Chronic fatigue syndrome: a possible role of mechanical treatment? A case study

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A patient (46 years; female) with a 6 year history of chronic fatigue syndrome was treated with a thoracic rotational movement, which effected an instant and major improvement in symptoms. It is argued that dysautonomia, enhanced by joint hypermobility syndrome, was the major factor behind this patient's condition. It is believed that due to a previous injury and joint hypermobility syndrome, the sympathetic nervous system tissues were traumatised, resulting in dysautonomia. In this paper it is proposed that dysautonomia is a primary presentation of chronic fatigue syndrome (CFS), fibromyalgia syndrome (FMS) and joint hypermobility syndrome (JHS). Because of their similarities it is suggested that there may be a subgroup of CFS and FMS patients who are also hypermobile. It is believed that by finding a directional preference, physiotherapy may be able to influence the autonomic nervous system symptoms associated with these conditions. This would suggest a potential role for physiotherapy in the treatment of these prevalent and intractable conditions, and highlights the importance of further research in this area.

Keywords: chronic fatigue syndrome, joint hypermobility syndrome, fibromyalgia syndrome, dysautonomia

Introduction

Chronic fatigue syndrome (CFS) is a major health problem. The United States Centre for Disease Control and Prevention revised its definition of criteria for CFS in 1994:[1] diagnosis of CFS requires the presence of persistent or relapsing fatigue that is recent or of definite onset and is not improved by rest, over at least a 6 month period. The fatigue must significantly reduce occupational, educational, social and personal levels of activity. Also, during this six month period the person must have suffered from four or more of the following symptoms: impaired short-term memory or concentration severe enough to cause major problems in activity levels in the areas indicated, tender cervical glands, a sore throat, multijoint pains, muscle pain, unrefreshing sleep, a new type of headache, and post-exertional fatigue lasting longer than 24 h. Many patients also experience anorexia, nausea, gastrointestinal problems, dizziness, drenching night sweats, and intolerance to alcohol and some medications which affect the central nervous system (CNS).[1–3]

It is difficult to obtain an accurate estimate of the prevalence of CFS due to inconsistency in the definition used, the type of population surveyed, and the methods used in studies: estimates for the prevalence of current CFS range from 0·007 to 3%.[2] The cause of this syndrome is unknown despite significant research and debate during the past two decades. There is no single test available to diagnose this condition; it is generally believed that the aetiology and pathogenesis is complex and multifactorial.[2,3]

Recent research has suggested that dysautonomia is the major cause of symptoms in CFS.[2,4–7] Hakim and Grahame[8] report that 60% of joint hypermobility syndrome (JHS) patients suffer from dysautonomia. The autonomic dysfunction found in conditions such as JHS, CFS and fibromyalgia syndrome (FMS) share common characteristics.[2] Rowe *et al.*[9] reports that CFS patients responded no better than placebo to the routine treatment which is effective for other patients with orthostatic intolerance (OI) due to sympathetic neurocirculatory failure. Naschitz *et al.*[7]

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found that dysautonomia, which manifests primarily as disordered regulation of cardiovascular responses to stress, in CFS was different in behaviour during a head-up tilt test (HUTT) to that found in other conditions such as FMS, neurally-mediated syncope, and non-CFS fatigue.

This would suggest a potential role for physiotherapy: can physiotherapy influence these symptoms? The following provides an account of the experiences of one patient, in which a mechanical effect greatly alleviated her symptoms of CFS, and returned her to a near-normal life.

History

Subject Characteristics

At the onset of her illness, she was 46 years old, married, with three children. She was a physiotherapist, working 30 h per week.

General health was reported as excellent, with above-average fitness: gym 6 days/week involving both cardio and weight training, plus bike riding or tramping during weekends. Previous medical history was mostly unremarkable: acromioplasty and debridement of right shoulder (slap lesion) in 1999; previous injuries: was a pedestrian knocked over by a car in 1995, injuring right medial collateral ligament and posterior cruciate ligament of her knee and fracturing head of fibula. The subject was not taking medication.

History of Current Complaint

November 2000

Diagnosed with CFS by a medical specialist as a result of reactivation of glandular fever; it was believed that she contracted glandular fever from her daughter, who had it in July of the same year.

February 2001

Feeling better after a 5 week holiday over Christmas; she rarely went to the gym during this time. On return to work, symptoms started to return: these were fatigue, a feeling of not enough sleep, and poor sleep. Night sweats were becoming very common. Exercise was harder; she was walking to and from work, a 15 min walk involving a 70 m hill. By now she could only go to the gym 2 days/week and it took 2 to 3 days to recover.

April 2001

Unable to attend gym. She could no longer walk home. Had intense headaches by 2pm each day, which were relieved by rest. Headaches then came on earlier and earlier, then progressed to a constant headache which was not relieved by rest or medication. She needed time off work.

May 2001

More time off work. The main problems were fatigue, nausea, bloating, plus abdominal pain, constipation, headaches, light headedness, foggy head, blurred vision, not sleeping and night sweats. Her GP made another specialist appointment so a brain scan could be requested. She had her vision checked – she did not need new glasses.

June 2001

She had had two near misses while driving, could no longer concentrate or focus her vision, fatigue was much worse as were headaches and nausea. By the end of her working day when she went outside she could no longer focus properly (the background was 'rushing towards' her). By this time she was having trouble reading as it was too 'blurry'. She was unable to take in and process written material. She could not problem solve; this progressed to being unable to make everyday decisions and she felt panic, like she could not cope, and was not able to process road/traffic conditions while driving.

6th June 2001

The subject experienced an acute episode. She could not get out of bed, because of fatigue and an intense headache. She felt nauseous as soon as she sat up; on trying to walk her heart rate increased, and she fainted (for the first time ever).

25th June 2001

The specialist confirmed a diagnosis of CFS. She was prescribed low dose antidepressants to see if these would help the fatigue and sleep; by this stage she was typically getting two hours of sleep per night. Antidepressants were ineffective. The specialist also recommended graduated walking each day: the subject attempted this but was unable to progress every day.

March 2002

The subject returned to work for 2 h per day.

August 2002

She managed to progress work to 3 h per day; however, after this, she had to lie down for the remainder of the day. The only household tasks she could perform were looking after the washing. Sitting for longer than 15–20 min and standing for longer than 5 min caused an exacerbation of symptoms.

2004

There was no significant improvement over the intervening two years. Some days she attempted

walking to work; she was only able to cook the dinner 20% of the time. The subject reported that by this stage, she had no social life.

2005

By this stage, she was able to walk to work most days, and to cook dinner at home 80% of days. She still needed to go to bed for 1 h after lunch everyday, and was still only getting 2 to 3 h sleep/night.

December 2005

The subject got an opportunity to undertake a Clinical Pilates course, and registered for two levels over 4 full days.

Patient's experience

The Pilates course attended in December 2005 included a section on hypermobility, dysautonomia, chronic fatigue and fibromyalgia; she was selected by the instructor to be the model for this section.

Demonstration

She was asked whether she had ever been in an MVA accident. She had been knocked over by a car in 1995.

On examination, Brighton Criteria for joint hypermobility syndrome was positive (see Table 1).

Thoracic rotation Left>Right

Exercise 1

She was given an exercise to do using the trapeze table. In a seated position, using her arms, she pulled the bar down and included a mid-range right thoracic rotation, $\times 30$ reps. On completion, she found her foggy head and vision had cleared; her nausea had gone, as had the churning and bloating in her abdominal area.

Exercise 2

The instructor then asked her permission to turn her the other way. He got the class to look at her face before she started. She then rotated to the left. All symptoms returned sharply, she became clammy, and her legs would hardly hold her up. It was reported by the class that her face had gone a grey colour and her eyes had dulled.

Exercise 3

She then rotated again to her right with the same effect as previously.

Patient's reaction

The patient did not believe this was a placebo effect as she had no idea that the section on hypermobility, chronic fatigue and dysautonomia was part of the course, nor did she know what was going to happen as a result of the first and second sets of rotations. The instructor had not discussed what autonomic symptoms she was feeling before the demonstration; the subject had not considered what might happen during the session, as nothing had previously relieved the symptoms. The subject noted she was very tired, as it was the third day of the course, and she had not been able to have her normal lie down after lunch.

Table 1 Diagnostic criteria for joint hypermobility syndrome

Beighton score
- Passively dorsiflex the fifth metacarpophalangeal joint to greater than or equal to 90°
- Oppose the thumb to the volar aspect of the ipsilateral forearm
- Hyperextend the elbow to greater than or equal to 10°
- Hyperextend the knee to greater than or equal to 10°
- Place the hands flat on the floor without bending the knees

To have a positive score you need 4/9. Each of the first four categories you score one for each side and one for the final category

Brighton criteria

Major criteria
- A Beighton score of 4/9 or greater (either currently or historically)
- Arthralgia for longer than 3 months in four or more joints

Minor criteria
- A Beighton score of 1, 2 or 3/9 (0, 1, 2 or 3 if aged 50+)
- Arthralgia (>three months) in one to three joints or back pain or spondylosis, spondylolysis/spondylolisthesis
- Dislocation/subluxation in more than one joint, or in one joint on more than one occasion
- Three or more soft-tissue lesions (e.g. epicondylitis, tenosynovitis, bursitis)
- Marfonoid habitus
- Skin striae, hyperextensibility, thin skin or abnormal scarring
- Eye signs: drooping eyelids or myopia or antimongoloid slant
- Varicose veins or hernia or uterine/rectal prolapse

Scoring

Benign joint hypermobility syndrome is diagnosed in the presence of

Two major criteria or

One major and two minor criteria

Or

Four minor criteria

Two minor criteria will suffice where there is an unequivocally affected first-degree relative

Hakim and Grahame[8]

She was very surprised and excited by the effect of the exercise: she felt much better than she had in the previous 6 years. Indeed, she was feeling so good after class she went for a swim.

Possible explanations for the positive response

The treatment was able to reproduce or effectively eliminate (in the subject's words: 'turn on and off') her CFS (dysautonomia) symptoms by rotating her thoracic spine either to the left or right. The following will assess the factors that could have contributed to this effect, including JHS, dysautonomia, and the link with CFS and other conditions such as FMS. Possible explanations for how this simple movement could affect the autonomic nervous system are also considered.

Dysautonomia

Dysautonomia refers to a change in the autonomic nervous system function (ANS) that adversely affects health.[10]

Joint hypermobility syndrome (JHS)

JHS is a heritable disorder of connective tissue. Recent research[8] has shown that neurophysiological abnormalities, such as joint proprioceptive impairment, resistance to local anaesthetics, ANS dysfunction (such as faints, palpitations and gastrointestinal disturbance), fatigue, anxiety and psychological distress are associated with JHS; it has been known for some time that recurrent widespread pain patterns, which can lead on to chronic pain and deconditioning, are also associated. Joint instability, joint deformity, subluxation and/or dislocation can be other presenting symptoms.

Previously, the gold standard for classifying JHS was the nine-point Beighton score; now it has been superseded by the 1998 Brighton criteria. These new criteria allow for stiffening of joints with age, for hypermobility to be pauci articular rather than poly articular, and for other joints to be hypermobile. They also take into account hypermobility of other structures, such as skin extensibility, varicose veins, hernias, prolapses and soft tissue lesions, etc. Table 1 presents the criteria and scoring.

Dysautonomia is a common manifestation of JHS.[8] Hakim and Grahame[11] looked at the prevalence of non-musculoskeletal symptoms experienced on a regular basis. These included: '(Pre) syncope (feeling faint, actually faint, dizziness and light-headedness);

Cardiorespiratory (CR) (palpitations, chest pain and shortness of breath);

Gastrointestinal (GI) (nausea, stomach ache, diarrhoea and constipation);

Common JHS concerns (fatigue, joint pain, anxiety and depression);

Non-specific (migraine, allergy, rash, nocturia, dysuria, flushing, night sweats, fever, lymph gland pain and poor sleep).' (Pre) syncope, CR or GI symptoms were reported by 60% of JHS patients. Hakim and Grahame concluded that non-musculoskeletal symptoms were more common in JHS patients, who also reported more fatigue, anxiety, migraine, flushing, night sweats and poor sleep than the controls.

Gazit *et al.*[12] performed ANS function tests in patients and healthy controls to investigate a pathological basis for frequently reported autonomic symptoms such as palpitations, light headedness/dizziness/blurred vision, presyncope and syncope, and heat intolerance. All patients reported five or more orthostatic symptoms for at least 6 months. Gazit *et al.* found a pathophysiological basis for the ANS related symptoms, particularly dysautonomias, such as syncope, postural orthostatic tachycardia syndrome and mild orthostatic hypertension: they suggested that dysautonomia is an extraarticular manifestation in JHS.

Chronic fatigue syndrome (CFS)

There is increasing evidence that dysautonomia manifests in CFS, primarily as disordered regulation of the cardiovascular response to stress. Freeman *et al.*[6] looked at the role of the ANS in CFS patients' symptoms: these patients reported that the following symptoms occurred frequently: light headedness, nausea, diarrhoea, constipation, early satiety, urinary frequency, urinary urgency, erectile difficulty in men, excessive perspiration, and cold extremities. No control subjects reported that any of these symptoms occurred frequently. Freeman *et al.* ran a number of tests to evaluate sympathetic and parasympathetic nervous system function, and were able to confirm that patients with CFS exhibit alterations in sympathetic and parasympathetic nervous system function. They were able to show that confounding factors, such as depression and anxiety (which are frequently reported in CFS), did not correlate with any of the measures of autonomic dysfunction. Freeman *et al.* were also able to show that deconditioning was not responsible for the autonomic abnormalities, and suggested that orthostatic intolerance (OI) contributes to the fatigue in these patients.

OI is a clinical manifestation of sympathetic neurocirculatory failure.[10] Rowe *et al.*[9] suggest there

is a particular form of dysautonomia in patients with CFS, and report that by studying prolonged head-up tilting, all seven consecutive patients who satisfied the criteria for CFS showed neurally-mediated hypotension. Such patients have abnormal blood pressure or pulse rate responses, with sudden hypotension or severe bradycardia or tachycardia. This is accompanied by a decreased level of consciousness. Bou-Holaigah *et al*.[5] found 70% of CFS patients and no controls showed an abnormal response to upright tilting. Rowe *et al*.[9] reports that CFS patients with OI due to sympathetic neurocirculatory failure did not respond to normal treatment (a sodium-retaining steroid fludrocortisone combined with a high salt diet): it was no more effective than a placebo.

Naschitz *et al*.[7] went further and found that CFS patients responded differently to those with other dysautonomias in the head-up tilt test (HUTT). They used the 'hemodynamic instability score' (HIS), which involves blood pressure and heart rate changes being computed during the course of a HUTT; this test was found to have 97% sensitivity and 96·6% specificity for differentiating CFS patients from healthy subjects. They also compared CFS patients with those with disorders similar in clinical presentation to CFS, and disorders in which dysautonomia is known to be present. The specificity of HIS for CFS was 85·1%; this suggests unique features may be present in CFS dysautonomia, and that dysautonomia is pivotal in the pathophysiology of CFS, at least in a large number of patients. Thus, manipulating the autonomic nervous system (ANS) may be effective in the treatment of CFS.

Afari and Buchwald[2] in their review of CFS highlighted that findings from MRI studies show significantly more abnormalities in the subcortical white matter of CFS patients than in other patients. In single photon emission computed tomography (SPECT) scans CFS patients were found to have lower levels of regional cerebral blood flow throughout the brain. Abnormalities in CNS perfusion have also been found, particularly hypofusion. During cognitive testing, a significant deficit in information processing, poor learning of information, and impaired working memory have also been shown. Neuroendocrine studies have shown up abnormalities in the hypothalamic-pituitary-adrenal axis, CNS serotonin physiology and hormonal stress responses. Afari and Buchwald[2] also report autonomic dysfunction in tilt-table testing, demonstrating hypotension with bradycardia or hypotension with tachycardia in CFS patients.

Other conditions

It has been suggested that fibromyalgia syndrome (FMS), which is characterised by chronic diffuse body pain, fatigue and characteristic tender points, plus many of the same ANS symptoms as CFS, should be classified as a different manifestation of the same condition. Afari and Buchwald[2] found that 20–70% of patients with FMS also meet the criteria for CFS, and 35–70% of those with CFS-like illnesses have concurrent FMS. Hakim and Grahame[8] report conflicting evidence of the association between FMS and JHS, and suggest that there may be subgroups of patients with CFS or FMS who are also hypermobile. Dysautonomia is the primary presentation.

Nijs[13] reported that 20·8% of FMS patients also have generalised joint hypermobility (Beighton Score); Nijs also reported that 58·8% of CFS patients fulfilled the criteria for BJHS (Brighton criteria) and that 25% met the Beighton criteria.[13,14] This suggests that a subgroup of both CFS and FMS patients also present with JHS. These prevalence rates are higher than in the general population.

Effect of mechanical treatment on the autonomic nervous system.

As suggested above, manipulating the ANS may be effective in treating the dysautonomia which manifests in JHS, CFS and FMS. However, what evidence is there that physiotherapy techniques may influence the autonomic nervous system?

The ANS is divided into sympathetic and parasympathetic nervous systems; all central nervous system (CNS) information to and from the limbs, head and neck, trunk, pelvis and viscera must pass through the thoracic area where the sympathetic nervous system (SNS) is located. From here it travels to the entire body. The cell bodies of the parasympathetic nerves are in the CNS and their fibres exit either via fibres in the gray matter of the brainstem within cranial nerves III, VII, IX and X, making up the cranial parasympathetic outflow; or via fibres exiting the ventral roots of spinal nerves S2 to S4 or the pelvic splanchnic nerves, which make up the sacral parasympathetic outflow. The SNS cell bodies are found in the grey matter in the lateral horn of the spinal cord segments T1 through to L2 or L3. Sympathetic fibres leave the spinal nerves through the white rami communicantes to join the sympathetic trunks (ST). Here they take one of three courses. They may synapse with a paravertebral ganglion at that level, or they may ascend or descend up to six segments and synapse there, or they may pass through, without synapsing, as part of an

abdominopelvic splanchnic nerve. One preganglionic neuron may synapse with up to twenty postganglionic neurons within the ST before exiting via the grey rami communicantes. Sympathetic fibres enter all the branches of the spinal nerves to reach their destinations. The head and neck are supplied from levels T1 to T4, the upper trunk and upper body from T1 to T9, and the lower trunk and limb from T9 to L3.[15–17]

The sympathetic nerves may become compromised in several places. Fibres pass the dorsal root ganglion, which sits in the intervertebral foramen. The ST is an irregular line which lies on or lateral to the costovertebral joints, branching and looping on the necks of the ribs. It has branches that pass over the vertebral bodies or costovertebral joints, and it forms part of the sinus vertebral nerve which supplies the posterior disc, facet joints, ligaments and dura mater. Evans[16] suggests that in all these places there is potential for trauma (such as compression, entrapment, ischemia), and the ST may become stretched, or buried in osteophytes. Butler and Slater[18] report that as we move our upper and lower quadrants, we put tension on the thoracic neural tissues; they go on to suggest that loss of normal movement and tension requirements of the SNS may be a mechanism for sympathetically maintained pain. They believe that the STs, ganglia and rami are particularly vulnerable to mechanical interference from pathological changes in interfacing tissues. This damage may be direct (as in laceration) or caused by overstretching (as in high impact accidents such as motor vehicle accidents), traumatising the STs, or a sudden slump movement in sport.[18,19] Alternatively, the STs may be indirectly injured by mechanical irritation such as from a sprained or swollen costovertebral or zygoapophyseal joint, or irritation of a spinal nerve as it emerges from the intervertebral foramen. Distortions caused by postural changes or by blood congregating around neural tissue have also been suggested. Butler and Slater[18] also suggest the ST may be implicated in chronic injuries from occupational and sporting overuse. Osteophytes, particularly in the area of the costovertebral joints and the vertebral body, may compromise the STs. Nathan[20] studied 1000 cadavers and found that 65% had osteophytes compressing the sympathetic structures, with 65·5% of those affected showing compression on the right side. Nathan[21] also found that all 400 of the vertebral columns studied had evidence of osteophytes by the fourth decade. The highest incidence of osteophytes in the thoracic spine was found around the tenth thoracic vertebra, with a predominance clearly found on the right hand side form the fifth thoracic vertebra down. Zusman[22] suggests that prolonged nociceptor-initiated reflex muscle spasm could lead to inappropriate repair and contracture of articular and neural connective tissues and thus the SNS. The name 'T4 syndrome' has been given for the syndrome of a hypomobile joint around T2-T7, where patients experience pain and SNS symptoms. It has been suggested that this is caused by the proximity of the sympathetic ganglion to the costovertebral joint when these have become swollen or sprained. This is then thought to result in increased stimulation of sympathetic outflow by vasoconstriction.[18,19]

A number of studies have looked at autonomic effects, in particular the sympathetic response, during and after manual therapy techniques. All the early studies involved asymptomatic subjects. Sympathoexcitatory effects have been shown to be technique-specific and active treatments to be more effective than placebos. Under controlled conditions, Slater *et al.*[23] loaded the ST by performing a 'sympathetic slump' and evaluating skin conductance and skin temperature to assess peripheral SNS response; their results demonstrated that this technique differentially increased sympathetic activity in the ipsilateral upper limb. Simon *et al.*[24] applied an anteroposterior glide to the glenohumeral joint and found a generalised increase in SNS activity in response to the technique: skin conductance showed a significant increase, and skin temperature was significantly decreased, thus showing a sympathoexcitatory effect. Paungmali *et al.*[25] similarly found a sympathoexcitatory effect in response to a mobilisation with movement for lateral epicondylalgia, including changes to heart rate, blood pressure, and cutaneous sudomotor and vasomotor function. Peterson *et al.*[26] had previously reported similar effects in response to cervical mobilisations in normal subjects; Vicenzino *et al.*[27] confirmed sympathoexcitatory effects on asymptomatic patients while performing a neural mobilisation technique.

Vicenzino *et al.*[28] later confirmed such effects in symptomatic subjects: manual therapy techniques were found to produce hypoalgesic and sympathoexcitatory effects, by producing changes in sudomotor, cutaneous vasomotor, respiratory and cardiac activity. On the basis of these findings, Vincenzio *et al* proposed that a central control mechanism might be activated by manual therapy, through the activation of descending pain inhibitory system(s) (DPIS) mediated through the midbrain periaqueductal gray region (PAG). Sterling *et al.*[29] provided further evidence of this proposal by looking at motor

activity as well as effects on pain and SNS activity. Wright[30] has proposed that by stimulating the dDAG (lateral column) we get a sympathoexcitatory response and an analgesic response, as well as motor facilitation; however, stimulating the ventrolateral column inhibits SNS function, resulting in reduced motor activity by redirecting the blood flow from the muscles to the viscera, and it also produces analgesia.

In patients who present with SNS symptoms such as in 'T4 syndrome' it is understood that by restoring the movement in the hypomobile joint, through manual therapy, we effect a reversal of the SNS symptoms.[17,18,31]

Discussion

As the current patient was classified with JHS she has the potential to suffer from dysautonomia; she was diagnosed with CFS as a result of presenting with dysautonomia, and the belief that glandular fever can result in CFS. She had also been involved in a high impact injury which resulted in a right rotational force to her thoracic spine as she fell to the ground (her foot was trapped under the wheel of the car). Because of her hypermobility, she may have had more potential to traumatise the SNS. As the symptoms did not appear immediately, it is less likely that the SNS was involved in excessive stretching at the time, to cause an acute onset of dysautonomia. The symptoms appeared to develop over time, perhaps as scarring occurred. Beyond this, when the symptoms became more noticeable, the patient was doing a lot of 'bodycombat classes' at a gym. Because of her shoulder problem (and then surgery), she may have protected her shoulder by involving more trunk rotation to the left, and thus may have over stretched the sympathetic trunk or ganglion on the right, which may have been involved in prior scarring. Another potential cause may have been osteophytes: as noted above Nathan[21] found that 100% of cadavers studied had osteophytes by the fourth decade. Grieve[32] reports that osteophytes are found predominantly on the right hand side, with a peak incidence at levels T5-T12. Because of the previous trauma the subject may be more likely to have osteophytes, which may have then involved the SNS. However, if this was the case, it is logical to ask, why she has not experienced any thoracic back pain? Bogduk[33] reports that there is no physiological evidence that all costotransverse or zygoapophyseal joints affected by degenerative joint disease become painful. It is also well accepted that pain from somatic structures may closely simulate visceral disease.[34] Many such patients have pain-free back movements and it is not until the spine is examined that pain is provoked; Dreyfuss *et al.*[35] found that 27·5% of subjects did not report pain after having thoracic zygoapophyseal joint capsules distended by joint injections.

Studies have assessed peripheral SNS effects during manual therapy techniques applied to peripheral joints, spinal joints, and by stretching neural structures. In the current case, mid-range thoracic rotations appear to have had an effect on the ANS; to date no study has systematically assessed the effects of specific exercises on the ANS.

Simon *et al.*[24] has proposed that abnormal SNS activity may exacerbate pathological conditions, and that physiotherapy techniques may serve to modulate this activity. Whether therapy mediates a sympathetic inhibitory effect via vPAG, or an excitatory effect via the dPAG, depends on which part of the brain is activated. Slater[36] highlights the importance of autonomic regulation occurring through neuronal cell pools located in the brainstem: this central autonomic network has indirect and direct reciprocal connections with the parasympathetic and the sympathetic nervous systems from the cranial outflows and the spinal cord. Slater suggests that this arrangement may serve as a feedback mechanism to regulate sympathetic, parasympathetic and endocrine functions. There are also well-developed interconnections between these neuronal cell pools and autonomic integrative centres in other areas of the brain. This may explain why the dysautonomia symptoms experienced by JHS, CFS and FMS subjects do not always represent a decrease in an SNS response (and thus more parasympathetic nervous system symptoms), but can also represent increased SNS symptoms and a mixture of the two, as this feedback mechanism becomes dysfunctional.

JHS is familial.[8] Those who present with dysautonomia but have not been recognised as being hypermobile may be diagnosed with CFS. Research suggests that CFS may be familial but, based on the studies done to date, this cannot be generalised to a broader population.[2] There may be a subgroup of CFS suffers who are also hypermobile. This may explain why the research suggests CFS is familial; however, further research is required to elucidate any such link.

Conclusion

There appears to be a subgroup of patients who are hypermobile and who are also suffering from

dysautonomia, whether they have been diagnosed with CFS, JHS or FMS. By finding a directional preference and exercising these patients in that direction, we may potentially help this large group of patients. This paper suggests that physiotherapy has the potential to relieve symptoms, particularly for those who have been involved in accidents which involve a rotational movement. Research now needs to be focused on whether physiotherapy can help this subgroup.

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