Osteopathic Manual Treatment in Patients With Diabetes Mellitus and Comorbid Chronic Low Back Pain: Subgroup Results From the OSTEOPATHIC Trial

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**Context:** Chronic pain is often present in patients with diabetes mellitus.

**Objective:** To assess the effects of osteopathic manual treatment (OMT) in patients with diabetes mellitus and comorbid chronic low back pain (LBP).

**Design:** Randomized, double-blind, sham-controlled, 2×2 factorial trial, including OMT and ultrasound therapy (UST) interventions.

**Setting:** University-based study in Dallas-Fort Worth, Texas.

**Patients:** A subgroup of 34 patients (7%) with diabetes mellitus within 455 adult patients with nonspecific chronic LBP enrolled in the OSTEOPATHIC Health outcomes In Chronic low back pain (OSTEOPATHIC) Trial.

**Main Study Measures:** The Outpatient Osteopathic SOAP Note Form was used to measure somatic dysfunction at baseline. A 100-mm visual analog scale was used to measure LBP severity over 12 weeks from randomization to study exit. Paired serum concentrations of tumor-necrosis factor (TNF)-α obtained at baseline and study exit were available for 6 subgroup patients.

**Results:** Key osteopathic lesions were observed in 27 patients (79%) with diabetes mellitus vs 243 patients (58%) without diabetes mellitus (P=0.01). The reduction in LBP severity over 12 weeks was significantly greater in 19 patients with diabetes mellitus who received OMT than in 15 patients with diabetes mellitus who received sham OMT (mean between-group difference in changes in the visual analog scale pain score, −17 mm; 95% confidence interval [CI], −32 mm to −1 mm; P=0.04). This difference was clinically relevant (Cohen d=0.7). A corresponding significantly greater reduction in TNF-α serum concentration was noted in patients with diabetes mellitus who received OMT, compared with those who received sham OMT (mean between-group difference, −6.6 pg/mL; 95% CI, −12.4 to −0.8 pg/mL; P=0.03). This reduction was also clinically relevant (Cohen d=2.7). No significant changes in LBP severity or TNF-α serum concentration were associated with UST during the 12-week period.

**Conclusion:** Severe somatic dysfunction was present significantly more often in patients with diabetes mellitus than in patients without diabetes mellitus. Patients with diabetes mellitus who received OMT had significant reductions in LBP severity during the 12-week period. Decreased circulating levels of TNF-α may represent a possible mechanism for OMT effects in patients with diabetes mellitus. A larger clinical trial of patients with diabetes mellitus and comorbid chronic LBP is warranted to more definitively assess the efficacy and mechanisms of action of OMT in this population.
Diabetes mellitus affects approximately 26 million persons (8%) in the United States, including an estimated 7 million persons in whom the disease is undiagnosed. Type 2 diabetes mellitus (T2DM) comprises up to 95% of US adults with diagnosed diabetes mellitus. Moreover, the prevalence of T2DM in children and adolescents is increasing as a consequence of the continuing rise in obesity in this population. Early identification and treatment may help control diabetes mellitus and prevent or delay such associated consequences as blindness, kidney damage, and lower-limb amputations. Such interventions may also help contain the costs of diabetes mellitus, which were estimated to be $174 billion in the United States, including both medical expenditures and costs associated with lost productivity.

Osteopathic philosophy is based on 4 key principles: (1) the body is a unit; (2) the body possesses self-regulatory mechanisms; (3) structure and function are reciprocally interrelated; and (4) rational therapy is based on an understanding of body unity, self-regulatory mechanisms, and the interrelationship of structure and function. Osteopathic manual treatment (OMT) may be used to alleviate somatic dysfunction, which is defined as impaired or altered function of related components of the somatic (body framework) system: skeletal, arthrodial, and myofascial structures, and related vascular, lymphatic, and neural elements. Osteopathic philosophy also maintains that visceral disorders may have somatic manifestations caused by underlying disease processes. Such somatic manifestations of visceral disorders have been termed viscerosomatic reflexes (VSRs) and are identified by osteopathic palpatory examination for TART criteria (ie, tissue texture abnormalities, asymmetry of landmarks, restriction of motion, and tenderness) or Chapman reflex points. In addition, osteopathic philosophy maintains that OMT of VSRs may have a specific impact on the underlying disease processes via reciprocal somatovisceral reflexes.

The scant osteopathic literature on diabetes mellitus appears to support both the existence of pancreatic VSRs in individuals with type 1 diabetes mellitus and the purported benefits of OMT specifically targeted at such VSRs. Bandeen reported crude data on the temporal relationship between pancreatic stimulatory and inhibitory OMT techniques and alterations in blood glucose levels in patients with and without diabetes mellitus. A contemporary reanalysis of these data revealed that pancreatic stimulatory techniques decreased blood glucose levels within 30 to 60 minutes, whereas pancreatic inhibitory techniques increased blood glucose levels within this time frame. More recently, it was hypothesized that tissue texture abnormalities at the level of the thoracic (T) 11 through lumbar (L) 2 spinal segments are VSRs indicative of nephropathy in patients with T2DM. The augmented VSR responses observed in patients with T2DM of longer duration and comorbid hypertension strengthened the argument for causality between underlying renal pathology and palpated tissue texture abnormalities at the T11-L2 spinal segmental levels.

Patients with T2DM often have other comorbid conditions, such as obesity, that may predispose them to developing somatic dysfunction and chronic pain that is not caused by T2DM-specific disease processes. Chronic daily pain is highly prevalent in patients with diabetes mellitus. Chronic pain also adversely impacts aspects of diabetes mellitus self-management, including diet, exercise, and medication adherence. The tenets of osteopathic medicine lend themselves to a proactive approach to the treatment of patients with diabetes mellitus. The purpose of the present study was to use data from the OSTEOPATHic Health outcomes In Chronic low back pain (OSTEOPATHIC) Trial to further explore the potential benefits of OMT and ultrasound therapy (UST) in the management of chronic low back pain (LBP) in patients with diabetes mellitus.
Methods

Overview of the OSTEOPATHIC Trial
The OSTEOPATHIC Trial was approved by the Institutional Review Board at the University of North Texas Health Science Center in Fort Worth, Texas, and was registered with ClinicalTrials.gov (NCT00315120). Methodologic aspects of the trial have been reported in detail elsewhere. The trial was conducted between August 2006 and January 2011 in Dallas-Fort Worth, Texas, using a randomized, double-blind, sham-controlled, 2×2 factorial design to study the efficacy of OMT and UST in patients with nonspecific chronic LBP. Patients were aged 21 to 69 years and did not have any of the following: “red flag” conditions; a history of recent low back surgery, receipt of worker’s compensation benefits, or ongoing litigation involving back problems; medical conditions that might impede OMT or UST protocol implementation; corticosteroid use in the past month; or clinical evidence of lumbar radiculopathy as determined by the presence of ankle dorsiflexion weakness, great toe extensor weakness, impaired ankle reflexes, loss of light touch sensation in the medial, dorsal, and lateral aspects of the foot, or shooting posterior leg pain or foot pain on ipsilateral or contralateral straight-leg raising.

Assessment of Diabetes Mellitus
Patients were queried about a diagnosis of diabetes mellitus using a standard checklist of 33 diseases or medical conditions prior to randomization. Specifically, patients were asked whether they currently had any of the listed diseases or whether any of the diseases had been diagnosed within 3 months prior to screening for trial eligibility. The checklist did not differentiate between type 1 diabetes mellitus or T2DM. Generally, unless deemed critical to determining trial eligibility, no further attempt was made to confirm patients’ self-reported diagnoses through examination of medical records.

Measurement of Somatic Dysfunction and LBP
The methodology used for osteopathic structural examination performed prior to randomization has been previously described. The musculoskeletal table of the Outpatient Osteopathic SOAP Note Form was used to record the severity of somatic dysfunction based on TART criteria. The severity scale consisted of 4 levels, including the following descriptors: none (no somatic dysfunction or background level); mild (more than background level; minor TART elements); moderate (obvious TART elements—in particular, restriction of motion and/or tissue texture abnormality, with or without symptoms); and severe (key lesion present; significant; symptomatic; restriction of motion and/or tissue texture abnormality stands out with minimum search or provocation). We focused on the severity of somatic dysfunction in the following anatomic regions: T10-12; ribs; lumbar; sacrum/pelvis; and pelvis/innominate. The severity of LBP was measured at baseline and throughout the study with a 100-mm visual analog scale (VAS) anchored at 0 mm (no pain) and 100 mm (worst possible pain).

Measurement of Cytokine Serum Concentrations
Measurements of cytokine serum concentrations were obtained for patients who were randomized after November 2009. Blood samples were collected 30 minutes prior to the first treatment and at the exit visit at week 12, which occurred 4 weeks after the final treatment session. The laboratory protocol for measuring serum concentrations of interleukin (IL)-1β, IL-6, IL-8, IL-10, and tumor necrosis factor (TNF)-α has been described in detail elsewhere.

The OMT and Sham OMT Protocols
The OMT and sham OMT interventions were provided at weeks 0, 1, 2, 4, 6, and 8 using an algorithmic approach. Fifteen minutes were allocated for these interventions at each treatment session. The OMT protocol
concluded that the number of OMT sessions attended by subjects varied, with a median of 6. The OMT procedures consisted of 6 commonly used techniques aimed at the lumbosacral, iliac, and pubic regions. Sham OMT involved hand contact, active and passive range of motion, and sham techniques that simulated OMT. These sham OMT techniques were aimed at the same anatomic regions as active OMT. A similar sham OMT protocol achieved a robust placebo response in the North Texas Chronic Low Back Pain Trial, compared with other placebo treatments for pain. This sham OMT protocol has been adopted by others to deliver sham manipulation. Additional details on both the active and sham OMT protocols have been published elsewhere.

**The UST and Sham UST Protocols**

The UST intervention was delivered after the OMT intervention, using the Sonicator 730 (Mettler Electronics Corp) with a 10-cm² applicator at an intensity of 1.2 W/cm² and a frequency of 1 MHz in continuous mode. Conductivity gel was used to enhance absorption and produce deep-muscle thermal effects. An area of approximately 150 to 200 cm² of the lower back was treated. Sham UST was delivered in the same manner at a subtherapeutic intensity of 0.1 W/cm².

**Statistical Analysis**

The baseline characteristics of patients were summarized using descriptive statistics. We dichotomized the severity of somatic dysfunction by combining the 3 lowest levels (none, mild, and moderate) in contrast with the highest level (severe), which represented the presence of a key osteopathic lesion. Such key lesions are important because they maintain a dysfunctional pattern that includes other secondary dysfunctions. Preliminary analyses of the baseline cytokine serum concentrations revealed that only TNF-α was normally distributed and potentially amenable to meaningful statistical analysis, given the small number of patients in the diabetes mellitus subgroup. Statistical analysis of results for patients in the diabetes mellitus subgroup subsequently failed to reject the hypothesis of normality for changes in either LBP severity or TNF-α serum concentration over 12 weeks. General linear modeling for a change in LBP severity also failed to detect statistically significant interaction between OMT and UST (such modeling was not feasible for changes in TNF-α serum concentration because of more severe sample size constraints). Consequently, we used the Student t test to assess OMT and UST effects in reducing LBP severity and in bringing about changes in TNF-α serum concentration in the subgroup of patients with diabetes mellitus. We further investigated the relationship between changes in both LBP severity and TNF-α serum concentration over 12 weeks using a scatterplot and simple linear regression. Database management and analyses were performed using the SPSS Statistics Version 20 software package (IBM Corporation). Hypothesis testing was conducted at the .05 level of statistical significance. The clinical relevance of statistically significant findings was further assessed using Cohen d, which was computed as the between-group difference in mean changes in a given variable over 12 weeks divided by the standard deviation of the variable at baseline among patients with diabetes mellitus. Cohen d was interpreted using effect size guidelines established by the Cochrane Back Review Group, with any finding exceeding the threshold for a medium (Cohen d=0.5) or large (Cohen d=0.8) effect considered to be clinically relevant.

**Results**

A total of 34 (7%) of the 455 patients self-reported a diagnosis of diabetes mellitus. Baseline patient characteristics according to diabetes mellitus status are presented in Table 1. Patients with diabetes mellitus were statistically significantly older than those without diabetes mellitus, and they were more likely to report comorbid hypertension, osteoarthritis, and depression. Patients with diabetes mellitus were also more likely to report previous hospitalization for LBP, greater back-specific disability, poorer general health, and more frequent use of prescription drugs for LBP than patients without diabetes mellitus at baseline. Although LBP...
severity was greater in patients with diabetes mellitus compared with their counterparts without diabetes mellitus, the difference was not significant (P=.10). A total of 27 patients (79%) with diabetes mellitus had a key osteopathic lesion vs 243 patients (58%) without diabetes mellitus (P=.01). The baseline characteristics of patients with diabetes mellitus according to treatment group are presented in Table 2. Overall, these results indicate that the original randomization was successful in achieving comparable treatment groups for assessment of OMT and UST main effects in patients with diabetes mellitus.

The changes in LBP severity over 12 weeks in patients with diabetes mellitus are displayed in Figure 1. The 19 patients in the OMT group reported significant

<table>
<thead>
<tr>
<th>Characteristic, No. (%)</th>
<th>Without Diabetes Mellitus (n=421)</th>
<th>With Diabetes Mellitus (n=34)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>40 (12)</td>
<td>52 (12)</td>
<td>&lt;.001*</td>
</tr>
<tr>
<td>Women</td>
<td>258 (61)</td>
<td>26 (76)</td>
<td>.08</td>
</tr>
<tr>
<td>Completed College Ed.</td>
<td>187 (44)</td>
<td>13 (38)</td>
<td>.48</td>
</tr>
<tr>
<td>Employed Full Time</td>
<td>203 (48)</td>
<td>12 (35)</td>
<td>.15</td>
</tr>
<tr>
<td>Medically Uninsured</td>
<td>156 (37)</td>
<td>7 (21)</td>
<td>.054</td>
</tr>
<tr>
<td>Current Smokers</td>
<td>112 (27)</td>
<td>7 (21)</td>
<td>.44</td>
</tr>
<tr>
<td><strong>Comorbid Conditions</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>52 (12)</td>
<td>19 (56)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Osteoarthritis</td>
<td>27 (6)</td>
<td>6 (18)</td>
<td>.03*</td>
</tr>
<tr>
<td>Depression</td>
<td>75 (18)</td>
<td>15 (44)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Chronic LBP for &gt;1 y</td>
<td>208 (49)</td>
<td>20 (59)</td>
<td>.29</td>
</tr>
<tr>
<td>Previously Hospitalized</td>
<td>15 (4)</td>
<td>6 (18)</td>
<td>.003*</td>
</tr>
<tr>
<td>Previously Had Surgical</td>
<td>8 (2)</td>
<td>2 (6)</td>
<td>.17*</td>
</tr>
<tr>
<td>VAS Score for LBP, mean</td>
<td>43 (22)</td>
<td>49 (25)</td>
<td>.10*</td>
</tr>
<tr>
<td>Roland-Morris Disability</td>
<td>6 (5)</td>
<td>10 (6)</td>
<td>&lt;.001*</td>
</tr>
<tr>
<td>SF-36 General Health</td>
<td>69 (20)</td>
<td>47 (19)</td>
<td>&lt;.001*</td>
</tr>
<tr>
<td>Used Drugs for LBP in</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous 4 wk</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonprescription</td>
<td>209 (50)</td>
<td>13 (38)</td>
<td>.20</td>
</tr>
<tr>
<td>Prescription</td>
<td>47 (11)</td>
<td>12 (35)</td>
<td>&lt;.001*</td>
</tr>
<tr>
<td>Key Osteopathic Lesion</td>
<td>243 (58)</td>
<td>27 (79)</td>
<td>.01</td>
</tr>
<tr>
<td>TNF-α Serum Concentration, mean (SD), pg/mL</td>
<td>6.2 (2.6)</td>
<td>5.9 (2.5)</td>
<td>.79</td>
</tr>
</tbody>
</table>

* Data presented as No. (%) unless otherwise indicated.
* P values are based on the Mann-Whitney U test, because the baseline scores were not normally distributed.
* P values are based on the Fisher exact test.
* A 100-mm visual analog scale (VAS) was used to measure low back pain (LBP), with higher scores indicating greater pain severity.
* A 24-point Roland-Morris Disability Questionnaire was used to measure back-specific functioning, with higher scores indicating greater disability.
* A 100-point Medical Outcomes Study Short Form-36 Health Survey (SF-36) general health scale was used to measure general health, with higher scores indicating better health.

Abbreviations: OMT, osteopathic manual treatment; SD, standard deviation; TNF, tumor necrosis factor.
criteria. The between-group difference in VAS pain score changes for the 17 patients in each of the UST and sham UST groups was not significant (mean, 0 mm; 95% CI, −16 mm to 17 mm; \( P = .96 \)).

Paired measures of TNF-α serum concentration were available for a subset of 6 patients with diabetes mellitus, including 4 who were assigned to receive OMT and reductions in LBP during this period, whereas the 15 patients in the sham OMT group did not. The between-group difference in VAS pain score changes was significant (mean, −17 mm; 95% confidence interval [CI], −32 mm to −1 mm; \( P = .04 \)). This difference corresponded to Cohen \( d = 0.7 \), an effect size that is classified as medium based on the Cochrane Back Review Group criteria.
who were assigned to receive sham OMT. The changes in TNF-α serum concentration over 12 weeks are shown in Figure 2. Patients in the OMT group had a significant reduction in TNF-α serum concentration, whereas patients in the sham OMT group did not. The between-group difference in changes in the TNF-α serum concentration over 12 weeks was significant (mean, −6.6 pg/mL; 95% CI, −12.4 pg/mL to −0.8 pg/mL; \(P = .03\)). This difference corresponded to Cohen \(d = 2.7\), an effect size that is classified as large based on the Cochrane Back Review Group criteria. The between-group difference in TNF-α serum concentrations for patients in the UST and sham UST groups was not significant (mean, −5.6 pg/mL; 95% CI, −13.1 pg/mL to 1.8 pg/mL; \(P = .10\)). A scatterplot showing changes in both LBP severity and TNF-α serum concentration over 12 weeks is displayed in Figure 3. The corresponding linear regression plot explained a fair amount of the variance in the change in LBP severity (\(R^2 = 0.28\)). However, the change in TNF-α serum concentration was not a significant explanatory variable in the regression model because of the small sample size used in the present analysis (\(n = 6\); \(P = .28\)). The distribution of patient data points according to treatment dyad generally reflects the changes in LBP severity and TNF-α serum concentration described above.

**Comment**

Patients with diabetes mellitus demonstrated a trend toward greater LBP severity and statistically significantly greater back-specific disability and poorer general health than patients without diabetes mellitus at baseline. Moreover, these between-group differences may have been attenuated because patients with diabetes mellitus were significantly more likely to be using prescription drugs for LBP than their counterparts without diabetes mellitus.
Previous studies\textsuperscript{9,10} have found chronic pain to be common in patients with diabetes mellitus. The present study indicates that severe somatic dysfunction, as manifested by a key osteopathic lesion, is also found more often in patients with diabetes mellitus than in those without diabetes mellitus.

The results of the present study may have important implications for the osteopathic medical management of diabetes mellitus, by further corroborating that findings from osteopathic palpatory assessment—in particular, tissue texture abnormalities—are associated with diabetes mellitus.\textsuperscript{3,22,23} The high prevalence of key osteopathic lesions in patients with diabetes mellitus and comorbid chronic LBP, whether specifically or not specifically attributable to the pathophysiologic processes of diabetes mellitus, provides a strong rationale for using OMT in these patients. Overall data from the OSTEOPATHIC Trial have demonstrated that the number of key osteopathic lesions is significantly associated with LBP severity and back-specific disability in a general population of patients with nonspecific chronic LBP,\textsuperscript{24} and that OMT provides statistically significant and clinically relevant reductions in LBP severity in this population.\textsuperscript{25} The present study indicates that these general findings may be extended to patients with diabetes mellitus. Furthermore, based on the subgroup patient characteristics (Table 1) and results (Figure 1) presented herein, the magnitude of association with somatic dysfunction and with clinical response to OMT appears to be significantly greater in patients with diabetes mellitus than in those without diabetes mellitus.

It has been shown that TNF-\( \alpha \) induces insulin resistance in the skeletal muscle of healthy patients via inhibition of Akt substrate 160 phosphorylation.\textsuperscript{26} Circulating TNF-\( \alpha \) has been associated with insulin resistance even after controlling for obesity and other markers of inflammation, and the level of TNF-\( \alpha \) protein in muscle was elevated in patients
with T2DM, particularly in type 2 muscle fibers.\textsuperscript{27} However, the TNF-α serum concentration remained unchanged after weight loss in morbidly obese patients with insulin resistance syndrome, despite significant reductions in the concentration of other inflammatory proteins.\textsuperscript{28} It is particularly interesting to note that, in our patients with diabetes mellitus who received OMT, TNF-α serum concentration decreased significantly over 12 weeks (mean change in TNF-α serum concentration, \(-4.0\) pg/mL; 95% CI, \(-7.7\) pg/mL to \(-0.4\) pg/mL), albeit in a small sample of 4 patients. By comparison, the change in TNF-α serum concentration over 14 months following vertical-ring gastroplasty in 37 patients with insulin resistance syndrome was not significant (mean change in TNF-α serum concentration, \(-2.1\) pg/mL; 95% CI, \(-6.7\) pg/mL to \(2.5\) pg/mL).\textsuperscript{28}

The present study has several potential limitations. First, the study was not powered to conduct subgroup analyses such as those described herein. Thus, it is possible that the significant associations involving diabetes mellitus may simply have been coincidental and that associations between diabetes mellitus and other factors may have been obscured because of insufficient sample size. Second, subgroup analyses are more vulnerable to confounding than are planned overall analyses in clinical trials because study variables may no longer be distributed at random within the subgroups.\textsuperscript{29} Consequently, unknown confounders may have biased our results and conclusions. Nevertheless, there was no significant difference between the OMT and sham OMT groups with regard to any baseline patient characteristic (Table 2). Third, despite well-accepted guidelines for the diagnosis of diabetes mellitus, the possibility of misclassification of diabetes mellitus status exists because self-reported diagnoses were not systematically corroborated by information in patient medical records. Our results most likely reflect the experience of patients with T2DM on the basis of disease prevalence; however, we cannot definitively confirm this because patient medical records were not routinely accessed and reviewed. Fourth,
although we sought to include only patients with chronic LBP of nonspecific etiology in the OSTEOPATHIC Trial, it is possible that some patients with diabetes mellitus may have had LBP of a specific etiology. For example, patients with T2DM have increased levels of somatic dysfunction at spinal segmental levels T11-L2, possibly representing the presence of diabetic nephropathy and a corresponding VSR. Finally, the factorial design created difficulties in disentangling the independent treatment effects of OMT and UST. Although the hypothesis of statistical interaction between OMT and UST was rejected when the change in LBP severity was analyzed, additive or synergistic treatment effects relating to a change in TNF-α serum concentration potentially may have occurred, as is suggested in Figure 3.

Conclusion
Severe somatic dysfunction (as manifested by a key osteopathic lesion) was prevalent in the subgroup of patients with diabetes mellitus and comorbid chronic LBP in the OSTEOPATHIC Trial. Patients with diabetes mellitus experienced significant reductions in LBP severity over 12 weeks with OMT. A possible mechanism for OMT effects in patients with diabetes mellitus is decreased circulating levels of TNF-α. A larger clinical trial of patients with diabetes mellitus and comorbid LBP is warranted to more definitively assess the efficacy and mechanisms of action of OMT in this population.

Acknowledgments
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References


29. Hennekens CH, Demets D. The need for large-scale randomized evidence without undue emphasis on small trials, meta-analyses, or subgroup analyses. JAMA. 2009;302(21):2361-2362.

Editor’s Note: In this article, the authors use the term osteopathic manual treatment to describe the techniques used to treat patients with somatic dysfunction. The style guidelines of The Journal of the American Osteopathic Association and AOA policy prefer the term osteopathic manipulative treatment. Given the context of this article, the authors believe that the term osteopathic manual treatment is more appropriate because it is more encompassing than osteopathic manipulative treatment.