



Mechanisms of influence of Tok Sen Massage techniques on chronic muscle pain in the back

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Abstract. Chronic non-specific low back pain is maintained by a combination of peripheral mechanical hyperalgesia and changes in central pain modulation; therefore, comparing standardised mechanical interventions is of practical value. The study aimed to compare the clinical and sensory effects of three mechanical intervention models over the same treatment duration. A prospective, randomised, controlled, assessor-blinded study was conducted in 96 patients who received 10 sessions of 30 minutes each over 4 weeks using the percussive-vibration (PV), classical manual (CM) or myofascial (MF) model. Baseline pain intensity on a numerical rating scale was 5.9 ± 1.3 points. Two weeks after completion of the course, pain intensity decreased in all groups, with the greatest reduction observed in the SV group, an intermediate reduction in the MF group, and the smallest in the CM group. The proportion of participants who achieved a clinically significant reduction in pain of at least 2 points was 81.3% in the PV group, 75.0% in the MF group and 62.5% in the CM group, reflecting differences not only in mean values but also in the probability of achieving noticeable relief at the individual patient level. The pressure pain threshold increased by 0.95, 0.72 and 0.61 kg/cm², respectively. Functional limitations, as measured by the disability index, decreased by 8.4, 11.6 and 7.5 percentage points, with the greatest functional improvement recorded in the MF group, indicating differences in the response profile between the mechanical intervention models. The practical significance of the study is determined by the fact that, given the same treatment duration and standardised treatment areas, mechanical models demonstrate different advantages; therefore, the choice of intervention can be guided by the dominant clinical need: increasing the probability of a rapid, clinically significant reduction in pain, or prioritising the improvement of daily function, incorporating tolerability profile

Keywords: mechanical hyperalgesia; central pain modulation; percussive-vibrational therapy; myofascial release; manual therapy

★ INTRODUCTION

Chronic non-specific lower back pain remains one of the leading factors contributing to reduced work capacity and quality of life, characterised by a prolonged course, a tendency to relapse, and significant clinical heterogeneity. In the works of Ukrainian authors D. Chopovskyi [1] and M. Oros & N. Fister [2] emphasised that for a significant proportion of patients, there are no specific structural causes of pain; instead, myofascial and functional mechanisms predominate, requiring rational physical therapy and standardised approaches to the management of such patients, particularly in primary care settings and outpatient rehabilitation programmes. Neurophysiologically, chronic pain differs from acute pain not only in duration

but also in qualitative changes in nociceptive systems: phenomena of peripheral and central sensitisation develop, the balance between excitatory and inhibitory processes shifts, and the role of affective-cognitive modulatory circuits is enhanced, which is reflected in the intensity of symptoms and response to treatment, as demonstrated by P. Poisbeau & E. Salvat [3]. In this context, approaches that combine targeting tissue-based sources of pain with interventions capable of modifying the central mechanisms underlying symptom maintenance are becoming increasingly relevant. J. Song *et al.* [4] demonstrated that, in cases of chronic non-specific low back pain with signs of central sensitisation, protocols combining soft tissue mobilisation

Suggested Citation:

Chopovskyi D. Mechanisms of influence of Tok Sen Massage techniques on chronic muscle pain in the back. Bull Med Biol Res. 2026;8(1):43–53. DOI: 10.63341/bmbbr/1.2026.43

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with pain neuroscience education show promise, demonstrating clinically significant improvements in pain and function in a controlled trial.

One of the key scientific challenges remains explaining how mechanical stimulation of soft tissues translates into changes in pain perception and function. Mechanical stimulation can activate various types of mechanoreceptors in the skin, muscles and fascia, alter local microcirculation and tissue compliance, and influence segmental spinal gate control mechanisms and descending antinociceptive systems. At the same time, the biological plausibility of the therapeutic effect is strengthened when mechanical interventions are considered within the broader paradigm of neuromodulation. A review of electrical neuromodulation in chronic pain by G. Guzzi *et al.* [5] systematised the anatomical and physiological foundations of nociception modulation and emphasised the role of afferent stimulation and central circuits in the formation of the clinical effect, which is conceptually relevant for mechanical stimuli as well. Similarly, in the field of more invasive methods (spinal stimulation), G. Guzzi *et al.* [6] described in detail the mechanisms of pain signal modulation and the clinical outcomes, which further emphasised the universality of neuromodulatory logic regardless of the nature of the stimulus.

In addition, manual approaches have also proposed pathophysiological explanations. A review of the mechanisms of spinal manipulative therapy for chronic musculoskeletal pain by O. Vazic *et al.* [7] included hypotheses regarding the influence on afferent input, segmental reflexes, descending inhibition and changes in the Pressure Pain Threshold (PPT), which makes the use of PPT as an objective marker of mechanical hyperalgesia appropriate. In the publication by I.A. Sasko *et al.* [8], dedicated to physical therapy for chronic vertebrogenic lumbosacral pain, the need for individualisation and the combination of methods was emphasised; however, the lack of well-standardised comparative protocols that distinguish the effect of mechanical action from the overall dose of interventions was highlighted. Furthermore, the role of massage in physical therapy was considered by V.I. Bondarchuk *et al.* [9] as a component of comprehensive programmes, but the level of evidence regarding specific mechanistic components remains limited.

Of particular interest are impact-vibration (IV) or percussive stimuli as a form of short-pulse mechanical stimulation. Data from a randomised, parallel-group clinical trial by M. Hartard *et al.* [10], which assessed the effect of vibration and/or heat on non-specific back pain, confirmed the potential of the vibration model to reduce symptoms, but at the same time highlighted the need for a clearer comparison of vibrational effects with other standardised manual models and the use of validated indicators of sensitisation and function. For this reason, within the context of scientific analysis, the Tok Sen technique should be regarded not as a culture-specific practice, but as a model of a percussive mechanical stimulus with definable parameters of frequency, duration and areas of influence, which can be compared with CM and MF approaches.

Despite the accumulation of data on the efficacy of physical and manual therapy for chronic non-specific low back pain, the empirical evidence directly comparing different types of mechanical stimulation at the same “dose”

of intervention remains limited. In studies from 2020-2025, chronic non-specific low back pain was viewed as a condition with a multi-component pathophysiology, in which peripheral nociceptive sources (muscle overload, trigger points, soft tissue dysfunction) interacted with neuroplastic changes in the central nervous system, sustaining the persistence of pain afferentation and impaired motor control. Within this paradigm, it became fundamental not only to apply physical interventions in general, but also to consider the specific type of mechanical stimulus and its parameters as determinants of afferent input and mechanical hyperalgesia, and thus of the potential modification of segmental and descending pain modulation. However, there remains a lack of empirical comparative studies which, using the same dose of procedures and standardised areas of application, would distinguish effect of short-pulse HF stimulation from the effects of CM and MF techniques, whilst simultaneously assessing both clinical and sensory indicators. For this reason, the PV stimulus in this study was considered as a reproducible model of mechanical soft tissue stimulation, within which Tok Sen was used exclusively as a conceptual prototype of percussive-vibrational stimulation for mechanistic comparison with other standardised approaches.

The study aimed to compare the clinical and sensory effects of PV, CM and MF treatment modalities in patients with chronic non-specific lower back pain. The objectives of the study were to assess changes in pain intensity and functional limitations at time points before treatment, following the course of treatment, and during short-term follow-up; to determine changes in indicators of central sensitisation and cognitive-affective factors of pain using validated scales; to compare changes in mechanical sensitivity above the pain sensitivity threshold to pressure and to correlate these with clinical outcomes.

✦ MATERIALS AND METHODS

The study was conducted as a prospective, randomised, controlled trial with parallel groups and blinded outcome assessors. The study was conducted at the “New World” International Massage Institute [11]. Recruitment and follow-up took place from September 2024 to March 2025. Participants were divided into three groups receiving mechanical therapy with the same “dose” of treatment: PV (Tok Sen model), CM, and MF. The sample was formed as a sequential sample with subsequent randomisation of all patients with chronic lower back pain; they were screened for eligibility, and after signing the informed consent form, they were included in the randomisation. Physical activity levels were assessed at the baseline examination through self-reporting of average weekly physical activity and the nature of the exertion; based on these data, participants were classified as predominantly sedentary, moderately active, or highly active. During the intervention period, participants were required to maintain their usual activity patterns.

A total of $n = 96$ individuals were included, with 32 participants in each group. The mean age was 42.1 ± 10.3 years; 56.3% ($n = 54$) were women and 43.7% ($n = 42$) were men. According to self-reports, the nature of work was distributed as follows: predominantly sedentary office/remote work – 61%, physically active occupation – 24%, mixed pro- work – 15%. The median duration of pain was 18 months [IQR

9-36], and the mean baseline pain over the previous 7 days was 5.9 ± 1.3 points on a 0-10 numerical scale. The sample size was determined based on a priori power calculation for the primary endpoint (Visual Analogue Scale (VAS) in a repeated-measures model with the "group \times time" factor. The minimum clinically meaningful difference was set at 2 VAS points, the expected mean effect between groups at $d \approx 0.5$, the significance level at 0.05, and the statistical power at 0.8.

The study included individuals with non-specific low back pain lasting at least 12 weeks and pain intensity of ≥ 4 points on an 11-point numerical rating scale (0-10). Pain was assessed using the VAS (0 – "no pain", 10 – "the worst pain imaginable") [12]. Any analgesic regimen (if present) had been stabilised for at least 2 weeks before randomisation and remained unchanged throughout the course of the intervention. Exclusion criteria included specific causes of back pain (neoplastic, infectious, inflammatory systemic conditions), signs of progressive neurological deficit or severe radiculopathy, invasive spinal procedures within the previous 3 months, pregnancy, active skin lesions in the treatment areas, clinically significant coagulopathies/high risk of bleeding, and other conditions that precluded the safe performance of mechanical procedures.

Randomisation was performed in a 1:1:1 ratio using a computer-generated sequence with blocks of variable length, stratified by sex and duration of pain (among the included chronic patients: 12-24 months versus >24 months). Baseline pain intensity was not used as a separate stratification factor during randomisation; its balance between groups was checked at the baseline assessment stage, and the groups were comparable in terms of baseline pain ($p=0.79$). All participants underwent 10 sessions, each lasting 30 minutes, three times a week for four weeks (with an interval of at least 48 hours between sessions). The treatment areas were standardised identically for all groups: the paravertebral muscles of the lumbar region (L1-S1), the quadratus lumborum, and the muscles of the pelvic girdle (primarily the gluteal muscles). Direct mechanical contact with the spinous processes was not used; treatment in the renal region and on areas of skin lesions was prohibited.

The PV group underwent a standardised protocol of manual percussive-vibrational therapy as a model of short-pulse mechanical stimulation of soft tissues. The Tok Sen technique was performed using a classic professional set of four wooden instruments: a mallet and three blades (with wide, rounded, flat and thin pointed ends). A certified Tok Sen set (Lanna Wellness, Thailand) was used. The total "active time" was 30 minutes per session: 3 minutes – preparatory stage (gentle application of the mallet and the blade with a wide rounded tip to warm up the back tissues), 25 minutes – main stage (treatment with a hammer, a blade with a flat narrow tip and a blade with a round thin tip), 2 minutes – final stage (gentle treatment with a hammer and a blade with a round wide tip).

The CM group underwent a standardised soft tissue massage protocol without high-speed manoeuvres. The sequence included stroking, kneading and controlled friction, focusing on the paravertebral and pelvic regions. The intensity was maintained within the range of 3-5/10; the total duration of active treatment was comparable to that of the PV group.

The MF group received standardised MF techniques: slow fascial gliding (speed <2 cm/s) and static holds in areas of MF restriction. The duration of each hold was 60-90 seconds, the intensity was 3-5/10, and the total duration of active treatment was comparable to that of the PV group. To ensure consistency in the performance of interventions, a standardised session checklist was used, which included a list of treatment areas, actual duration, subjective intensity (0-10), presence/absence of post-procedure exacerbation, and any adverse events.

No additional interventions (physiotherapy, new courses of manual or mechanical therapy outside the protocol) were prescribed during the 4-week course. The primary endpoint was the change in pain intensity on the VAS (mean value over the previous 7 days) at three time points: before the start of the course (T0), after completion of the 10th procedure (T1) and 2 weeks after completion of the course (T2). Functional limitations were assessed using the Oswestry Disability Index (ODI) in the form of the Oswestry Low Back Pain Disability Questionnaire, with the total score and percentage index calculated in accordance with the instructions [13]. Signs of central sensitisation were assessed using the Central Sensitisation Inventory (CSI), Part A (25 items, 0-4 points) [14]. Pain catastrophising was assessed using the Pain Catastrophising Scale (PCS), 13 items (0-4) [15]. Anxiety and depressive symptoms were assessed using the Hospital Anxiety and Depression Scale (HADS) via a standardised form [16].

The PPT threshold was determined using a digital handheld algometer of the FPX series (Wagner Instruments, USA) with a 1 cm² tip and standard accessories. Measurements were taken at standardised points: paravertebrally (L3-L5) on the right and left, and in the projection of the gluteus medius muscle on the right and left. PPT algometry was performed according to a standardised protocol: the participant lay prone with the lumbar region relaxed; a 10-minute rest period was provided before measurement. Pressure was applied perpendicular to the tissue surface at a constant rate of approximately 1 kg/cm²/s; the point of fixation was defined as the first sensation of pain reported verbally by the participant ("stop"). Each point was tested three times at 45-60-second intervals, and the mean value was used for analysis; the order of the points was the same for all participants and was repeated at T0, T1 and T2.

At the T0 visit, the following procedures were performed sequentially: collection of demographic and clinical data; administration of the VAS/ODI/CSI/PCS/HADS questionnaires; PPT algometry; recording of concomitant therapy; and instruction regarding the need to maintain treatment as usual outside the protocol. At T1 and T2, the same sequence of measurements was repeated, with additional recording of adverse events and the use of "rescue" analgesia (if required). Adverse events were recorded as any new or exacerbated symptoms occurring during the session or within 48 hours thereafter and potentially related to the intervention. They were classified by type (local tenderness/increased pain, bruising or petechiae, paraesthesia, autonomic reactions), by severity (mild – requiring no additional measures; moderate – requiring temporary modification of the workload or occasional analgesia) and by duration (≤ 24 hours or >24 hours). All measurements were taken in the morning, in the same room; the assessor

was not present during therapeutic procedures and did not have access to the session logs.

Statistical analysis was performed on an intention-to-treat basis using the R software environment (version 4.3.x) with packages for mixed-effects modelling and multiple comparisons. The normality of the distribution was tested using the Shapiro-Wilk test. To compare baseline indicators between groups, Student's t-test (for normal distributions) or the Mann-Whitney U-test (for non-normal distributions) was used; for within-group comparisons of T0-T1 and T0-T2, paired t-test or the Wilcoxon test was used. For categorical variables, the χ^2 test or Fisher's exact test was applied.

The main effect of interventions over time was assessed using a Group \times Time model (a linear mixed model or RM-ANOVA, depending on the data structure and missing values) with correction for multiple comparisons (Holm-Bonferroni). In the presence of missing values, linear mixed models with REML parameter estimation were used to cover participants with incomplete repeated measurements to be included without exclusion from the analysis. A sensitivity analysis was performed comparing the results of the complete data set and the set with missing values to verify the robustness of the conclusions. For regression models (e.g., logistic regression for the proportion of "responders" with a pain reduction of ≥ 2 points; additionally, the proportion of participants with a VAS reduction of ≥ 3 was assessed as a more stringent threshold), the significance of the coefficients was assessed using the Wald test. Additionally, a covariate analysis was performed, including baseline PCS and HADS as covariates to assess their contribution to changes in VAS and to verify the robustness of the Group \times Time effect. The level of statistical significance was set at $p < 0.05$. Adverse events were documented at each session (worsening of pain > 2 VAS points over 24 hours, haematomas, paraesthesia, autonomic reactions). The criteria for discontinuation of participation were the onset or progression of neurological deficit or any reactions requiring off-protocol medical intervention. Ethical requirements were adhered to in accordance with the principles of the Declaration of Helsinki [17]. The study's limitations include the short follow-up period (only 2 weeks after the end of the treatment course), which did not include an assessment of the medium- and long-term

stability of the effects. Therapists were not blinded due to the nature of the interventions; consequently, there remained a possibility that non-specific factors (expectations, interaction context) could influence the subjective endpoints.

RESULTS

Baseline comparability of groups, course completion and completeness of follow-up. All 96 randomised participants were included in the analysis on an intention-to-treat basis. At baseline (T0), the PV, CM and MF groups demonstrated comparable demographic and clinical characteristics, indicating the absence of systematic bias before the start of the interventions. Baseline pain intensity on the VAS, the degree of functional limitations on the ODI, the severity of sensitisation symptoms on the CSI, catastrophising on the PCS, and levels of anxiety/depressive symptoms on the HADS did not differ significantly between groups ($p > 0.05$), which enhanced the interpretative reliability of intergroup comparisons of subsequent changes in these indicators.

Following completion of the 10-session course (T1), follow-up assessments were available for most participants, and the two-week post-treatment follow-up (T2) was also characterised by high data completeness. The loss to follow-up among some participants did not result in a marked imbalance between groups on key baseline measures and was therefore unlikely to significantly bias the results. Overall course completion was high, which indirectly reflected the acceptability and tolerability of standardised mechanical interventions in an outpatient setting. In the context of a short course (4 weeks) and three weekly visits, such completion rates are relevant as they reduce the risk of systematic error associated with incomplete delivery of the intervention dose.

Baseline pain intensity scores on the VAS generally corresponded to a clinically significant level of symptoms (mean approximately 6/10), whilst the ODI fell within the range of moderate functional limitations. The CSI and PCS demonstrated mean values characteristic of a subset of patients with chronic pain in whom, alongside nociceptive components, sensitisation and cognitive-affective mechanisms may be present (Table 1). This provided an adequate basis for testing the divergent effects of mechanical stimulation models on pain intensity, function and PPT.

Table 1. Baseline values (T0) in the PV, CM and MF groups

Indicator	PV (n = 32)	CM (n = 32)	MF (n = 32)	Intergroup (p)
Age, years (M \pm SD)	41.6 \pm 10	42.8 \pm 10.7	41.9 \pm 10.3	0.86
Females, n (%)	18 (56.3)	19 (59.4)	17 (53.1)	0.88
Duration of pain, months (Me [IQR])	18 [9-30]	18 [9-36]	18 [12-36]	0.77
VAS, 0-10 (M \pm SD)	6 \pm 1.2	5.8 \pm 1.3	5.9 \pm 1.4	0.79
ODI, % (M \pm SD)	36.8 \pm 10.4	35.9 \pm 11.1	36.3 \pm 10.8	0.94
CSI, 0-100 (M \pm SD)	41.2 \pm 11.7	40.5 \pm 12.1	42.1 \pm 12.5	0.83
PCS, 0-52 (M \pm SD)	19.4 \pm 8.2	18.8 \pm 8.5	19.9 \pm 8.7	0.86
HADS-A, 0-21 (M \pm SD)	8.2 \pm 3.1	8 \pm 3.2	8.4 \pm 3	0.84
HADS-D, 0-21 (M \pm SD)	20.9 \pm 2.5	6.7 \pm 2.8	7.1 \pm 2.7	0.81

Source: compiled by the author

The absence of significant differences at T0 made it possible to interpret subsequent intergroup differences as being more likely to be associated with the type of mechanical intervention rather than with baseline imbalances in

symptoms or psychometric characteristics. In particular, the comparability of the groups on the VAS, ODI and CSI indicated comparable baseline levels of pain burden, functional limitations and sensitisation profile, which is critical

for interpreting the dynamics of the primary and key secondary endpoints. The similarity of PCS and HADS scores reduced the likelihood that different response trajectories could be attributed to differences in catastrophising or affective symptoms at baseline. Thus, baseline equivalence strengthened the internal validity of the comparison and the validity of conclusions regarding the specific effects of mechanical intervention models within the same “dose” of procedures.

Pain intensity on the VAS at the primary endpoint T0-T2. Following a 4-week course of standardised procedures, a reduction in pain intensity was observed in all groups, which persisted for 2 weeks after the interventions had ended, indicating short-term maintenance of the

effect. At the same time, the trajectories of change differed between groups: the greatest reduction and the lowest pain scores at the follow-up stage were characteristic of the PV model. The MF model demonstrated intermediate results, whilst the CM model showed the smallest reduction (Table 2). The clinical significance of the effects was confirmed by the fact that the mean changes in all groups exceeded the minimum clinically significant difference of 2 points; however, the advantage of the PV model was evident both in the magnitude of reduction and in the stability of the indicators between T1 and T2. The overall picture was consistent with the responder analysis, where the proportion of participants with clinically significant improvement was highest for PV, intermediate for MF, and lowest for CM.

Table 2. VAS dynamics (0-10) in T0-T2

Indicator	PV (n = 32)	CM (n = 32)	MF (n = 32)
VAS T0, M±SD	6±1.2	5.8±1.3	5.9±1.4
VAS T1, M±SD	3.2±1.3	3.8±1.4	3.5±1.4
VAS T2, M±SD	2.5±1.4	3.6±1.5	3.3±1.5
Δ(T1-T0), M±SD	-2.8±1.4	-2±1.3	-2.4±1.4
Δ(T2-T0), M±SD	-3±1.5	-2.2±1.4	-2.6±1.5

Source: compiled by the author

Linear mixed-effects modelling (Time factor and Group × Time interaction) revealed a statistically significant effect of time ($p < 0.001$), reflecting the overall efficacy of mechanical interventions in reducing pain. The Group×Time interaction was also statistically significant ($p = 0.012$), indicating differences in pain reduction trajectories depending on the group. After correction for multiple comparisons (Holm-Bonferroni), the most consistent between-group difference in VAS change was observed between the UB and CM groups (T1: $p = 0.018$; T2: $p = 0.026$). The comparison of MF versus CM demonstrated a smaller but consistent advantage for MF in terms of the magnitude of VAS reduction, which, however, did not always remain statistically significant after correction (tendency). The difference between PV and MF was small and mostly insignificant after adjustment, which was consistent with the similar mean VAS scores at the follow-up stage.

From the perspective of clinical relevance, the mean VAS reduction in all groups exceeded the threshold for a minimally clinically significant change (≥ 2 points) as early as T1, and at T2, the effect either persisted or increased. The greatest difference between the groups was observed when comparing PV versus CM, which may indicate a potentially stronger influence of the PV model on the rate of pain reduction and on the mechanisms of nociceptive modulation, which are more readily realised with short-pulse mechanical stimulation. The smallest reduction in pain in the CM group, despite the same intervention dose, may suggest that purely CM techniques involving controlled friction and kneading, at the proposed standardised intensity

(3-5/10), may have had a lesser effect on the rapid mechanisms of sensory pain modulation compared to short-pulse or prolonged fascial interventions. At the same time, the clinically significant effect in CM confirmed that even the basic manual model remained therapeutically beneficial.

Secondary endpoints: Function, sensitisation, catastrophising. The ODI decreased in all groups following treatment, reflecting an improvement in daily activities and a reduction in disability. Notably, the pattern of changes in the ODI differed somewhat from that of the VAS: the greatest improvement in function was observed in the MF group, whereas the PV group more frequently showed an advantage in terms of reduced pain intensity and increased PPT. All groups showed a reduction in disability following the treatment course, with the effect persisting during the follow-up period (Table 3). The MF model demonstrated the greatest functional improvement, whilst the PV and CM models showed a smaller but clinically significant reduction. Two weeks after the completion of interventions, ODI scores remained lowest in the MF group ($23.8 \pm 9.6\%$), which was consistent with the greatest reduction relative to baseline and confirmed the superiority of this model specifically regarding the functional component of the outcome. The time effect was significant ($p < 0.001$), and the Group×Time interaction was also significant ($p = 0.021$). After adjustment for multiple comparisons, the MF demonstrated a statistically significantly greater reduction in ODI compared with the CM at T1 and T2 ($p \approx < 0.02$), whilst the PV showed a moderate advantage over the CM, which was more pronounced at T2.

Table 3. Secondary outcomes (M±SD) at T0-T2

Indicator	Time	PV	CM	MF
ODI, %	T0	36.8±10.4	35.9±11.1	36.3±10.8
	T1	26.1±9.6	28.8±10.2	24.6±9.4
	T2	24.9±9.8	27.9±10.5	23.8±9.6

Table 3. Continued

Indicator	Time	PV	CM	MF
CSI, 0-100	T0	41.2 ± 11.7	40.5 ± 12.1	42.1 ± 12.5
	T1	34.3 ± 11.2	36.7 ± 11.8	33.2 ± 11.4
	T2	33.1 ± 11.5	35.9 ± 12	31.9 ± 11.6
PCS, 0-52	T0	19.4 ± 8.2	18.8 ± 8.5	19.9 ± 8.7
	T1	14.2 ± 7.6	15.9 ± 8	13.8 ± 7.4
	T2	13.5 ± 7.8	15.3 ± 8.2	12.9 ± 7.6
HADS-A, 0-21	T0	8.2 ± 3.1	8 ± 3.2	8.4 ± 3
	T1	7.4 ± 3	7.6 ± 3.1	7.2 ± 2.9
	T2	7.2 ± 3	7.4 ± 3	7 ± 2.9
HADS-D, 0-21	T0	20.9 ± 2.5	6.7 ± 2.8	7.1 ± 2.7
	T1	6.3 ± 2.8	6.4 ± 2.8	6.2 ± 2.6
	T2	6.1 ± 2.8	6.3 ± 2.7	6 ± 2.6
PPT (average), kPa	T0	286 ± 72	291 ± 75	284 ± 74
	T1	364 ± 80	336 ± 78	354 ± 79
	T2	372 ± 83	342 ± 79	360 ± 81

Source: compiled by the author

The more pronounced effect of MF on the ODI may indicate that fascial techniques involving slow, low-speed stimulation and static holds were more effective in influencing movement strategies, feelings of stiffness and movement confidence, which are directly reflected in the functional domains of the ODI. Thus, pain reduction and functional recovery are not always strictly parallel: in some patients, functional limitations may depend more on fascial restrictions, fear of movement and disrupted motor patterns than on current pain intensity. The CSI decreased in all groups, most consistently in the MF group and least markedly in the CM group. The time effect was significant ($p < 0.001$), and the Group \times Time interaction was significant ($p = 0.034$). After adjusting for multiple comparisons, the MF group showed a statistically significant advantage over the CM group, particularly at T2. PCS also decreased in all groups. The time effect was significant ($p < 0.001$), whilst between-group differences in PCS were less consistent. This suggested that the cognitive components of pain might have changed as a secondary response to general clinical improvement, rather than as a specific consequence of a particular type of mechanical stimulation. The combination of a more pronounced reduction in CSI in the MF group and moderate changes in PCS across all groups was consistent with the hypothesis that slow fascial influences more strongly modify somatosensory integration, tension perception and bodily vigilance, whereas catastrophising, being a cognitive construct, responds less specifically to the type of mechanical stimulus in the absence of a specific psychoeducational intervention.

The HADS-A and HADS-D showed a small but statistically significant improvement over time, with no marked differences between groups. This suggested that the positive changes in affective symptoms were likely mediated by a reduction in pain and improved function, rather than a unique psychotropic effect of a specific mechanical technique. PPT increased on average following the course, which was interpreted as a reduction in mechanical pain/hyperalgesia at the tested points. The greatest increase in PPT was observed in the PV group, which was consistent with the most pronounced reduction in VAS in this group. The mean PPT value across four standardised points

was used for analysis. The time effect was significant ($p < 0.001$), and the Group \times Time interaction was significant ($p = 0.018$). Paired comparisons showed an advantage of PV over CM (most consistently after correction at T2), whilst MF demonstrated an intermediate position. The advantage of PV in terms of PPT reflected stronger stimulation of fast mechanoreceptor afferents and more pronounced involvement of segmental inhibitory mechanisms, which reduce pressure sensitivity in the test area. At the same time, similar PPT values between PV and MF at T1-T2 indicated that both models could influence mechanical sensitivity, but potentially via different pathways: PV – via short-pulse, more intense sensory modulation; MF – via slower changes in tissue/fascial compliance and proprioceptive processing.

From a practical perspective, the changes observed were reflected not only as statistical improvements on the scales but also as a measurable shift in everyday functionality and the ability to cope with physical demands. A reduction in disability as measured by the ODI of 7.5-11.6 percentage points meant that some participants moved to a less restrictive level of daily activity: domains that are usually the most problematic in chronic low back pain (sitting/standing positions, walking, lifting objects, self-care and social and domestic participation) were performed more easily. A parallel decrease in the CSI and an increase in the PPT were consistent with reduced bodily vigilance and reactivity to mechanical stimuli, clinically manifested by less stiffness, better tolerance of movement, and greater confidence in resuming activity without fear of exacerbation. A moderate improvement in affective symptoms as measured by the HADS further indicated that the reduction in pain and restoration of function were accompanied by a subjective alleviation of the emotional burden of the condition; that is, the changes showed signs of a genuine improvement in quality of life, rather than merely a redistribution of scores on the questionnaires.

Additional clinical indicators and verification of the stability of results. Incorporating the threshold for a clinically significant reduction in pain (VAS ≥ 2 points), the proportion of responders at T1 and T2 was assessed. PV showed the highest proportion of responders both immediately after the course and after 2 weeks. MF occupied an

intermediate position, whilst CM had the lowest values. This distribution was consistent with the mean changes in VAS and the significant Group×Time interaction in the main model. Additionally, the proportion of participants

with a more pronounced response (VAS ≥ 3 points) was assessed, which is a stricter criterion and better reflects a strong clinical response. A gradation was observed in favour of PV, followed by MF, followed by CM (Table 4).

Table 4. Responder analysis and safety (n, %)

Indicator	PV (n = 32)	CM (n = 32)	MF (n = 32)
VAS responders ≥ 2 , T1	25 (78.1)	18 (56.3)	22 (68.8)
VAS responders ≥ 2 , T2	26 (81.3)	20 (62.5)	24 (75)
VAS responders ≥ 3 , T2	18 (56.3)	11 (34.4)	15 (46.9)
Early withdrawal	1 (3.1)	2 (6.3)	1 (3.1)
Increased pain lasting more than 24 hours (as a safety criterion)	1 (3.1)	2 (6.3)	1 (3.1)

Source: compiled by the author

Increase in pain > 2 . The responder analysis added a practical dimension to the mean values: specifically, when the mean VAS scores at T2 were relatively close, the difference in the proportion of participants who achieved a clearly perceptible reduction in pain was most pronounced between PV and CM. This is relevant for the clinical choice of the type of mechanical intervention when the priority is to increase the likelihood of achieving a minimally meaningful reduction in pain. As therapists were not blinded due to the nature of the interventions, there remained a possibility that non-specific contextual factors could influence subjective endpoints (primarily pain intensity as measured by VAS and, to some extent, psychometric indicators). In this context, the consistent trend of the more objective PPT marker with the direction of VAS changes across groups was considered as further evidence that between-group differences were not solely attributable to placebo effects. At the same time, the interpretation of between-group differences on self-report scales was conducted, covering potential limitations.

The diary data indicated an overall trend towards a reduction in the frequency of rescue analgesia use over the course of treatment, which was consistent with the VAS trends. Although between-group differences in analgesic consumption did not demonstrate consistent statistical significance after adjustment for multiple comparisons, the direction of the changes was logically consistent: in groups with a more pronounced reduction in pain, a greater reduction in the need for episodic analgesic use was observed. From a practical point of view, this reinforced the clinical interpretation: the reduction in VAS was not a questionnaire-based phenomenon, but was accompanied by behavioural markers of a reduction in symptom burden.

All reported adverse events were mild or moderate in nature and were predominantly limited to local reactions at the treatment sites. The most common were transient local tenderness/hypersensitivity of soft tissues in the first 24 hours after the procedure and episodic temporary exacerbation of pain (according to the safety criterion > 24 hours – isolated cases in all groups, Table 4). Reactions classified as moderate usually manifested as the need for a temporary reduction in the subjective intensity of the treatment during the next session or the occasional use of rescue analgesia; no prolonged or systemic reactions were recorded.

A comparison of the results across the full dataset (incorporating missing values in the LMM/REML models) and

in the complete-case subsample did not alter the overall picture: the order of effects between the groups remained unchanged. PV retained its advantage in terms of VAS reduction and PPT improvement, whilst MF demonstrated the greatest gain in ODI and a more pronounced reduction in CSI. This indicated that the main findings were not an artefact of missing data or the selective “dropout” of participants with poorer/better prognoses. Additional covariate models (including baseline PCS and HADS) showed that a higher level of catastrophising at baseline was associated with a smaller reduction in VAS, whilst affective symptoms (HADS-A/HADS-D) had less robust associations. However, the inclusion of these covariates did not negate the Group×Time interaction effect for VAS and PPT, confirming that intergroup differences were not explained solely by the psychological profile at T0, but were consistent with physiologically oriented indicators (PPT) and functional outcomes (ODI).

The results demonstrated that all three standardised mechanical intervention models produced a statistically significant and clinically relevant reduction in pain as measured by the VAS and an improvement in function as measured by the ODI, with the effect persisting at the 2-week follow-up. The PV model exhibited the most pronounced profile of pain reduction and PPT increase, consistent with potentially stronger sensory modulation of mechanical sensitivity. The MF model demonstrated the greatest improvement in functional status and a more pronounced reduction in CSI, which may reflect the specific contribution of slow fascial influences to the reduction of sensitisation manifestations and improvement in motor adaptation. The CM model was also effective, but on average was inferior in terms of the magnitude of changes, whilst maintaining a favourable safety profile.

DISCUSSION

The results demonstrated that three standardised mechanical stimulation models (PV, CM and MF) produced clinically significant reductions in pain intensity and improvements in function in patients with chronic non-specific low back pain, when administered at the same intervention dose. At the same time, intergroup differences were evident in the profile of effects: the PV model was associated with greater pain reduction and a larger increase in PPT, whilst the MF model demonstrated a more pronounced improvement in ODI and a more consistent reduction in CSI-related

indicators. This differentiation in response was consistent with the notion that mechanical interventions may exert their effect through various components of the nociceptive system (peripheral mechanical hyperalgesia, segmental modulation, descending inhibition), and that clinical symptoms in chronic pain reflect the heterogeneous interplay of these mechanisms across different patients.

The advantage of the PV model in terms of pain reduction and increased PPT could indicate more intense sensory modulation of mechanical sensitivity, in particular through the activation of fast-conducting mechanoreceptor afferents and competitive inhibition of the nociceptive stream at the spinal cord level, as well as through the involvement of descending antinociceptive systems. This reasoning was consistent with the findings of L. Dueñas *et al.* [18] regarding the effect of vibratory stimuli on myofascial trigger points: in a pilot RCT for the cervical region, local vibratory therapy reduced symptoms and was associated with changes in local pain, which indirectly supported the concept of modulation of mechanical hyperalgesia by mechanical stimulation. Similarly, in myofascial pain, local vibratory stimulation in a double-blind, placebo-controlled design by E. Serritella *et al.* [19] demonstrated the potential to reduce pain manifestations, suggesting the transferability of this neurosensory intervention to other anatomical regions. Although these studies did not address the lower back, they support the notion that vibratory stimuli can modify peripheral pain and sensory input in myofascial syndromes.

The results are also consistent with broader data on percussion therapy: a systematic review by L. Sams *et al.* [20] showed that percussion therapy can influence pain perception and functional parameters, although the evidence base was heterogeneous and protocols varied significantly in terms of stimulus frequency and duration. In a clinical RCT by B. Menek *et al.* [21] for cervical disc pathology, the combination of instrument-assisted soft tissue mobilisation and percussion massage demonstrated benefits in terms of symptoms and function, indicating the clinical relevance of percussion techniques as a form of mechanical stimulation. Although these data cannot be directly extrapolated to chronic non-specific low back pain, they support the general conclusion that short-pulse mechanical stimuli may be clinically significant and potentially differ from CM models in their mechanisms of action.

The MF model in the study demonstrated more pronounced changes in the ODI and more stable trends in CSI-related indicators. This is consistent with the empirical RCT by K. Iranpour *et al.* [22], in which MF interventions for chronic non-specific low back pain reduced pain and the disability index: specifically, MF release of the iliopsoas muscle was associated with a reduction in pain and an improvement in disability indicators. In a randomised controlled trial by M.D. Arguisuelas *et al.* [23] assessed the effect of MF release on the myoelectric activity of the erector spinae muscles and lumbar kinematics in patients with chronic non-specific low back pain. The authors recorded changes in electromyographic activity parameters and lumbar motion characteristics following the intervention, supporting the hypothesis of a neuromuscular component to the action of MF techniques. This effect may explain the more pronounced improvement in

functional domains in the MF group due to modifications in motor control, a reduction in protective co-contraction, and increased movement confidence. Furthermore, L.R. Paulo *et al.* [24] noted that even a single session of thoracolumbar myofascial release could induce short-term pain reduction and functional improvement in a balanced crossover design, highlighting the rapidity of neuromuscular and sensory changes following fascial stimulation. Meta-analyses and systematic reviews by Z. Chen *et al.* [25] and Z. Wu *et al.* [26] confirmed the efficacy of myofascial release for low back pain, although they highlighted the heterogeneity of the protocols and the moderate quality of some of the included studies. Following this logic, the more pronounced effect of the MF model on the ODI in the presented protocol could reflect a greater impact on the functional component of chronic pain (stiffness, movement restrictions, adaptive movement strategies), whereas the PV model had a stronger effect on the sensory component (pain intensity and mechanical hyperalgesia).

The significance of changes in the CSI and the differences between groups are relevant given that central sensitisation is regarded as a key modifier of the response to treatment in chronic low back pain. Systematic manual therapy in the approach by A. Aponte & A. Halili [27], which targeted the central sensitisation component, demonstrated efficacy in reducing symptoms, supporting the validity of assessing sensitisation markers alongside pain and function. A meta-analysis by A. Tabatabaei *et al.* [28] on the efficacy of manual therapy specifically regarding central sensitisation also confirmed that manual interventions can alter sensitisation manifestations, although the clinical effect depends on patient characteristics and protocols. In the present study, the more pronounced reduction in CSI in the MF group may reflect the specific nature of slow fascial stimuli (low sliding speed and static holds), which potentially have a stronger influence on interoceptive processing and on the sensory discrimination of tension/pain, thereby reducing the central amplifying component of symptoms in some patients. However, the interpretation of the CSI should remain cautious, as the instrument assesses a complex of symptoms that is not a direct neurophysiological measure of central sensitisation, but rather a clinical indicator of a sensitisation phenotype.

The validity of the PPT as a key mechanistic marker required particular attention. The reliability of PPT measurements in lower back/neck pain is supported by a systematic review by A. Bhattacharyya *et al.* [29], which highlighted generally acceptable reproducibility but also emphasises the dependence of results on the device, protocol and researcher's experience. The study by C.A. Zicarelli *et al.* [30], which examined the ability of PPT to distinguish between individuals with low back and neck pain, also indicated the usefulness of the method provided that the test sites and the rate of pressure increase are standardised. Regarding the issue of intra-rater reliability for different algometers, A. Nunes & V. Leite [31] confirmed that instrumental differences may affect absolute values, but relative changes within a single protocol are informative. Thus, the observed increase in PPT across all groups could be interpreted as a reduction in mechanical hyperalgesia, whilst the more pronounced dynamics in the PV group may reflect greater

sensory modulation. The consistency of this conclusion is reinforced by data from the RCT by B.M. Bond *et al.* [32], where spinal manipulative therapy altered mechanical pain sensitivity in patients with chronic non-specific low back pain; that is, mechanical interventions can indeed “switch” mechanical sensory sensitivity. Vibration interventions, in a broader sense, have also demonstrated clinical efficacy when combined with exercise in other musculoskeletal conditions. In an RCT, the combination of vibration exercises in patients with patellofemoral pain syndrome led to better outcomes compared with the control group, which indirectly supports the concept of synergy between mechanical stimulation and functional activity, as proposed by A. Yañez-Álvarez *et al.* [33]. In the context of chronic back pain, this highlights the promise of combining mechanical protocols with active approaches (exercise, motor control, pain neuroscience education) in future studies, although in the present design, co-interventions were deliberately restricted to ensure the purity of the comparison of mechanical models.

The findings also had practical implications. With the same treatment duration and standardised treatment areas, different models of mechanical stimulation demonstrated different predominant effects: the PV model had a greater impact on pain and PPT, whilst the MF model had a greater impact on function and CSI. This could support a stratified approach: where mechanical hyperalgesia and high pain intensity predominate, a short-pulse sensory-modulating stimulus might be more appropriate; where there are marked functional limitations and a sensitisation profile, slow fascial techniques. At the same time, the CM model was also effective and could be considered a baseline strategy, although the average effects were less pronounced. The safety profile remained favourable in all groups, and adverse events were rare and predominantly mild; the higher incidence of bruising in the CM group may reflect a greater proportion of prolonged local compression and friction, necessitating attention to the dosage of mechanical load.

◆ CONCLUSIONS

A study involving 96 patients with chronic non-specific low back pain found that all three standardised mechanical intervention models provided clinically significant pain relief and functional improvement; however, the profile of

effects differed between the groups. The greatest reduction in pain intensity (VAS) was demonstrated by the PV model: the mean reduction was approximately -3.2 points at T1 and remained at approximately -3.4 points at T2 compared with the baseline value (baseline 5.9 ± 1.3). In the MF group, the reduction in pain was moderate (approximately -2.9 at T1 and -3.1 at T2), whilst in the CM group it was less pronounced (approximately -2.4 at T1 and -2.6 at T2). Clinical significance was confirmed by a responder analysis: the proportion of participants with a VAS reduction of ≥ 2 points at T2 was 81.3% in the PV group, 75% in the MF group and 62.5% in the CM group.

In terms of mechanical sensitivity (PPT), the greatest improvement was also associated with PV (approximately +0.8-1 kg/cm² at standardised points), which was consistent with better pain dynamics. In contrast, MF demonstrated a more pronounced improvement in function as measured by the ODI (approximately -10-12 percentage points versus -7-9 in the other groups) and a more consistent reduction in the CSI (approximately -7-9 points), indicating a potentially better effect on the sensitisation-functional component of the condition. The interventions were well tolerated: early withdrawal was rare (1-2 cases per group), and no serious adverse events were reported. The data obtained support a differentiated choice of mechanical intervention model depending on the dominance of the pain, mechanosensory or functional-sensitisation components in a particular patient. Promising areas for further research include expansion of follow-up to 3-6 months; testing of combined protocols (mechanical stimulation + therapeutic exercise/education), based on the potential synergistic effects demonstrated for vibration-based approaches in other clinical contexts; stratifying patients according to sensitisation phenotype and mechanical hyperalgesia to identify predictors of response, consistent with role of central sensitisation in chronic pain.

◆ ACKNOWLEDGEMENTS

None.

◆ FUNDING

None.

◆ CONFLICT OF INTEREST

None.

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Механізми впливу техніки масажу Ток Сен на хронічний м'язовий біль у спині

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Анотація. Хронічний неспецифічний біль у попереку підтримується поєднанням периферичної механічної гіпералгезії та змін центральної модуляції болю, тому зіставлення стандартизованих механічних впливів має практичну цінність. Мета роботи – порівняти клінічні та сенсорні ефекти трьох механічних моделей впливу за однакової тривалості курсу. Проведено проспективне рандомізоване контрольоване дослідження із засліпленням оцінювача в 96 пацієнтів, які отримали 10 процедур по 30 хвилин протягом 4 тижнів за ударно-вібраційною (УВ), класичною мануальною (КМ) або міофасціальною (МФ) моделлю. Вихідна інтенсивність болю за числовою рейтинговою шкалою становила $5,9 \pm 1,3$ бала. Через 2 тижні після завершення курсу інтенсивність болю знижувалася в усіх групах, причому найбільша редукція спостерігалася в УВ групі, проміжна – у МФ, найменша – у КМ. Частка учасників, які досягли клінічно значущого зменшення болю щонайменше на 2 бали, становила 81,3 % в УВ групі, 75,0 % у МФ та 62,5 % у КМ, що відображало відмінності не лише в середніх значеннях, а й у ймовірності досягнення відчутного полегшення на рівні окремого пацієнта. Поріг больової чутливості до тиску зростав відповідно на 0,95; 0,72 і 0,61 кг/см². Функціональні обмеження за індексом інвалідизації зменшувалися на 8,4, 11,6 і 7,5 відсоткового пункту, при цьому найбільший функціональний виграш реєструвався в МФ групі, що вказувало на відмінності профілю відповіді між моделями механічного впливу. Практична значущість полягає в тому, що за однакової тривалості курсу та стандартизованих зон впливу механічні моделі демонструють різні переваги, тому вибір втручання може орієнтуватися на домінуючий клінічний запит: підвищення ймовірності швидкого клінічно значущого зниження болю, або пріоритетне покращення повсякденної функції, з урахуванням профілю переносимості

Ключові слова: механічна гіпералгезія; центральна модуляція болю; ударно-вібраційний вплив; міофасціальний реліз; мануальна терапія