS may experience a return of fatigue, albeit with much reduced intensity, if the prescription of the Active Prism lenses is not correct. This highlights an inherent aspect of this treatment programme: as the proprioceptive system of a patient with PDS gradually recovers during the course of the treatment, the type of prism they require may change over time. Put simply, each set of prism lenses has a 'sweet spot', and as a patient recovers their prism requirements may move away from the sweet spot of their glasses, leading them to require an alternative lens prescription. It is therefore essential that the patient undergoing treatment for PDS monitors their own symptoms carefully, and that they are tested regularly for PDS. In fact, a patient may suffer from side-effects if they wear the wrong prism prescription or if the correctly prescribed Active Prism lenses are misaligned on the patient's face; this explains why Patient 3 was advised to stop wearing her Active Prism lenses at the four month follow-up. On the other hand, the clinical experience of the first author shows that some patients with PDS who continue to adhere closely to the treatment programme for several years no longer need to wear glasses with Active Prism lenses as long as they continue to follow the other aspects of the programme.

Both ME/CFS and PDS are disorders of the nervous system, but we do not conflate the two. Rather, this case study provides evidence that a patient with PDS can recover at least partially from the characterising symptoms of ME/CFS if they are treated following the protocol for PDS described in Section 2.2, and moreover, for some patients the recovery can be rapid, complete and robust.

We note that PDS is a syndrome of physiological (rather than psychological) basis, and the treatment protocol introduced by Martins da Cunha and the first author is designed to provide the body with the correct proprioceptive input from many different sources. It is striking that this approach not only prevents a further decline in the function of the proprioceptive system, but that it actually corrects the malfunction in the proprioceptive system without surgical intervention.

Some patients undergoing treatment for PDS focus primarily on the glasses, but it is important that patients also embrace all aspects of the treatment protocol including the postural reprogramming. It is well known that some patients with ME/CFS can have postural deficiencies, and some clinicians advocate postural improvements (e.g. the Alexander technique) to assist recovery from ME/CFS. Our study provides a potential explanation as to why postural improvements can assist in a recovery from symptoms of ME/CFS, at least for those patients with ME/CFS who also have PDS. Especially relevant is recent work of Rowe et al. [23, 22] demonstrating that patients with ME/CFS have a greater prevalence of impaired range of motion of the limbs and spine than healthy control patients. It would be interesting to discover how many of the patients with ME/CFS studied by Rowe et al. also have PDS; for those who do, the presence of hypertonic muscles induced by a proprioceptive dysfunction would provide a natural explanation for the impaired range of motion.

To conclude, we emphasise that in this case study, all five of the patients with the characterising symptoms of ME/CFS that we studied also had PDS. It is important now to understand just how prevalent is PDS among patients with ME/CFS in the wider community, because for those ME/CFS patients who also have PDS, the treatment programme described in section 2.2 provides an opportunity to build a physiological recovery from all of the characterising symptoms of ME/CFS. Clearly, a larger study is required to obtain a clearer picture of the prevalence of PDS among patients with ME/CFS.

5. CONCLUSIONS

This case study shows that patients with all of the characterising symptoms of ME/CFS may concurrently have PDS and, moreover, by committing to the treatment programme for PDS, the patient may undergo a rapid recovery from all symptoms of ME/CFS. For patients such as Patients 1 and 5 studied here, the dysfunction of their proprioceptive system appears not to be a consequence of their having ME/CFS, but rather, the underlying cause of their symptoms. The potential impact of this observation stems from the fact that diagnosis of PDS takes no more than a few minutes for the experienced clinician, while treatment is non-invasive, pain-free and very inexpensive. Contrast this with the current position for the treatment of ME/CFS, where the efficacy of therapies available for the management of symptoms is not well understood. Put simply, for some patients, the characterising symptoms of ME/CFS result from a physiological dysfunction that can be diagnosed and treated effectively.

⁴Six months after the conclusion of this case study, Patient 3 was retested for PDS and required a third prism prescription: 2 at 125 OD and 2.5 at 55 OE (upper temporal base). The pattern of prism changes required by Patient 3 follows that of Patient 1.

While these observations, and especially the progress made by Patients 1 and 5, provide cause for optimism among some patients with severe symptoms of ME/CFS, we would like to sound a note of caution. Treatment for PDS is not a panacea: successful treatment requires a collection of lifestyle changes by the patient that must be maintained at all times, and the treatment is unlikely to succeed for patients who do not commit to all aspects of the programme.

At this stage further research into links between ME/CFS and PDS is required so that we can better understand which patients with ME/CFS are more likely to benefit from the treatment that we have described. Nevertheless, this case study suggests that, where possible, the simple tests for PDS should be carried out by a trained clinician before a patient with the symptoms of ME/CFS is forced to wait for six months in order to receive a diagnosis of ME/CFS. In addition, several aspects of the PDS treatment programme, and specifically those listed in Appendix B, could be implemented at little or no cost by anybody with the symptoms of ME/CFS, thereby reducing the strain on their proprioceptive system.

Acknowledgements The author thanks all five patients for giving their consent for their anonymised details to be

presented in this article. He also thanks Alastair Crow, Margarida Dolan, Susan Byrne, Trevor Aldous and Emiko Dupont for providing a great deal of help throughout this case study. Thanks also to Tom Leinster and Sam Miller for comments on an earlier version of this article.

APPENDIX A. THE BELL FATIGUE SCALE

The fatigue level of patients is recorded according to the Bell disability/fatigue scale (0-100). This scale allows for a single, quantitative value on disability that can be compared easily over the passage of time. Note however that there are obvious deficiencies with this scale. First, as even Bell [6] remarks, this scale *'has the potential problem that a patient may be at a score of 50 in one area but a score of 30 in another, and in this case the physician may make the overall determination'*. To offset this deficiency, we record an increase in the Bell Fatigue Scale rating only when *all* statements given for the new, higher rating hold for that patient. Second, the Bell Fatigue Scale is non-linear, in that a jump from 20 to 30 or from 30 to 40 indicates a much greater improvement in health of the patient than a jump from 80 to 90 or 90 to 100.

The Bell Fatigue Scale

-
- | | |
|-----|--|
| 100 | No symptoms at rest; no symptoms with exercise; normal overall activity level; able to work full-time without difficulty. |
| 90 | No symptoms at rest; mild symptoms with activity; normal overall activity level; able to work full-time without difficulty. |
| 80 | Mild symptoms at rest; symptoms worsened by exertion; minimal activity restriction noted for activities requiring exertion only; able to work full-time with difficulty in jobs requiring exertion. |
| 70 | Mild symptoms at rest; some daily activity limitation clearly noted. Overall functioning close to 90% of expected except for activities requiring exertion. Able to work full-time with difficulty. |
| 60 | Mild to moderate symptoms at rest; daily activity limitation clearly noted. Overall functioning 70%-90%. Unable to work full-time in jobs requiring physical labour, but able to work full-time in light activity if hours flexible. |
| 50 | Moderate symptoms at rest. Moderate to severe symptoms with exercise or activity; overall activity level reduced to 70% of expected. Unable to perform strenuous duties, but able to perform light duty or desk work 4-5 hours a day, but requires rest periods. |
| 40 | Moderate symptoms at rest. Moderate to severe symptoms with exercise or activity; overall activity level reduced to 50%-70% of expected. Not confined to house. Unable to perform strenuous duties; able to perform light duty or desk work 3-4 hours a day with rest periods. |
| 30 | Moderate to severe symptoms at rest. Severe symptoms with any exercise; overall activity level reduced to 50% of expected. Usually confined to house. Unable to perform any strenuous tasks. Able to perform desk work 2-3 hours a day, but requires rest periods. |
| 20 | Moderate to severe symptoms at rest. Unable to perform strenuous activity; overall activity 30%-50% of expected. Unable to leave house except rarely; confined to bed most of day; unable to concentrate for more than 1 hour a day. |
| 10 | Severe symptoms at rest; bedridden the majority of the time. No travel outside of the house. Marked cognitive symptoms preventing concentration. |
| 0 | Severe symptoms on a continuous basis; bedridden constantly; unable to care for self. |
-

APPENDIX B. POSTURAL REPROGRAMMING

There are several aspects to the postural reprogramming protocol introduced by Martins da Cunha:

- (1) *Postural correction.* The patient is taught specific positions for lying, sitting, standing and walking, with an emphasis on keeping their hips and shoulders level and maintaining parallel feet at all times.
- (2) *Diaphragmatic breathing.* The patient is taught diaphragmatic breathing techniques, both in a supine position with knees bent up and feet resting on a chair, and in standing position with the back flat against a wall; each to be carried out for 5 minutes a day.
- (3) *Ergonomics.* The patient is taught to improve their ergonomics in several ways:
 - (a) *Suitable footwear* should be worn to maximise the sensory input to the feet from the ground. Such footwear is wide, the sole does not bend easily, and the sole directly beneath each big toe should be in contact with the ground when the patient is standing;
 - (b) A *foot block* should be used when seated to ensure that the body is supported by the feet rather than the back of the chair. The block should be large enough to fully support both feet, and the height of the block is chosen so that the back of the knees are 2cm above the seat; and
 - (c) *Screens* should be placed so that the patient need not turn, raise or lower their head when looking at the screen.
- (4) *Thin foam mattress on a wooden board.* To avoid deformation of the body while lying down, a mattress of low density foam of 3-5cm thickness should be used, and this mattress should lie on a hard wooden board that is thick enough to remain completely flat when supporting the patient (even when placed on the slats of a bed). No pillow should be used, and the patient is taught how to adopt a suitable sleeping position.

APPENDIX C. ADDITIONAL TESTS

During the course of a diagnosis for PDS, additional tests can be performed to assist diagnosis. These tests also serve to convey to the patient that they have a proprioceptive dysfunction. Examples include:

- *Parallel feet.* While the patient looks away, the clinician adjusts the patient's feet so that they are parallel. The patient is then asked to show using their hands in which direction each foot is pointing without the patient looking at their feet. The indicator for PDS is that the patient believes that their feet are pointing inwards.
- *Arm raise.* The patient sits upright and, while maintaining a straight back and straight arms, lifts their arms up

to point towards the ceiling until the biceps are touching the ears. The indicator for PDS is that the patient finds it much easier to perform this test while wearing the Active Prism lenses.

Also, while the Clemont Clarke synoptophore can be used to diagnose the type of Active Prism that is suitable for a patient, the Maddox rod test determines the appropriate prism strength:

- *The Maddox rod test.* This test detects displacement of an image without vertical heterophoria. When a circle of light passes through an optical cylinder, the circle of light is transformed into a line parallel to the base of the cylinder. The clinician performs a Maddox rod test [2] with the optical cylinder aligned vertically in front of one eye and a neutral lens in front of the other; the patient fixates a point source of light and sees a horizontal line with one eye and a disc of light with the second eye. When the patient has a vertical heterophoria, the horizontal line is seen above or below the middle of the circle. The clinical experience of the first author demonstrates that the same phenomenon occurs for patients with PDS. After repeating this test using the appropriate prism lenses, the patient sees the horizontal line of light as passing through the centre of the disc of light. Thus, the Maddox test can be used to detect (by trial and error) the correct prism strength.

REFERENCES

- [1] <https://www.orlandoalvesdasilva.org/protocols-diagnostic-active-prisms>.
- [2] American Academy of Ophthalmology. *The Maddox rod test*, 2019. An excerpt from the 'Basic and Clinical Science Course' from <https://www.aao.org/bcscsnippetdetail.aspx?id=c964238e-4121-4346-90e2-043186bb1869>.
- [3] O. Alves da Silva. Scotometrie directionelle et corrections prismatiques dans le syndrome de deficiencia posturale. *Agressologie*, 28(9):945–946, 1987.
- [4] ME Association. *Symptoms, Testing and Assessment*. See <https://www.meassociation.org.uk/>, 2018.
- [5] J. Baron. Musculature extrinseque et equilibre des poissons. *Comptes rendus hebdomadaires des seances de l'Academie des sciences*, 230(25):2231–2233, 1950.
- [6] D. S. Bell. *CFIDS Disability Scale*, 1994. From <http://www.oiresource.com/cfsscale.htm>.
- [7] P. V. Bérard and A. Silva. Coup d'oeil. *Ophthalmologique*, 10:29–39, 1994.
- [8] R.J. Crum and R. J. Loisele. Oral perception and proprioception: A review of the literature and its significance to prosthodontics. *Journal of Prosthetic Dentistry*, 28(Issue 2):215 – 230.
- [9] A.M. Eber, D. Strubel-Streicher, M. Guillot, and M. Collard. Equilibrium disorders, vertigo and nystagmus induced by maximal deviation of gaze. role of extra-ocular proprioception. *Rev Neurol (Paris)*, 140(2):131–137, 1984.
- [10] K. Lienbacher and A. K. E. Horn. Palisade endings and proprioception in extraocular muscles: a comparison with skeletal muscles. *Biological Cybernetics*, 106(11):643–655, Dec 2012.

- [11] H. Martins da Cunha. Le syndrome de déficience posturale – S.D.P. *Actualités en rééducation fonctionnelle et réadaptation*, 4 série, L Simone Ed. Paris: Mallon, 1979.
- [12] H. Martins da Cunha. Le syndrome de deficiencia postural (SDP). *Agressologie*, 28(9):941–943, 1987.
- [13] H. Martins da Cunha and O. Alves da Silva. Alteracoes da funcao binocular no sindrome de deficiencia postural, 1980. Acta IV Congresso Lusio-Hispano-Brasileiro de Oftalmologia, Vol II.
- [14] H. Martins da Cunha and O. Alves da Silva. Diplopias posturales. *Acta Estrabologica*, ENE-DIC, XIV, 1986.
- [15] H. Martins da Cunha and O. Alves da Silva. Disturbances of binocular function in the postural deficiency syndrome. *Agressologie*, 27(1):63–67, 1986.
- [16] NICE. *Consultation on the diagnosis and management of ME/CFS*, 2017. Details, including project documents, available from <https://www.nice.org.uk/guidance/indevelopment/gid-ng10091>.
- [17] Institute of Medicine. *Beyond Myalgic Encephalomyelitis/Chronic Fatigue Syndrome: Redefining an Illness*. The National Academies Press, Washington, DC, 2015.
- [18] World Health Organization. International classification of diseases, eleventh revision. Available at <https://icd.who.int/browse11/l-m/en#/http://id.who.int/icd/entity/569175314>, 2018.
- [19] U. Proske and S. C. Gandevia. The proprioceptive senses: Their roles in signaling body shape, body position and movement, and muscle force. *Physiological Reviews*, 92(4):1651–1697, 2012.
- [20] J.P. Roll and R. Roll. Eye movements from physiology to cognition. Selected/Edited Proceedings of the Third European Conference on Eye Movements, Dourdan, France, September 1985:57–68, 1987.
- [21] J.P. Roll, J.P. Vedel, and E. Ribot. Alteration of proprioceptive messages induced by tendon vibration in man: a microneurographic study. *Experimental Brain Research*, 76:213–222, 1989.
- [22] P.C. Rowe, C.L. Marden, M.A. Flaherty, S.E. Jasion, E.M. Cranston, K.R. Fontaine, and R.L. Violand. Two-year follow-up of impaired range of motion in chronic fatigue syndrome. *The Journal of pediatrics*, 200:249–253, 2018.
- [23] P.C. Rowe, C.L. Marden, M.A. Flaherty, S.E. Jasion, E.M. Cranston, A.S. Johns, J. Fan, K.R. Fontaine, and R.L. Violand. Impaired range of motion of limbs and spine in chronic fatigue syndrome. *J Pediatr.*, 165:360–6, 2014.
- [24] C. S. Sherrington. The integrative action of the nervous system. *New Haven, CT: Yale University Press*, 1906.
- [25] C. S. Sherrington. On the proprioceptive system, mespecially in its reflex aspect. *Brain*, 29(4):467–482, 1907.
- [26] N. Ushio, M. Hinoki, K. Nakanishi, and J. B. Baron. Role of ocular muscle proprioception in the maintenance of body equilibrium with particular reference to the cervical reflex. *Agressologie*, 21(E):143–52, 1980.
- [27] N. Ushio, K. Matsuura, M. Hinoki, J. B. Baron, and P. M. Gagey. 2 reflex stages in equilibrium provoked by the proprioceptors of the ocular muscles. Analysis using Fukuda’s step test and the orthostatic reflex test. *Agressologie*, 16 Spec No D:39–52, 1975.

POSTURMED, RUA CASTILHO 23- 5B, 1250-067 LISBOA, PORTUGAL.

Email address: o.alvesdasilva@gmail.com

URL: <https://www.orlandoalvesdasilva.org>