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An evaluation of the effectiveness of osteopathic treatment on symptoms associated with Myalgic Encephalomyelitis. A preliminary report

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Introduction

The term Myalgic Encephalomyelitis (ME) was initially used in the 1950s [1]. ME describes a syndrome where there is general muscle pain associated with evidence of a disturbed nervous system [2]. ME, commonly referred to as Chronic Fatigue Syndrome (CFS), or post-viral fatigue syndrome is a condition in which mental and physical fatigue predominate. It is characterized by gross abnormal muscle fatigue which occurs after relatively mild activity. Other symptoms regularly complained of include sleep disturbance, headaches, cognitive dysfunction, feeling depressed, bouts of low grade fever (not exceeding 38·6°C), increased sensitivity to light, back and neck pain, sore throat, irritable bowel and bladder [3]. The symptoms of ME typically become apparent following a viral infection [4], although other trigger factors have been noted. Vaccinations against cholera, tetanus, typhoid and influenza have been associated with the onset of ME [5]. It has also been observed that any psychological disturbances in ME occur secondary to, or share a common pathophysiology with an immunological dysfunction [6]. In many cases there appears to be no apparent triggering factor [7].

A chance discovery during the past four years of conventional osteopathic practice of the first author, revealed a plausible correlation between a mechanical dysfunction of the thoracic spine and the incidence of ME [8]. Promising results achieved by this author with osteopathic treatment of ME patients have led to a hypothesis that a cause of ME is a mechanical dysfunction affecting the upper back which leads to a chronic disturbance of the sympathetic nervous system. Furthermore, this dysfunction responds favourably to biomechanical treatment, which involves manipulation of the intervertebral apophyseal joints of the thoracic spine and massage of the surrounding soft tissues to increase blood supply and stimulate lymphatic drainage.

Treatment advocated for ME in the past has included anti-inflammatory drugs with muscle relaxants [9]. However anti-viral drugs showed no greater effect on ME than a placebo [10]. Other studies concluded that treatment should be based on supportive counselling coupled with psychiatric treatment, and that the patient should be encouraged to gradually increase everyday activity [11]. Yet, none of these treatments has proved wholly satisfactory and absence of the ultimate curative drug has led to many alternative treatment approaches such as oral anti-fungal drugs, and strict exclusion diets. This use of anti-fungal agents has resulted in some cases of hepatitis [12]. Alternative treatments have included intramuscular injections of magnesium sulphate [13], but at present there still remains much scientific uncertainty regarding the aetiology, diagnosis and treatment of ME. This has led to a refusal by many practitioners to admit its very existence and a recent study in Australia showed that 70 per cent of a group of doctors were reluctant to make a diagnosis of CFS [14].

Recent research has alluded to a possible deficit within the central nervous system [15]. Although there is still a large school of thought that suggests a viral cause, many new research studies have demonstrated the unlikely event of viruses being the underlying cause of this disease [16–19].

The conventional advice for relief of the symptoms includes psychotherapy, physiotherapy, exercise programmes, acupuncture, or antidepressants [20]. Dietary programmes are being investigated, with evidence to suggest that an essential fatty acid intake must be normalized in the management of ME [21]. The amount of research into a viable treatment of ME is negligible compared with world-wide investigation of the actual mechanism causing the disease. Since the actual existence of the disorder is still a source of controversy, a universally accepted treatment for ME remains highly unlikely until there is a change of attitude.

The authors believe that by demonstrating the efficacy of osteopathic treatment of this disease, more importance will be given to mechanical and physical aspects of the disease. Since ME as a specific entity has not been recognized by all the scientific world, and a proven method of diagnosis has not yet been documented, many previous clinical trials have been flawed in their claims to have treated the disease.

There has been a long-standing debate over the naming of this disorder. Some have expressed the opinion that ME is a highly specific disease, whereas CFS is an umbrella term covering many conditions which exhibit fatigue. It is our opinion that all the terms used for this

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(5) The subjects understood the importance of continuing the treatment until the end of the year, although they were free to leave the project at any time.

(6) The patient was willing to be part of a longer follow-up study.

Exclusion criteria

(1) Subjects receiving other treatment for their ME symptoms were excluded from being part of the patient group, unless they had received the other treatment as ongoing therapy for at least six months prior to the start of their participation in the project.

(2) Patient group members receiving any manual treatment for their ME symptoms other than that from the author were excluded from the study. Subjects were also eliminated from the project if they had received any prior physical therapy for their present symptoms.

(3) Control group members receiving any form of manual treatment for their ME symptoms were also excluded from the trials.

(4) No premorbid symptoms of depression.

(5) If there was a doubt as to the psychiatric state of the patient, or the subject was experiencing a primary depressive illness, they were excluded.

(6) No psychiatric history in the family.

(7) Subjects were excluded if they tested positive for any other untreated patho-physiological cause of the symptoms.

(8) A subject who had suffered from any other neurological disorder, was also excluded from the study.

Method

The experimental procedure involved two types of measurement. The first involved objective measures carried out in the laboratory to determine the physical condition of the leg muscles and the mobility of the thoracic spine. The second involved asking subjects to complete a series of questionnaires about their symptoms.

Laboratory measurements

It has been demonstrated previously that when a hand was exercised to induce fatigue, and at the same time blood flow was stopped by inflating a cuff around the upper arm, there was no recovery of power until the cuff was released and normal circulation was restored. This was the case even though the somatic innervation was still functioning [26]. This showed that in cases of impaired sympathetic function the resulting reduction in blood flow may precipitate a state of fatigue.

Since fatigue is a common clinical symptom of ME, it was considered essential to measure whether this fatigue was relieved by the treatment. Whilst carrying out this measurement it was imperative to avoid injury to the muscle. The knee extensors in the right thigh were chosen for this measurement because the fatigue effect of ME is particularly evident in these muscles. The fatigue test involved isometric measurement of the static torque exerted about the knee by the extensor muscles of that joint using a specially designed chair as shown in figure 1.

During each measurement the patient was seated with the leg hanging vertically. A lever attached to a torque transducer at the level of the knee joint axis was aligned along the lateral side of the leg with a padded extension that projected across the front of the shin, just above the ankle. During each test the lever was clamped in a fixed position. The patient was asked to exert as much force as possible against the padded extension and the resulting trace of torque at the knee was plotted against time on a pen recorder. After a set period of time the subject was instructed to stop pushing.

Functional electrical stimulation (FES) of the quadriceps might have provided a more accurate way of measuring fatigue as it could produce a set level of stimulation in the muscle [27]. However, it was felt that FES would have been too painful for these patients to withstand, and for ethical reasons we preferred to use active contraction controlled by the patient.

Weakness is defined as diminished ability of rested muscle to exert maximal force. Fatigue, however, is a loss of maximal force-generating capacity that develops during muscular activity [28].

The main problem encountered in measuring fatigue of the knee extensors, was that of achieving maximal force without causing major damage to the patients muscles. This problem was overcome by carrying out a pre-test in which the patient was asked to exert force on the leg pad a number of times and then resting for 3 min before the final test. These preliminary contractions served a dual purpose. Firstly they allowed patients to accustom themselves to the machine before applying maximal torque, and secondly they provided an exercise to induce some preset fatigue in the muscle.

Everyone has an in-built sensation of discomfort which safeguards them against exerting high levels of muscle force which can damage the muscle fibres. This is partly psychological and partly due to the physical sensation of pain. The psychological element of this is variable and will cause the subject to exert different levels of maximal force on different occasions. However, on any one occasion, if the subject attempted to maintain the exertion of muscle force over an extended period of time, fatigue eventually set in and caused the force to drop whether they wanted it to or not. Therefore, we

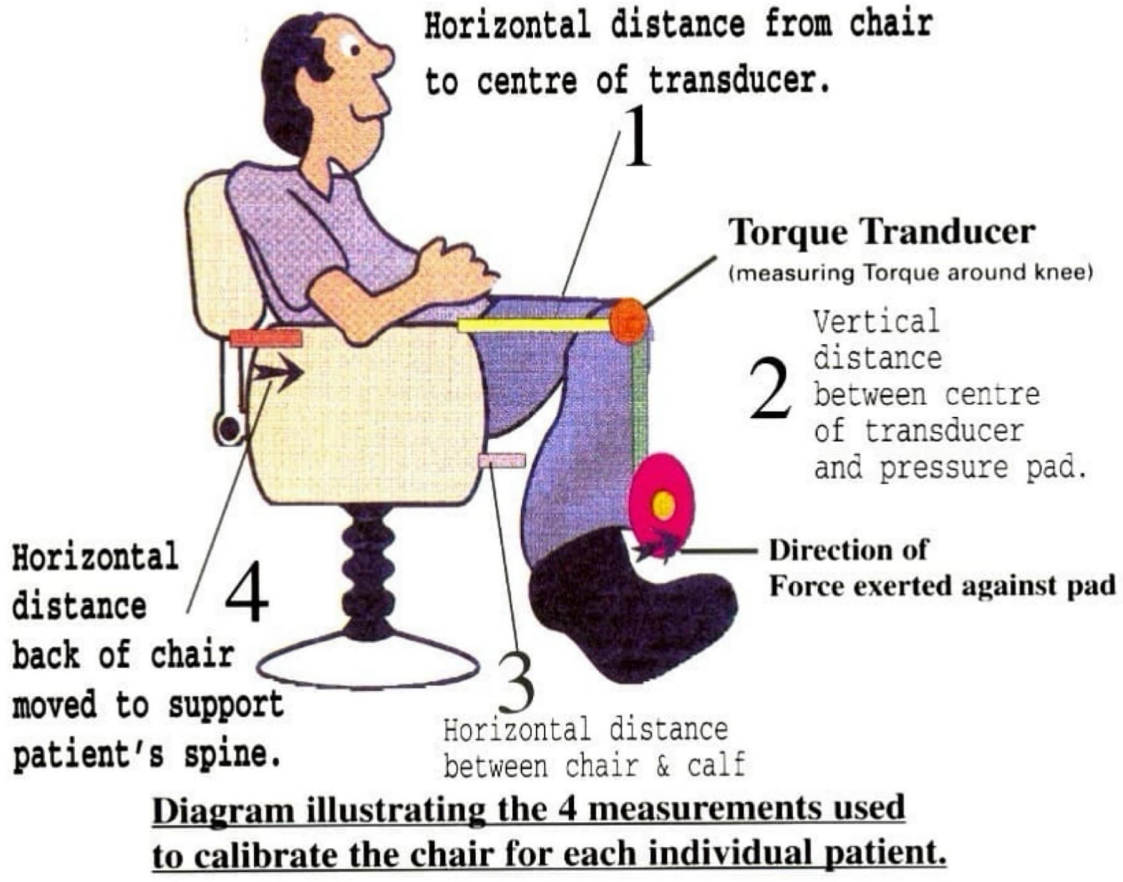

Figure 1. Illustration of the chair used for measurement of knee extension torque as part of the fatique assessment tests.

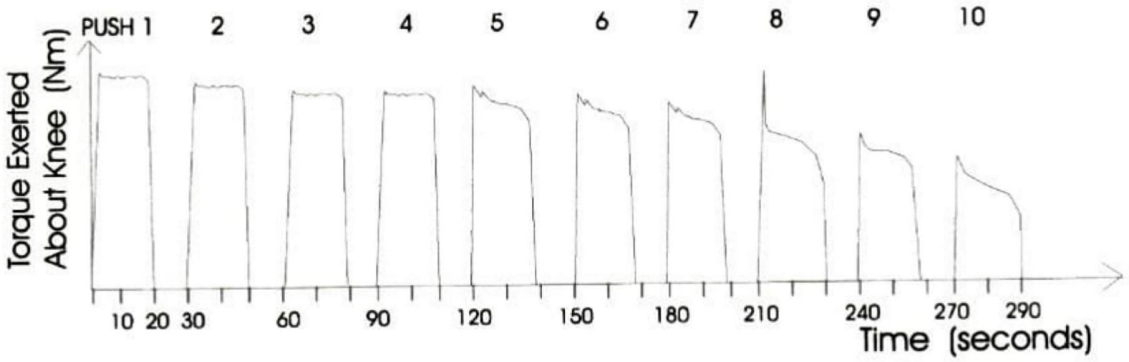

Figure 2. A typical plot of time against torque for the pre-test exercise sequence.

found that the rate of decline in muscle force due to fatigue was more significant than the peak force.

We found that the optimum sequence for the pre-test exercise was ten pushes, each lasting twenty seconds with an interval of ten seconds between each push. A typical plot with time of torque exerted about the knee during this sequence is shown in figure 2.

During the first four pushes after the initial peak, the torque was held relatively constant until the subject stopped pushing. During pushes 5, 6 and 7, after the initial peak, fatigue caused a gradual drop in torque over the 20 s time period when the patient was attempting to keep the torque at maximum. Sometimes, after a 10 s rest, the subject was able to momentarily achieve a torque as in push no. 8 similar to the original level at push no. 1. However, this was short lived and rapidly fell to a magnitude below push no. 7. By the tenth cycle of pushes the effect of fatigue had become fully established. A similar picture of fatigue with exercise pushes was evident in all the

patients, as well as in normals randomly selected from our University staff. Therefore, ten repeated push cycles was chosen for all our tests. After completing these ten cycles the subject was allowed three minutes rest for the muscle to recover. Then the patient was asked to push as hard as possible again, only this time they were requested to maintain the maximal push until they could no longer continue. A typical recording for this final push is shown in figure 3 where the torque about the knee axis in Newton metres is plotted vertically against time on the horizontal axis.

Newton's second law states: 'the change in linear momentum of a body under the action of an unbalanced force will be proportional to the product of the force and the time for which it acts'. This change is known as the impulse. The area under the pen recorder graph of torque against time was a measurement of the change in impulse which was directly related to the work done by the muscle. This was inversely proportional to the fatigue of the muscle and so was a good method for determining the fatiguability of the knee extensors. That is, Ft = Impulse and this was defined as the 'Effective Work Done'.

When the subject was asked to press for as long as possible, a difficulty arose in evaluating exactly what was meant by 'as long as possible'. It was not the intention of the researcher to cause muscle damage and this would have added too many subject factors into this experiment, thus increasing the margin of error. In a pilot study it was noted that the patients could sustain the final push for at least thirty seconds before relaxing. Subsequently the first thirty seconds of the final push was the time chosen to evaluate the fatiguability of the muscle. The patient was then allowed to stop after this time to prevent any injury.

According to the above definition, in figure 3 the shaded area under the graph is related to the effective work done by the patient during the final push of the test. Thus the larger this area, the less was the fatigue of the muscle over a 30 s time period.

After each test we also recorded the subject's rating in the Borg Scale of Perceived Exertion. This perceptual effort rating was formulated as a behavioural and psychological measurement of physical performance and work capacity. The real value of exertion is proportional to the heart rate of the patient. (That is if the pulse after exertion was 100 then the real value of exertion scored 10 on the Borg scale). The patient was asked to score the perceived amount of strain they felt during the exercise by using the Borg scale where 6 = minimum effort required and 20 = maximum effort required [29]. As long as the difference between the real and perceived exertion during the initial tests did not increase in the final test, then the torque measurement improvement was shown not to be due to the patient simply putting more effort in at the end compared to the beginning of the project.

It has been postulated that irritation of the sympathetic nerves at the spinal level could lead to adaptive or

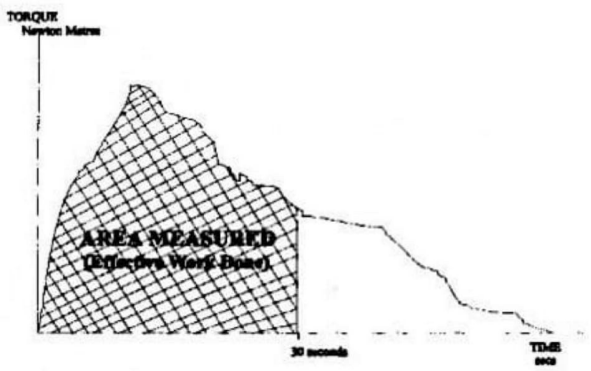

Figure 3. Graph of torque against time.

pathological changes in the tissues of origin. Altered excitability within the central nervous system may eventually be caused by the overactive afferent sympathetic supply. This may effect other tissues in viscera throughout the body leading to disease [30]. Based on this hypothesis, it was felt that a method to determine spinal mobility was needed. If the amount of movement in the thoracic spine was shown to be proportional to the severity of symptoms, then it could support the idea of physical irritation of nerve roots involved in the aetiology of ME.

Measurement of spinal mobility requires the shape of the spine to be recorded at different instants of time during movement of the vertebral column. In our study, we investigated several shape recording devices including the use of a flexicurve [31], as well The MAC Reflex gait study system (an infrared scanning technique involving automatic computerised digitization). The latter was found to be too time consuming, and was impractical because it had to be re-calibrated after each patient. Instead we chose to use the Salford Biomechanics Workstation. This device digitized movement of the thoracic spine from video film. The patient was positioned with the video camera placed laterally and focused on three probes that were fixed by adhesive tape to specific points on the subject's back overlying the spine. These points included T1, known as point C; T7 (point T) and L1 (point L). At maximum flexion, the angle CTL was recorded with the digitizer. This method was painless and required minimal time. Furthermore, the maximum error of distance measurement with this system was found to be 0·03% in the horizontal view, and 0·9% in the vertical view [32]. The position of C, T and L were recorded and remained unchanged. For each individual, the probes were placed exactly over the same segment as in the previous recording of the spinal movement, thus keeping the distances O–C; C–T and T–L constant for each subject.

Throughout the 12 months treatment period, each patient was also subject to graded clinical assessment of thoracic spine mobility, and muscular tone of paravertebral muscles. These assessments utilized palpation and proprioceptive techniques. In addition, tissue health was graded using a simple scoring system related to individual segments of the thoracic spine. This

grading system was scored by the researcher and randomly calibrated by another osteopath. It was carried out as part of a routine clinical visit.

Self report questionnaires

The following nine self-report questionnaires were filled out by all members of both the patient and control groups. The first two questionnaires were developed by the author specifically for this study and tested in a pilot study on nine patients before the start of the clinical trials. The pilot study's other aim was to evaluate the equipment used for objective measurements of muscle fatigue and spinal mobility. Questionnaires 3 to 8 were chosen for this project as they had already been validated in previous research studies, most involving ME. After examining other similar inventories and reviewing the literature on these questionnaires, they were deemed as suitable, precise, and easy to use.

Questionnaire no. 1. A General Health questionnaire, was developed especially for this study. It was based on twenty-six common symptoms complained of by ME patients. The higher the score, the worse the symptoms.

Questionnaire no. 2. Back pain questionnaire This was developed to examine a possible correlation between the amount of back pain and the severity of the other symptoms associated with ME.

Questionnaire no. 3. The revised Beck depression inventory (BDI) [33] and

Questionnaire no. 4. The Beck anxiety inventory (BAI) [34]. These were chosen as the most suitable depression and anxiety questionnaires for the project, as they were short, requiring only 5–10 min to complete, easy to score and had a cut off point. Given that anxiety and depression frequently coexist [35,36], the results from instruments designed to measure the severity of anxiety or depression are highly correlated with one another [34,37]. The BAI was formulated to measure symptoms of anxiety which are minimally shared with those of depression, and thus it was a suitable anxiety questionnaire to use with the BDI. Over the last 26 years the BDI and BAI have become widely accepted instruments for detecting and assessing the intensity of depression and anxiety in non-psychiatric patients [38].

Questionnaire no. 5. The Morgan-Gledhill sleep questionnaire [39]. Sleep disturbance is one of the most common symptoms of ME. A recent study showed that many patients with CFS had trouble staying asleep [40]. Actimetering measuring techniques [41] were considered for the project but were found to be too costly and difficult to use. The Morgan-Gledhill sleep questionnaire was one of only a few established sleep questionnaires, and can be scored to suit the needs of a particular project.

Questionnaire no. 6. Broadbent's cognitive function questionnaire [42]. Pronounced and frequent cognitive deficits have been found in patients with ME when attempting to carry out mental performance tests [43]. Since no direct contact was made with the control group by the authors, it was decided to use a cognitive function self-report questionnaire. The Broadbent's CFQ has been well validated and is suitable for use in this study as demonstrated by other ME research projects [44], and again this questionnaire was easy to complete and score.

Questionnaire no. 7. The Nottingham Health Questionnaire [45]. It was felt that The Nottingham Health Questionnaire is a quick, simple indicator for the general symptoms of ME and widely accepted. Also Dr Charles Shepherd, the medical advisor of 'The ME Association', advised us to include this questionnaire in the project in order to gain acceptance of our research findings by his nationally acclaimed group.

Questionnaire no. 8. The profile of fatigue related states (PFRS) [46]. The PFRS is a multidimensional measure incorporating nearly all the symptoms associated with ME and was developed at Brunel University especially to measure the symptoms of illness and to evaluate the effects of treatment [47]. It is longer than the other questionnaires, but is still quite easy to complete and not too difficult to score. It has four scales: emotional stress, cognitive difficulty, fatigue and somatic symptoms.

Control group

The control group was chosen by 'Action For ME' from a volunteer group which met the CDC criteria for Chronic Fatigue and The London Criteria. There were initially 40 members of this group but the numbers dwindled over the year to 23 by the end of the project.

All members of the control group started in April 1995 and were sent via the Action For ME, a set of the self-report questionnaires every 3 months. With the above questionnaires they were also sent a general questionnaire to determine which treatment they had been receiving during the past 3 months. This gave useful information regarding the efficiency of other therapies in treating this disorder. After each subject had completed five sets of questionnaires, they finished their participation in April 1996.

The patients continued to fill out the questionnaires quarterly until the project was completed.

Patient group

Forty patients meeting the Centres for Disease Control and Prevention's working case definition for Chronic Fatigue Syndrome were initially chosen to be part of the patient group. Five of these were disqualified for failing the Action for ME questionnaire based on the London Criteria, and one had to leave the country, unexpectedly. These patients were given code numbers RP01 to RP40 to protect their anonymity. They also received secret code numbers which were given to them by an independent

observer to be used by the subjects when completing their questionnaires. The identity of the secret code was kept hidden from the researcher until later on in the project. This number allowed freedom to answer the questionnaires truthfully without the researcher influencing the reply. This system was adopted to reduce bias and thus increase the validity of the questionnaires. Only one subject has subsequently 'dropped out', leaving thirty-three still having osteopathic treatment one year after signing on as a research subject.

Treatment

The treatment of each ME patient consisted of the following techniques:

(1) Soft tissue massage of the paravertebral muscles, the trapezii, levator scapulae, rhomboids and muscles of respiration.

(2) High and low velocity manipulation of the thoracic and upper lumbar spinal segments using supine and side-lying combined leverage and thrust techniques.

(3) Gentle articulation of thoracic and upper lumbar spine, plus the ribs. This was achieved by both long and short lever techniques.

(4) Functional techniques to the suboccipital region and the sacrum.

(5) Stimulation of the cranio-sacral rhythm by functional-cranial techniques.

(6) Efflourage to aid drainage in thoracic and cervical lymphatic vessels.

(7) Exercises to improve the mobility of the thoracic spine, and to improve the physical coordination.

All of the above techniques form part of the conventional clinical practice of osteopathy and are described in more detail in *The Handbook of Osteopathic Techniques*, 2nd edn by L. S. Hartman (Chapman and Hall, London, 1994).

Other advice

Osteopathic treatment is not synonymous with manipulation. Many treatments of numerous conditions were found to be insufficient if they relied on manual therapy alone [48]. As is standard in osteopathic practice, advice was also given to help improve general health.

Inflammation is usually combated by prescribing non-steroidal anti-inflammatory drugs (NSAIDs). A more natural method advised by many osteopaths is a technique known as contrast-bathing. This utilizes warm and cold compresses to dilate and contract the local blood vessels, thus stimulating the blood flow and accelerating the natural inflammatory process. In most cases of ME there is an inflammation of part of the spine and surrounding tissue. We chose contrast-bathing to reduce the inflammation, as it acts locally whilst avoiding the side effects of NSAIDs or steroids. Patients were advised to apply the contrast bathing of warm and cold compresses to tender areas of their backs, three times a day. In some severe cases this treatment was prescribed more often, whilst others used it less frequently.

Initially the patients were also instructed to reduce exercise and physical activity to half their capability. Once the patients' health had sufficiently improved to withstand slight physical exertion without any worsening of symptoms, they were advised to gradually increase walking activities, and if possible, to do back-stroke swimming.

It is important to understand that the entire treatment programme was being evaluated, and not just the manipulative methods.

Results

The fatiguability and spinal mobility tests were carried out on each member of the patient group every six months giving three readings over a 12 month period. They were also asked to fill out the questionnaires 1–8 every three months. The scores of the questionnaires were calculated by the researcher, and the mean improvement of both patient and control groups were compared. Questionnaire no. 1 had a minimum possible value of 26 as each separate complaint scored '1' when symptom free. The back pain questionnaire (no. 2) scored a minimum of 12, as each section of the back scored '1' if pain free. Likewise questionnaire no. 8 had a minimum value of 54 based upon the score of 1 for each symptom free complaint. All the other inventories scored zero for each symptom free item. The maximum and minimum possible scores for all the questionnaires are shown in table 1.

Figure 4 is a bar chart which plots the mean value obtained for each questionnaire answered by members of the control group at zero, 3 and 6 months after the start of the project. These values were plotted on the

Table 1. Scoring system of the questionnaires.

	Minimum	Maximum
Questionnaire No.1 Health	26	104
Questionnaire No.2 Back pain	12	48
Questionnaire No.3 Depression	0	63
Questionnaire No.4 Anxiety	0	63
Questionnaire No.5 Sleep	0	14*
Questionnaire No.6 Cognition	0	100
Questionnaire No.7 Nottingham	0	38
Questionnaire No.8 PFR	54	378

*The sleep questionnaire did not have a maximum limit, since one of the items evaluated was the time it took to fall asleep which has no upper limit, and which scored 1 point for every 10 min (e.g. 1 h=6pts). Many of the patients had severe sleeping problems scoring more than 14.

vertical axis as a percentage severity of symptoms with the questionnaire number indicated on the horizontal axis, where **0% = Symptom Free and 100% = Worst symptoms possible** (except no. 5 where 100% = 14 points on the sleep questionnaire).

The results for the questionnaire answers of the patient group are plotted in figure 5, with the severity of symptoms calculated as a percentage (again where: **0% = Sympton Free and 100% = Worst symptoms** (except no. 5).

QUESTIONNAIRE	1	2	3	4	5	6	7	8	AVE.
SCORE I	68	61.5	27	25.5	133	57	38	62	59
SCORE II	65	60.5	24	25	128	58.5	35	59.5	57
SCORE III	67.5	61.5	21.5	28.5	126.5	61.5	37.5	59	59

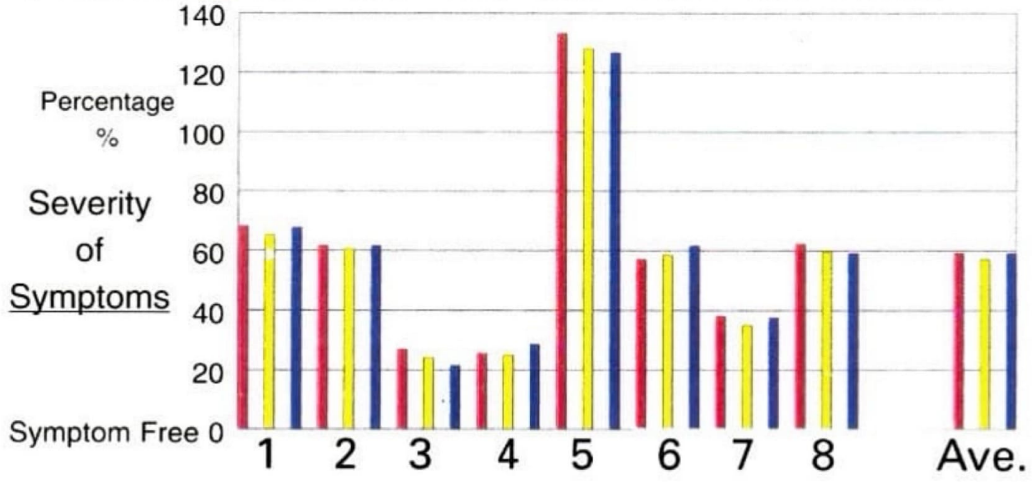

Figure 4. Control group: mean results of questionnaires. Score I (red bar)=Beginning of Project; Score II (Yellow bar)=After 3 months; Score III (Blue bar)=After 6 months.

QUESTIONNAIRE	1	2	3	4	5	6	7	8	AVE.
SCORE I	79.5	76.5	25	32.5	126.5	58	41.5	69	63.5
SCORE II	69.5	67.5	18	22	107	53.5	33.5	56	54
SCORE III	68	68	20	25.5	113	54.5	32.5	55	55

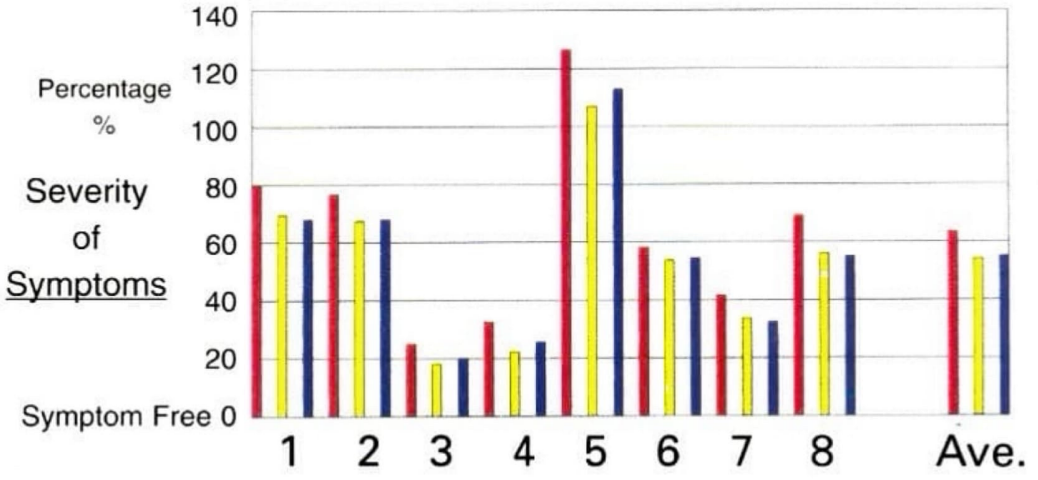

Figure 5. Patient group: mean results of questionnaires. Score I (red bars)=Beginning of Project; Score II (yellow bars)=After 3 months; Score III (blue bars)=After 6 months.

The final scores of the questionnaires of each individual subject have been recorded and compared with the scores recorded at the beginnning of the project. These can be seen in the bar charts (figures 6 (*a*) and 6 (*b*)).

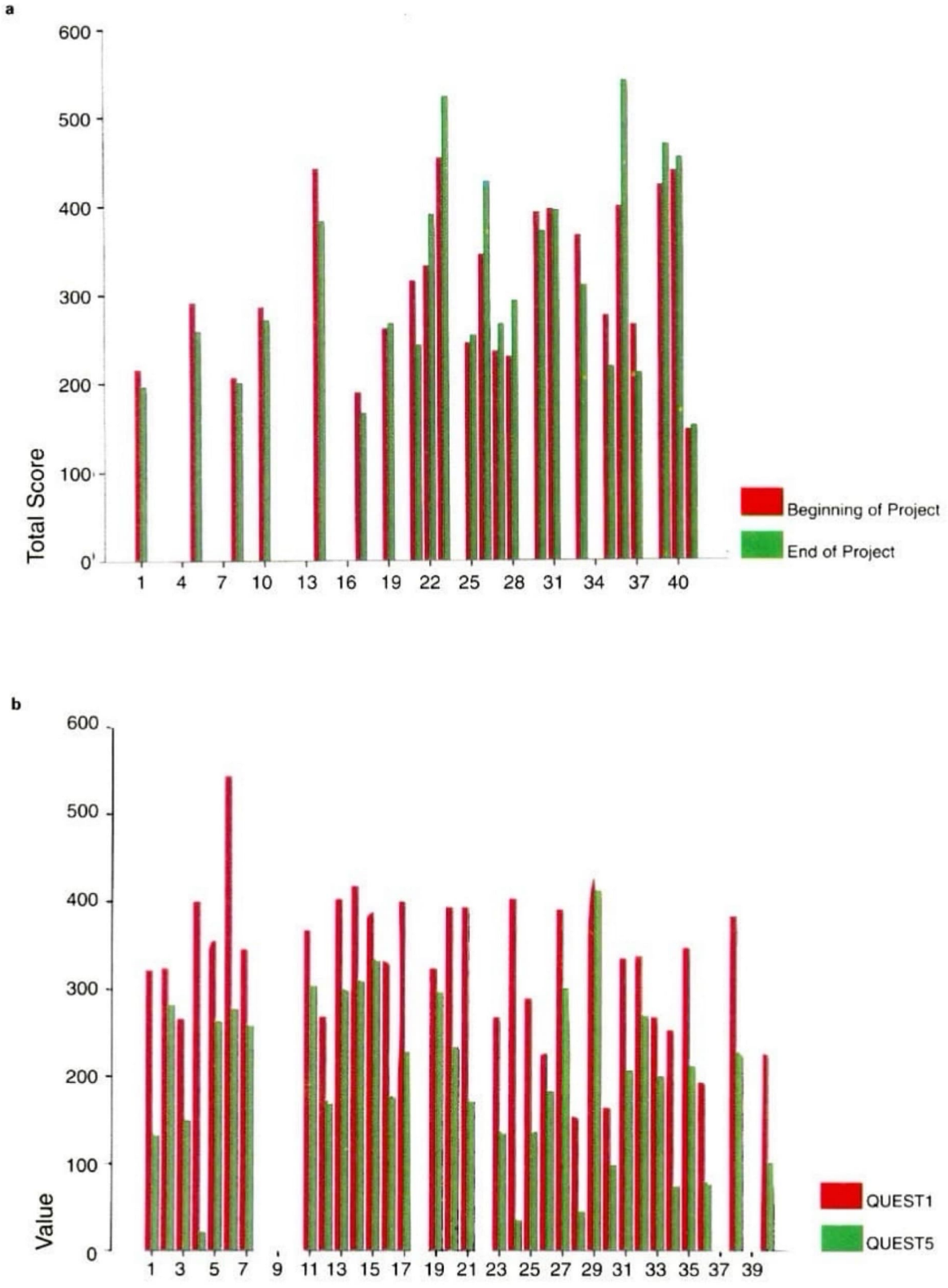

Figure 6. The change in score from the beginning to the end of the project for (a) the control group and (b) the patient group.

On these charts, the total score from all eight of the questionnaires for each subject is plotted vertically against the case number of the subject on the horizontal axis. The score values at the beginning of the project are marked in red while the corresponding values at the end of the project are marked in green.

For the control group figure 6 (*a*) shows that the change in score from the beginning to the end of the project is, in most cases, much smaller than in figure 6 (*b*) for the patient group. Furthermore, all the subjects in the patient group experienced a reduction in score (green bar lower than red), whilst for nearly 50% of the control group the score was greater at the end of the project (green bar higher than red).

The percentage change in these scores over the year was calculated for each subject, and the overall mean for both groups were compared as shown in table 2. This table shows that the mean score for the patient group improved by 40% as opposed to a 1% worsening in symptoms for the control group. There was no overlap in the standard deviations and with a p value of less than 0·0005, the results were highly significant.

Results of laboratory tests

The results of the knee extensor muscle fatiguability test utilizing the torque transducer chair were analysed comparing the individual patient scores at the start of the study to the scores at the end of the project. These are shown in table 3. An increase in this score measured in Newton metre seconds represents an improvement in the overall work done by the knee extensor muscles thus demonstrating a reduction in exercise induced fatiguability.

The mean results of the fatiguability tests were statistically analysed using a paired T-test comparing the torque × time before and after the year long study. The mean value at the beginning of the project was 1791 Nms, and 2259 Nms at the completion of the project. The difference in these values was highly significant with a p value of less than 0·0005.

In some patients the muscles were very weak at the start of the project (e.g. RP01). As they recovered, their improvement was more noticeable than those whose muscles were quite strong in the first place, e.g. RP07.

To determine spinal mobility, the maximum amount of spinal flexion was calculated by filming the patients in active full flexion and determining the minimum angle CTL (the smaller the angle the greater the flexion achieved). These results are shown in table 4 for patients RP01 to RP07 on five test occasions at three monthly intervals. It demonstrates that the thoracic mobility of these patients varied very little from the beginning to the end of the project. It can also be seen that there was no correlation with the improvement of ME symptoms.

Table 2. Comparison of the mean change in symptoms of both groups.

	Mean percentage change	Standard deviation	2-tail significance
Patient group	40%	15·8	$p<0·0005$
Control group	−1%	22	$p<0·0005$

Table 3. Scores of the effective work done in the right knee extensor muscles (measured in NMS)

Patient code no.	Test 1 (start of Project)	Test 2 (end of project)
RP01	606	2211
RP02	1077	1767
RP03	512	1255
RP04	716	1783
RP05	732	1182
RP06	1264	1559
RP07	1982	2641
RP11	583	711
RP12	1691	2971
RP13	1917	1366
RP14	5033	5014
RP15	2484	2945
RP16	2653	2990
RP17	1807	2604
RP19	245	704
RP20	518	826
RP21	659	1331
RP23	4776	4670
RP24	475	1179
RP25	1440	1777
RP26	5140	4658
RP27	277	1343
RP28	1793	2118
RP29	1109	1320
RP30	1594	1866
RP31	700	863
RP32	1695	3221
RP33	3002	2927
RP34	3698	3742
RP35	1727	2658
RP36	2757	3476
RP38	1364	1998
RP40	3093	3103

Table 4. Measurement of angle CTL indicating the maximal spinal flexion. Patients tested: RP01 – RP07.

	Test1	Test2	Test3	Test4	Test5
RP01	150·19	150·30	152·51	154·42	144·06
RP02	139·27	151·10	144·76	146·65	146·07
RP03	138·04	144·04	140·05	138·86	140·13
RP04	149·60	145·41	151·42	*****	150·97
RP05	138·87	3134·99	133·14	*****	133·57
RP06	139·43	136·62	138·94	*****	137·17
RP07	137·74	137·67	135	*****	135·45

Values of angle measured in degrees. **** = No measurement taken

Discussion

The main objective of this study was to demonstrate whether osteopathic treatment can reduce the severity of ME symptoms. Our results for the first 6 months confirmed the effectiveness of this treatment. Some of the results require detailed analysis and explanation. We have postulated that the pain in the neck and back of these subjects is related to a mechanical dysfunction, which osteopathic practitioners would expect to relieve by manual treatment. The questionnaire relating to back pain (no. 2.) showed that the patient groups' scores (figure 5) improved more than the control group (figure 4), which was the anticipated result considering the control group were not able to receive manual treatment for the duration of their involvement in the project.

The Beck Depression Inventory (Questionnaire no. 3), produced an interesting result. The scores in figure 4 clearly demonstrate that the depression level of the control group underwent a steady decrease. This particular result differs greatly from the other symptoms of the control group, which all deteriorated during the period of the project, and corroborates the claim that ME is not a depressive disorder. Otherwise other symptoms would have shown some improvement as the depression levels dropped. In some of the control subjects, this may be due to their taking antidepressants. The patient group's depression score initially improved as the treatment started to take effect. However, after six months the level of depression was slightly worse than at the three month period. The reason for this was possibly due to the fact that the rate of overall improvements in symptoms had reduced. This is evident from figure 5 which shows a far greater reduction of symptoms in the first three months than during the second period. The patients may have lost confidence in the treatment at this halfway stage, which increased their feeling of depression. This may equally explain why patient group anxiety levels, and sleep disturbance rose slightly in the second quarter. However, at the 6 months stage the depression, anxiety and sleep levels were all less than at the start of the project.

Broadbent's Cognitive Function Questionnaire, no. 6 produced significant results. The cognitive abilities of the control group (figure 4) gradually worsened over the first six months. Whereas the patient score increased during the same period. This reduction in congitive ability is very disturbing for the ME patient. The reasons for this dysfunction have been studied at length [49]. It is thought to be due to a reduced cerebral blood flow [50] which is predominantly controlled by the quantity of hydrogen ions in the cerebrospinal fluid. The blood flow is raised by an increase of blood carbon dioxide or by a reduction in the blood oxygen levels. The cerebral circulatory system has a strong sympathetic innervation that passes upward from the superior cervical ganglia. This innervation supplies both the large superficial arteries and the small arteries that infiltrate into the substance of the brain. It has long been thought that the sympathetic nerves play no role in regulating cerebral blood flow. Nevertheless, experiments have shown that the cerebral sympathetic stimulation can, under some conditions, markedly constrict the cerebral arteries. For instance, when the arterial pressure rises to a high level during strenuous exercise and other activities, the sympathetic nervous system constricts the large and intermediate sized arteries to prevent high blood pressure reaching the smaller blood vessels, thus preventing strokes. Also sympathetic reflexes are believed to cause vasospasm in the intermediate and large arteries in some instances of brain damage, e.g. after a cerebral stroke or in cases of subdural haematoma, or brain tumour [51]. It is thus feasible that the lack of normal cognitive function and disturbance of cerebral activity could be attributed to a dysfunction of sympathetic control mentioned above, which in turn leads to reduced cerebral circulation.

The results of the final questionnaire no. 8 (The Profile of Fatigue Related States) are significant, as this was the only established and tested questionnaire developed specifically for the symptoms associated with ME [46]. The overall improvement in PFRS scores of the patient group (figure 5) compared with the control group (figure 4) adequately demonstrated the validity of the treatment programme.

The tests involving measurement of work done by the knee extensor muscles (table 3) clearly demonstrated that there was a considerable improvement in fatigue resistance in the patient group.

A previous study on patients with ME [52] has demonstrated that the reduced capacity for dynamic exercise in this case is also associated with reaching exhaustion more rapidly than in normal subjects, at which point these patients have relatively reduced intracellular concentrations of ATP. The study concluded that there was a defect in oxidative metabolism with a resultant acceleration of glycolysis in the skeletal muscles of an ME sufferer. If those authors are correct, then impaired blood flow is a possible explanation for a reduced oxygen supply to the muscle resulting in the fatigue symptoms of ME.

It should be noted that in the present study, reduced fatiguability of the quadriceps was not achieved by any direct treatment on the lower extremity, nor by any exercise regime to improve muscle strength in the patients legs. The treatment programme was based solely on the hypothesis that by using manual techniques to reduce disturbed afferent sympathetic impulses, the overall sympathetic nervous system eventually begins to function normally, thus improving visceral function and circulation in skeletal muscle.

A major objective of this study was to determine the correlation between the mechanical dysfunction of the spine, and the incidence of the symptoms linked with ME. Physiological evidence supports the possibility that the clinical findings of thoracic spinal dysfunction, may be intrinsically linked in the pathogenesis of this disorder.

It has been demonstrated that postural changes in the spine produced alterations in the production of perspiration [53]. A team of physiologists developed a hypothesis relating to sympathetic nerve involvement in disease processes. The first conclusions that they reached were as follows.

(a) The manifestations of altered sympathetic activity represent an actual defect in the patterns of sympathetic activity.

(b) These distortions are due to effects of impulses originating from either the viscera, or somatic sources.

(c) Other components, such as adaptive or pathological changes and altered excitability within the central nervous system, may eventually become involved. This may directly affect local tissue without taking the expected route of nerve impulses.

Further studies revealed that the areas of altered sympathetic activity appeared in apparently normal subjects. It was suggested that this was due to subclinical bombardment of nerve impulses into the spinal cord. These impulses caused no symptoms themselves but, added to other stimuli affecting the same spinal segment, they could combine to cause major problems. Long lasting hyperactivity of innervating sympathetic pathways, seemed to be a prevailing theme in many clinical conditions. It was also suggested that spinal dysfunction would lead to disturbances in the muscular fatiguability, sensory, excitability, immunological mechanisms and endocrine functions due to an impairment in normal sympathetic efferent flow [30]. If these findings were correct then we would expect to find that the severity of symptoms depends upon the amount of spinal mobility. The present results of the spinal mobility test (see table 4) suggest that there is little correlation between thoracic spinal movement and ME symptoms.

Our hypothesis, based on clinical evidence, is that following osteopathic treatment the symptoms are reduced due to stabilizing *afferent* sympathetic flow. It is believed by the authors that this equilibrium may be achieved due to relaxation of soft tissue and an improvement in visceral function plus increased blood and lymph circulation.

Conclusion

This present study has revealed a demonstrable improvement in ME symptoms as a result of osteopathic treatment. In future studies we hope to examine which part of the treatment accounts for the aforementioned improvement. Also a year after completing the clinical trial, each patient will undergo a follow-up examination to determine if the improvement in symptoms has been sustained. It is envisaged that a longer term follow-up on these subjects will take place over a period of 5 years.

The findings of the present research indicate a need to examine the symptoms associated with ME from a biomechanical viewpoint.

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