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ORIGINAL ARTICLE

Relative immediate effect of ischaemic compression and activator trigger point therapy on active upper trapezius trigger points: A randomised trial

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Summary

Background: Trigger points are a common cause of severe and disabling pain in chiropractic practice. While trigger points may be found in any skeletal muscle the majority are found in the upper trapezius. Relatively few studies have investigated non-invasive treatments for upper trapezius trigger points. Common manual therapy treatments utilized for upper trapezius trigger points in chiropractic include manual pressure and myofascial release. The purpose of this study was to compare the effect of a single treatment of ischaemic compression and activator trigger point therapy on active upper trapezius trigger points.

Methods: Fifty-two subjects with active upper trapezius trigger points met the participation criteria and were randomised to an ischaemic compression or activator trigger point therapy group. The primary outcome measure was Patient Global Impression of Change. Secondary outcome measures were an 11-point numerical rating scale for change in pain, and change in pressure pain threshold using an algometer for trigger point sensitivity. While the treating clinician and subjects were not masked to treatment assignment, the examiner was blind to treatment assignment until data analyses were completed. An independent *t*-test was used to compare the groups at baseline on the continuous variables. The Mann—Whitney *U*-test was used to compare the groups at baseline on the non-continuous variables. Relative risk ratios of improvement for the primary and secondary outcome measures were calculated with 95% confidence intervals for clinical significance.

Results: Seventy volunteers were screened with 25 subjects randomised to the ischaemic compression group and 27 to the activator trigger point therapy group. There was no significant difference between the groups in any of the baseline variables. On the primary outcome measure both groups improved (78% of those in the activator group and 72% in the ischaemic compression group). Relative risk for

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improvement of 1.00 suggested that those treated with the Activator instrument were no more likely to improve than those treated with ischaemic compression (95% CI = 0.73—1.37). For the secondary outcome measure of pain reduction 41% of those treated with the Activator instrument improved compared to 36% of those in the ischaemic compression group. Those treated with the Activator instrument were 13% more likely to improve than those treated with ischaemic compression. However this relative risk of 1.13 in favour of the activator group was not significant (95% CI = 0.57—2.26). For the secondary outcome of reduction in trigger point sensitivity 32% of those in the ischaemic compression group improved compared to 30% in the activator group. Those treated with ischaemic compression were 8% more likely to improve; however, the relative risk of 1.08 was not significant (95% CI = 0.48—2.44). As risk of improvement on the outcome measures between the groups was not significantly different, number needed to treat was not calculated.

Conclusion: Based on the primary outcome measure the results suggest that both ischaemic compression and activator trigger point therapy have an equal immediate clinically important effect on upper trapezius trigger point pain.

Background

Myofascial pain syndrome, and its concomitant trigger points (TrPs), have been described using various terms for more than 100 years.[1] More than fifty years ago Bonica[2] contended that TrPs were a common cause of severe, disabling pain in general medical practice. Reviews since that time have reached the same conclusion.[3—6] Various clinical studies have also shown TrPs to be a prevalent condition.[7—12] Further, the upper trapezius muscle is reported to be the muscle most commonly affected with TrPs.[6,8,13—16]

There have been relatively few studies investigating non-invasive treatments for upper trapezius TrPs. These studies have suggested a therapeutic effect when used alone or in combination for such interventions as electrical muscle stimulation, therapeutic ultrasound, electrical nerve stimulation, repetitive magnetic stimulation, hot packs, cervical range of motion exercises, ischaemic compression, spray and stretch, transcutaneous electrical nerve stimulation, sustained stretching, massage, cervical manipulation and trigger point pressure release.[17—27]

In the first of a series of studies investigating various treatments for upper trapezius TrPs, we compared ischaemic compression to trigger point pressure release and placebo ultrasound.[28] Ischaemic compression was found to be more effective than trigger point pressure release and both were more effective than placebo ultrasound. For the next study, we compared the Activator instrument to myofascial band therapy and placebo ultrasound in patients with upper trapezius TrPs.[29] Activator was found to be more effective in reducing pain.

Therefore, the purpose of this current study was to directly compare ischaemic compression and the Activator instrument for the treatment of active upper trapezius TrPs based on relative risk using Patient Global Impression of Change (PGIC), pressure pain threshold (PPT), and levels of pain.

Methods

Participants

Seventy volunteers with upper trapezius TrPs were recruited from the students, faculty and staff of the Anglo-European College of Chiropractic (AECC). The diagnosis of an active upper trapezius TrP was confirmed by the clinician (HG) and then each subject was assessed for eligibility by the examiner (AA) and included in the study if they met the following inclusion criteria: (1) male or female between the age of 18 and 55, (2) presence of unilateral or bilateral active upper trapezius TrPs of not more than 12 weeks' duration, and (3) upper trapezius TrP pain rated at least 4 on an 11-point numerical rating scale (NRS). An active upper trapezius TrP was defined as a tender nodule in a taut band that referred pain in a pattern specific for this muscle and that reproduced the subject's usual pain.[15] Subjects were excluded if they had any of the following: (1) specific neck pain, e.g. radiculopathy, systemic or inflammatory pain; (2) evidence of spinal cord compression; (3) recent neck surgery or trauma; (4) long-term use of corticosteroids; (5) anticoagulant use, e.g. Warfarin; and (6) presence of a blood coagulation disorder.

The study was approved by the AECC Research Ethics Sub-Committee. Each subject received a Study Information Sheet and was asked to sign an Informed Consent Form prior to participation in the study.

Interventions

Ischaemic compression consisted of continuous, perpendicular deep thumb pressure to the identified upper trapezius TrP for 30—60 s. Pressure was released according to which of the following occurred first: a palpable decrease in TrP tension or once 60 s had passed. This sequence was methodologically similar to a chiropractic technique developed earlier by Nimmo.[30]

The Activator adjusting instrument IV has force settings ranging from 1 to 4. For this study a force setting of 3 was used (170 N). To treat the TrP, the Activator instrument was placed perpendicular over the identified TrP and 10 thrusts were delivered, with a rate of one thrust per second.

Outcome measures

The primary outcome measure was Patient Global Impression of Change. This is a seven-point scale (very much improved, much improved, minimally improved, no change, minimally worse, much worse, and very much worse) that has been validated as a standard outcome measure.[31—33] Assessment of overall improvement using this scale is considered to be important as it encompasses side effects and patient expectations, and not merely the effect of treatment. It is generally considered the subject has improved if they mark much improved or very much improved, as these categories have been shown to be valid indicators of clinically important change in longitudinal studies with multiple treatment sessions.[33,34] As the current study involved the effect of one treatment with immediate follow-up, we modified the minimum level for improvement on the PGIC to include minimally improved. Thus, a subject marking minimally improved, much improved, or very much improved was considered to have improved.

Secondary outcome measures consisted of the 11-point numerical rating scale (NRS) for pain and pressure pain threshold for TrP sensitivity. The NRS was used to measure pain pre- and post-treatment with subjects rating their current pain severity where 0 = no pain and 10 = worst pain possible. The scale has demonstrated good criterion validity and, although the NRS and visual analogue scale (VAS) have been shown to be equally sensitive (in their ability to detect change), the NRS is favoured due to its simplicity.[35] Further, Salaffi et al.[34] found the NRS to be more reliable than the VAS.

Farrar et al.[33] found that an average reduction of two points on the NRS represents a clinically important and relevant change. Salaffi et al.[34] also found that a reduction of two points on the NRS from baseline is highly correlated with the highest level of improvement on the PGIC. For these reasons, a reduction of two points on the NRS was used to indicate a clinically meaningful change.

Pressure pain threshold was measured using a pressure pain algometer (PPA). Each subject's active upper trapezius TrP was measured for sensitivity pre-treatment, and after 5 min but within 10 min post-treatment. Algometry is an objective method of quantifying soft tissue tenderness and has been shown to be a useful tool in the assessment of TrPs.[36,37] Pressure pain algometry has also been shown to be a reliable and valid measure of TrP sensitivity.[36,37]

An increase post-treatment on PPT of at least 1 kg/cm^2 was used to indicate a clinically important change. This value was estimated based on previous studies that found a mean difference on PPA of 0.05 kg/cm^2 between the left and right upper trapezius muscles,[36] and a side-to-side difference in another study of 2 kg/cm^2.[38] In addition, a recent study found an increase of at least 1 kg/cm^2 was associated with a statistically significant effect.[25]

Randomisation

The randomisation schedule was generated using the website: http://www.randomisation.com. Sealed opaque envelopes were prepared by the clinician (HG) and numbered consecutively, containing the assigned treatment. Subjects were given the assigned treatment based on the consecutively numbered envelope. The examiner was blind to treatment allocation while the clinician and patient were not. The randomisation scheme was concealed from the examiner until data analyses were complete. The allocation sequence was generated by the clinician (HG), the examiner (AA) enrolled subjects, and HG assigned subjects to their groups. Success of blinding was evaluated by asking the examiner if she was able to determine assignment; she was not able to do so.

Procedure

Prior to the study, forms and procedures were tested over a 4-week period using chiropractic students as test subjects. This also gave the clinician and examiner practice in the procedures to be used and to delineate any problem areas. As this was the third study in a series of treatments for upper trapezius TrPs, prior experience was helpful in this regard as the same clinician was involved in all the studies. The clinician, a registered chiropractor, has 28 years' experience treating myofascial TrPs and is a principal lecturer in myofascial pain medicine.

The examiner also spent many hours (prior to the study) using the PPA to gain experience and to feel comfortable in its use. This examiner was a 4th-year chiropractic student with 5 years' experience in massage therapy and diagnosis/palpation of myofascial TrPs.

Each subject completed an eligibility and medical history form, and those who were eligible then entered the study room with the examiner and filled out the pre-treatment NRS. If their current pain was rated at least 4, they were asked to read an information sheet explaining the study and to sign an informed consent form. The examiner then determined if an active TrP of the upper trapezius muscle existed using the criteria as delineated by Travell and Simons.[15] If more than one active TrP was found, the one that was most tender was used for the study. The area over the TrP was then marked with an X using a skin pencil.

Using PPA, the examiner then measured the PPT of the TrP. The rubber tip of the PPA was placed over the marked TrP, and held perpendicular to the muscle belly with the gauge turned away from the subject. The pressure was gradually increased at a rate of approximately 1 kg/cm^2/s as recommended by Fischer.[39] The subject was asked to indicate when the sensation of pressure changed to that of pain by saying 'yes'. The examiner then released the pressure and the gauge reading was recorded as the pre-treatment measurement of TrP sensitivity. Subjects were not informed of their scores to prevent subject bias influencing the results.

At this point, the examiner exited the room and the treating clinician (HG) entered. The clinician opened the next consecutively numbered envelope and then delivered the randomly assigned treatment to the area marked. Each subject was instructed not to discuss the type of treatment received with the examiner. To further blind the examiner to treatment allocation, 10 impulses were applied to the clinician's hand with the Activator instrument for those subjects not in the activator group. The clinician then exited the room and the examiner re-entered to conduct the post-treatment PPT after 5 min, but no longer than 10 min after treatment. Each subject was asked to rate their current pain on the post-treatment NRS, and to complete the PGIC scale.

Statistical analysis

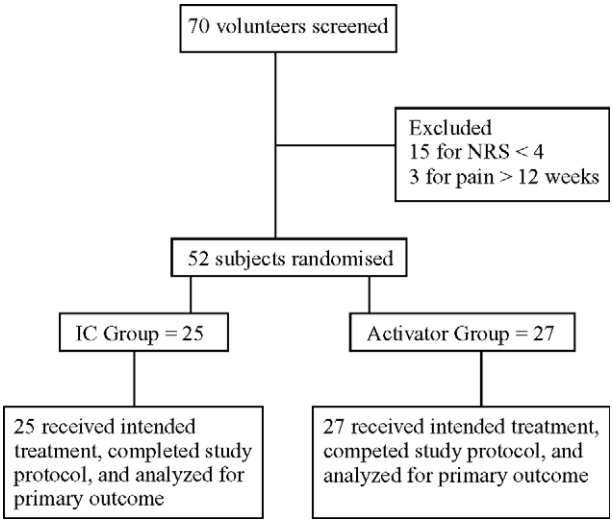
Data analysis was conducted using GraphPad Instat Version 3.0 for Windows 95, GraphPad Software, San Diego, California USA, www.graphpad.com. The independent Student's *t*-test was used to compare the groups at baseline on the continuous variables. The Mann–Whitney *U*-test was used to compare the groups at baseline on the non-continuous variables. Significance for baseline variables was set at $P < 0.05$.

Relative risk ratios for the primary and secondary outcome measures were calculated with 95% confidence intervals (CIs), to determine if a significant difference in the risk of improvement occurred between the two groups. For significant results number needed to treat (NNT) would be calculated.

Within group change from baseline to post-treatment for the secondary outcomes was determined using the dependent Student's *t*-test. For the activator group, on the outcome of PPT, these data failed the normality test, and the Wilcoxon matched-pairs signed-ranks test was used. Significance for change within the groups was set at $P < 0.05$.

Results

Participant flow



Recruitment occurred over 9 weeks during the Autumn term 2007 at AECC. Follow-up occurred within 10 min of the end of treatment. Baseline demographic and clinical characteristics of each group are shown in Table 1. There was no significant difference between the groups in any of the baseline variables ($P > 0.05$).

All participants in the ischaemic compression group (25 of 25) and all participants in the Activator instrument group (27 of 27) were included in the analysis for each outcome measure.

Table 2 shows the number of subjects who had a clinically meaningful change in each group. On the

Table 1 Baseline demographic and clinical characteristics.

Variable/Category	Activator group	Ischaemic compression group
Age (S.D.)	29 (8.5)	28 (9.1)
Gender		
Females	67%	72%
Marital status		
Married	22%	12%
Widowed	0%	0%
Divorced/separated	0%	4%
Not married; in relationship	33%	36%
Never married	45%	48%
Pain onset		
Sudden	18%	24%
Gradual	78%	72%
Due to injury	4%	4%
Weight (S.D.)	72 (15.5)	68 (11.7)
Height (S.D.)	174 (8.9)	170 (10.1)
BMI (S.D.)	23 (3.4)	23 (3.3)
NRS values (S.D.)	5 (0.8)	5 (1.2)
TrP side		
Right	44%	60%
PPT (S.D.)	3 (1.1)	2 (0.9)

Age in years; weight in kg; height in cm; PPT in kg/cm^2.
NRS = Numerical rating scale; PPT = Pressure Pain threshold; BMI = Body mass index.

primary outcome measure of PGIC 72% of the subjects in the ischaemic compression group improved compared to 78% of subjects in the Activator instrument group. Those treated with the Activator instrument were no more likely to improve than those treated with ischaemic compression. Relative risk was 1.00 (95% CI = 0.73—1.37), which was not significant.

Clinical improvement as determined by reduction in pain measured on the secondary outcome measure of the NRS was slightly higher for the activator group with 41% of subjects undergoing a clinically meaningful change compared to 36% for the ischaemic compression group. Those treated with the Activator were 13% more likely to improve than those treated with ischaemic compression. However this relative risk of 1.13 in favour of the activator group was not significant (95% CI = 0.57—2.26).

For the secondary outcome of reduction in trigger point sensitivity 32% of those in the ischaemic compression group improved compared to 30% in the activator group. Those treated with ischaemic compression were 8% more likely to improve than those treated with the Activator instrument. However, the relative risk of 1.08 was not significant (95% CI = 0.48—2.44).

As relative risk for improvement between the groups was not significantly different, number needed to treat was not calculated.

The mean reduction in pain from baseline to post-treatment for the ischaemic compression group was 1.1. (1.9), which was significant ($P = 0.0059$). Mean reduction in pain for the activator group was 1.4 (1.2), which was also significant ($P < 0.001$). The mean increase in PPT for the ischaemic compression group was 0.8 kg (1.1 kg), which was significant ($P = 0.0021$). The median difference between baseline and post-treatment for the activator group on PPT was 0.3 kg (1.3 kg), which was also significant ($P = 0.0463$).

Discussion

To our knowledge, this is the first randomised clinical trial to directly compare the effect of ischaemic compression and the Activator instrument for subacute upper trapezius TrP. Both treatments were equally effective based on patient impression of improvement, decrease in TrP sensitivity, and reduction in pain severity. The results suggest that the immediate effect of a single treatment with ischaemic compression or the Activator instrument to an active upper trapezius TrP produces a clinically meaningful improvement.

Our results confirm the findings of previous studies. Gemmell et al.[28] investigated the immediate effect of ischaemic compression, trigger point pressure release and placebo ultrasound on degree of lateral cervical flexion, neck pain and PPT of upper trapezius TrPs in subjects with non-specific neck pain. Ischaemic compression was found to be superior to trigger point pressure release and placebo

Table 2 Proportion of subjects to undergo a meaningful clinical improvement in each treatment group.

Outcome measure	Activator group ($n = 27$)	Ischaemic compression group ($n = 25$)	RR (95% CI)
PGIC	21 (78%)	18 (72%)	1.00 (0.73—1.37)
NRS	11 (41%)	9 (36%)	1.13 (0.57—2.26)
PPT	8 (30%)	8 (32%)	1.08 (0.48—2.44)

PGIC = Patient Global Impression of Change (primary outcome measure), NRS = Numerical Rating Scale, PPT = Pressure Pain Threshold, RR = Relative Risk.

ultrasound in immediately reducing pain. Blikstad and Gemmell[29] compared the immediate effect of the Activator instrument to myofascial release and placebo ultrasound in a population of neck pain patients and found the activator to be superior.

Fryer and Hodgson[27] compared manual pressure release to placebo in a group of asymptomatic university students and found manual pressure release to be superior. Their definition of manual pressure release correlates with the description of trigger point pressure release in Simons et al.[15] and not ischaemic compression. Further, Fryer and Hodgson's subjects were examined while in the supine position while other studies examined the seated participant. Also, clinical significance was not studied in the Fryer and Hodgson paper. These factors make it difficult to compare their study to the current study.

There are limitations to the current study. With the small sample size the study may have been underpowered. However, the confidence intervals were narrow suggesting that the results may reflect the true risk of improvement in the population. In other words, both treatments may be effective for treating active upper trapezius TrPs. The study only looked at the immediate effect of the treatments and there was no long-term follow-up. Further, based on the clinical experience of the primary author, with multiple treatments over an extended time period (6—8 over 3—6 weeks) a difference between the groups may have been obtained. However, due to time and budget constraints an extended study was not possible. Another concern is that the analgesic effect of the treatments may have been masked by post-treatment soreness. It is possible that ischaemic compression and activator trigger point therapy to tender active TrPs caused irritation and sensitised the TrP to post-tests. We attempted to control for this by waiting 5 min before conducting the post-tests, but this may have not been enough time to rule out post-treatment soreness and the actual treatment effect may therefore be even higher. While we used symptomatic subjects, those subjects were either chiropractic students or chiropractic school staff and faculty, and may not be representative of typical patients presenting to chiropractors.

Strengths of the study include the use of symptomatic subjects with active TrPs and pain of at least 4 on an 11-point NRS. The diagnostic experience of the examiner and the treatment experience of the clinician are also strengths of the study as their experience suggests that the diagnosis and treatment were adequate.

The promising results warrant further studies employing more than one treatment session with longer term follow-up before a decision can be made as to the true effectiveness of these treatments for upper trapezius TrPs.

Conclusions

The results suggest that a single session of ischaemic compression or activator trigger point therapy have an equal and clinically meaningful effect in treatment of active TrPs of upper trapezius muscles.

Competing interest

The authors declare they have no conflicts of interest.

Acknowledgements

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References

1. Skootsky SA, Jaeger B, Oye RK. Prevalence of myofascial pain in general internal medicine practice. *West J Med* 2000;**151**: 157—60.
2. Bonica J. Management of myofascial pain syndromes in general practice. *JAMA* 1957;**164**:732—8.
3. Hong C-Z. Treatment of myofascial pain syndrome. *Curr Pain Headache Rep* 2006;**10**:345—9.
4. Fricton JR. Myofascial pain syndrome. *Neurol Clin* 1989;**7**: 413—27.
5. Yap E-C. Myofascial pain—an overview. *Ann Acad Med Singapore* 2007;**36**:43—8.
6. Grosshandler SL, Stratas NE, Toomey TC, Gray WF. Chronic neck and shoulder pain. Focusing on myofascial origins. *Postgrad Med* 1985;**77**:149—58.
7. Sola AE, Williams RL. Myofascial pain syndromes. *Neurology* 1956;**6**:91—5.
8. Sola AE, Rodenberger ML, Gettys BB. Incidence of hypersensitive areas in posterior shoulder muscles. A survey of two hundred young adults. *Am J Phys Med* 1995;**34**:585—90.
9. Drewes AM, Jennum P. Epidemiology of myofascial pain, low back pain, morning stiffness and sleep-related complaints in the general population. *J Musculoskelet Pain* 1995;**3**(Suppl 1):68.
10. Fishbain DA, Goldberg M, Meagher BR, Steele R, Rosomoff H. Male and female chronic pain patients categorized by DSM-111 psychiatric diagnostic criteria. *Pain* 1986;**26**:181—97.
11. Marklund S, Wanman A. Incidence and prevalence of myofascial pain in the jaw-face region. A one-year prospective study on dental students. *Acta Odontol Scan* 2008;**66**:113—21.

12. Levoska S. Manual palpation and pain threshold in female office employees with and without neck-shoulder symptoms. *Clin J Pain* 1993;**9**:235—41.
13. Gerwin RD. Classification, epidemiology, and natural history of myofascial pain syndrome. *Curr Pain Headache Rep* 2001;**5**:412—20.
14. Sciotti VM, Mittak VL, Di Marco L, Ford LM, Plezbert J, Santi Padri E, et al. Clinical precision of myofascial trigger point locations in the trapezius muscle. *Pain* 2001;**93**: 259—66.
15. Simons DG, Travell JG, Simons LS. *Travell & Simons' myofascial pain and dysfunction, The trigger point manual, Upper half of body*, 2nd ed., vol. 1. Philadelphia: Lippincott Williams & Wilkins; 1999. p. 278—307.
16. Lewis J, Tehan P. A blinded pilot study investigating the use of diagnostic ultrasound for detecting active myofascial trigger points. *Pain* 1999;**79**:39—44.
17. Lee JC, Lin DT, Hong C-Z. The effectiveness of simultaneous thermotherapy with ultrasound and electrotherapy with combined AC and DC current on the immediate pain relief of myofascial trigger points. *J Musculoskelet Pain* 1997;**5**: 81—90.
18. Hsueh T-C, Cheng P-T, Kuan TS, Hong C-Z. The immediate effectiveness of electrical nerve stimulation and electrical muscle stimulation on myofascial trigger points. *Am J Phys Med Rehabil* 1997;**76**:471—6.
19. Majlesi J, Unalam H. High-power pain threshold ultrasound technique in the treatment of active myofascial trigger points: a randomised, double-blind, case—control study. *Arch Phys Med Rehabil* 2004;**85**:833—6.
20. Smania N, Corato E, Fiaschi A, Pietropoli P, Aglioti SM, Tinazzi M. Repetitive magnetic stimulation. A novel therapeutic approach for myofascial pain syndrome. *J Neurol* 2005;**252**: 307—14.
21. Srbely JZ, Dickey JP. Randomized controlled study of the antinociceptive effect of ultrasound on trigger point sensitivity: novel application in myofascial therapy? *Clin Rehabil* 2007;**21**:411—7.
22. Hou C-R, Tsai L-C, Cheng K-F, Hong C-Z. Immediate effects of various physical therapeutic modalities on cervical myofascial pain and trigger-point sensitivity. *Arch Phys Med Rehabil* 2002;**83**:1406—14.
23. Gam AN, Warming S, Larsen LH, Jensen B, Hoydalsmo O, Allon I, et al. Treatment of myofascial trigger points with ultrasound combined with massage and exercise—a randomised controlled trial. *Pain* 1998;**77**:73—9.
24. Jaeger B, Reeves JL. Quantification of changes in myofascial trigger point sensitivity with the pressure algometer following passive stretch. *Pain* 1986;**27**:203—10.
25. Ruiz-Saez M, Fernandez-de-las-Penas C, Blanco CR, Matrinez-Segura R, Garcia-Leon R. Changes in pressure pain sensitivity in latent myofascial trigger points in the upper trapezius muscle after a cervical spine manipulation in pain-free subjects. *J Manipulative Physiol Ther* 2007;**30**:578—83.
26. Hanten WP, Olson SL, Butts NL, Norwicki AL. Effectiveness of a home program of ischaemic pressure followed by sustained stretch for treatment of myofascial trigger points. *Phys Ther* 2000;**80**:997—1003.
27. Fryer G, Hodgson L. The effect of manual pressure release on myofascial trigger points in the upper trapezius muscle. *J Bodyw Move Ther* 2005;**9**:248—55.
28. Gemmell H, Miller P, Nordstrom H. Immediate effect of ischaemic compression and trigger point pressure release on neck pain and upper trapezius trigger points: a randomised controlled trial. *Clin Chiropract* 2008;**11**:30—6.
29. Blikstad A, Gemmell H. Immediate effect of activator trigger point therapy and myofascial band therapy on non-specific neck pain in patients with upper trapezius trigger points compared to sham ultrasound: a randomised controlled trial. *Clin Chiropract* 2008;**11**:23—9.
30. Nimmo RL. A technique for the correction of muscular imbalance of the temporomandibular joints. *The Receptor* 1980;**2**: 1—8.
31. Wyrwich K, Nienaber N, Tierney W, Wolinsky F. Linking clinical relevance and statistical significance in evaluating intra-individual changes in health-related quality of life. *Med Care* 1999;**37**:469—78.
32. Middel B, Stewart R, Bouma J, van Sonseren D, can den Heuvel W. How to validate clinically important change in health-related functional status. Is the magnitude of the effect size consistently related to magnitude of change as indicated by a global question rating? *J Eval Clin Pract* 2001;**7**:399—410.
33. Farrar JT, Young JP, Lamoreaux L, Werth JL, Poole RM. Clinical importance of changes in chronic pain intensity measured on a 22-point numerical rating scale. *Pain* 2001;**94**:149—58.
34. Salaffi R, Stantcanti A, Silvestri CA, Ciapetti A, Grassi W. Minimally clinically important changes in chronic musculoskeletal pain intensity measured on a numerical rating scale. *Eur J Pain* 2003;**8**:283—91.
35. Bolton JE, Wilkinson RC. Responsiveness of pain scales: a comparison of three pain intensity measures in chiropractic patients. *J Manipulative Physiol Ther* 1998;**21**:1—7.
36. Antonaci F, Sand T, Lucas GA. Pressure algometry in healthy subjects: inter-examiner variability. *Scand J Rehabil Med* 1998;**30**:3—8.
37. Nussbaum EL, Downes L. Reliability of clinical pressure-pain algometric measurements obtained on consecutive days. *Phys Ther* 1998;**78**:160—9.
38. Fischer AA. Documentation of myofascial trigger points. *Arch Phys Med Rehabil* 1988;**69**:286—91.
39. Fischer AA. Pressure algometry over normal muscles. Standard values and reproducibility of pressure threshold. *Pain* 1987;**30**:115—26.