

## Osteopathic philosophy and emergent treatment in acute respiratory failure

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**Osteopathic manipulation has been used to treat a wide range of diseases in largely outpatient settings. The authors describe the emergent use of osteopathic manipulative treatment to improve respiratory mechanics in a critically ill patient with acute respiratory failure. High-velocity mobilization of cervical and thoracic dysfunctions resulted in a decreased work of breathing, improved arterial oxygenation, resolution of tachycardia, and an overall improvement in the patient's clinical condition.**

**(Key words: respiratory failure, osteopathic manipulative treatment, somatic dysfunction, pneumonia, tracheostomy, chronic obstructive pulmonary disease)**

A 69-year-old white man was admitted to the telemetry unit of a large osteopathic hospital with chief complaints of respiratory failure and oral thrush. Monitoring consisted of continuous electrocardiographic (ECG) rhythm and bedside peripheral transcutaneous oxygen saturation (SaO<sub>2</sub>). The patient had recently completed chemotherapy and undergone neurosurgical resection of a high-grade intracranial malignant astrocytoma. His social history was significant in terms of his tobacco habit (100 packs/year). The patient's previous clinical course had included pneumonia, herpetic tracheobronchitis, and prolonged ventilator-dependent respiratory failure—for which

elective tracheostomy was performed. He was dysphagic and aphasic, yet able to follow simple commands.

The house officer was called on 2 consecutive nights to respond to the patient's clinical shortness of breath, which was characterized by SaO<sub>2</sub> desaturation into the middle 80th percentile. On both occasions, copious white-yellow secretions were suctioned from the tracheostomy tube, and tracheostomy tube changes were necessitated. Problems included anterior migration and a ruptured distal cuff of the tracheostomy tube.

An emergent chest x-ray film, which had been obtained on the initial call, was interpreted as showing hyperaerated lung fields, clear costophrenic and cardiophrenic angles, and no pneumothorax. Also, right upper and left middle lobe infiltrates were identified on x-ray film. Examination of the patient's lungs revealed coarse bilateral rhonchi, panlobar expiratory wheezing, and a prolonged expiratory phase.

On the second night, the house officer was paged again 2 hours after the initial call because of the patient's sustained desaturation. When the house officer arrived at the patient's room, the SaO<sub>2</sub> monitor read 82% with a heart rate of

160 beats/min, which correlated roughly with the heart rate on the ECG. Positive-pressure ventilation via bag valve mask (fraction of inspired oxygen = 100%) was already under way. In addition, serial nebulized  $\beta_2$ -agonists had recently been administered.

The tracheostomy tube was immediately examined as the probable cause of the patient's distress; however, the tube was patent and was in the correct anatomic position. A suction catheter was then passed into the trachea. The catheter met minimal resistance and scant secretions were obtained. The results of another lung examination were essentially unchanged from the previous examination, and the trachea was midline. The patient's facial expression indicated distress.

Anterior gross chest palpation revealed hypokinetic right-sided motion, and the left upper palpable ribs were caught in inhalation. The abdomen was slightly distended, yet did not appear to be compromising what was largely diaphragmatic breathing. Lower cervical and cervicothoracic passive spinal motion was decreased in all planes. A diagnosis of C4 through C7 side bent right, rotated left was made. A generalized nonspecific bilateral paraspinal and trapezius muscle spasm was also palpable throughout the two regions.

Cervical high-velocity low-amplitude mobilization of C4 through T1 was accomplished from the patient's right side using a single motion comprising components from both short- and long-lever techniques.<sup>1</sup> The patient's arms were then crossed over his anterior chest wall (left arm cephalad to right arm), leaving the elbows in a coronal plane. The physician's right thenar and hypothenar eminences were used to cradle the spinous processes of T6 through T8. The physician's left hand cradled the posterior aspect of the lower cervical segments. A low-velocity moderate-amplitude thrust was directed superior to the physician's right hand, toward the upper thoracic segments. The physician's left hand provided a simultaneous force of cephalad traction and slight flexion from the lower neck. A series of articulatory sounds were

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audible and concomitantly palpable with a global softening of the chest. The entire treatment sequence lasted approximately 30 seconds. On posttreatment examination, bilateral symmetric rib amplitude was increased and the left upper ribs were mobile.

During the following minute, the patient's SaO<sub>2</sub> steadily climbed to between 95% and 97% and remained in that range. Approximately 1 hour after treatment, the patient's SaO<sub>2</sub> was 97% on room air, his heart and respiratory rates had fallen to near baseline, and he appeared to be sleeping.

## Discussion

The interdependence of structure and function is well accepted in osteopathic medicine.<sup>1,2</sup> Pathologic alterations in structure result in inefficient or decreased ability to function. Restoration of normal structural relationships—with corresponding resumption of function—is the philosophic basis for osteopathic manipulative treatment (OMT). Our patient provided a classic example of how alteration in structural relationship can impede function. The osteopathic implication in this case is that maximizing the efficiency of thoracic wall motion will improve respiratory status. The treatment delivered to our patient was not aimed solely at clearing a mucous plug. There was no therapeutic anteroposterior chest compression, which would have increased intrathoracic pressures and directly forced air in the distal lung segments to dislodge a bronchial occlusion. Restoring functional cervical, vertebrocostal, and costal motion allowed this patient to generate the normal cyclic pressure gradients that restored and maintained airway patency and provided for adequate tidal volume.

The cervical region was treated first because upper thoracic dysfunction is often secondary to cervical dysfunction. Restricted rib motion, whether a primary somatic dysfunction or the result of viscerosomatic reflexes, presents a challenge for which the body must compensate. However, many critically ill patients may already be at the end of their ability to compensate structurally, metabolically, and physiologically. Examples of

such compensations in patients with chronic obstructive pulmonary disease include a barrel-shaped chest and flattened diaphragm, decreased HCO<sub>3</sub> (alkalosis), and pursed-lip breathing.

One may wonder why treatment was approached in what appears to have been a random manner. An SaO<sub>2</sub> of 82% corresponds with a PaO<sub>2</sub> less than 50 mm Hg.<sup>3</sup> This value lies on the steep part of the oxyhemoglobin dissociation curve and is an objective measure of a true emergency. Also, a heart rate of 160 beats/min is above the accepted maximum heart rate for a 69-year-old man. Tachycardia can lead to lethal rhythms or precipitate rate-dependent myocardial ischemia, which is independently proarrhythmic.<sup>4</sup> Considering the severity of the patient's condition, individual rib diagnosis was not deemed appropriate. Anecdotally, most perturbations of rib mechanics are secondary to corresponding thoracic dysfunction. Also, normalization of pathologically restrictive fascial planes throughout the shoulder and upper thoracic and lower cervical regions often results in mobilization of upper thoracic vertebral segments.

Given the emergent clinical picture, it was deemed appropriate to use semispecific mobilization of the thoracic cage and related cervical articulations to improve the respiratory mechanics of the body as a whole. It is acknowledged that the structural examination after treatment still revealed dysfunction. However, the improvement evident on palpatory examination was adequate to allow for an increase in respiratory efficiency. This improvement was evidenced by a decreased level of effort required for the patient to breathe, improved arterial oxygen saturation, resolution of tachycardia, and clinical stabilization.

Inpatient respiratory failure is usually a complicated pathophysiologic scenario and is often the end result of a series of clinical processes. In the case of our patient, the etiologic factors of neurologic dysphagia, tracheostomy, chronic obstructive pulmonary disease, pneumonia, deconditioning, and copious secretions in conjunction with each other challenged his ability to maintain adequate

pulmonary status. Tracheostomy carries particular risk factors relevant to this case. Luminal obstruction has been reported as an acute life-threatening complication,<sup>5</sup> and bronchial mucous plugging has been reported as the major precipitating factor of acute respiratory failure in patients with neuromuscular disease.<sup>6</sup> Because tracheostomy bypasses the glottis, it prevents the positive-pressure buildup necessary for initiation of a forceful cough. Pressure and flow generation studies in intubated and nonintubated patients have shown comparable intraluminal pressures, but poor flow generation initiation before onset of cough.<sup>7</sup> Additionally, tracheostomy (or intubation) deprives the patient of the compensatory mechanism of self-positive end-expiratory pressure.

Respiration has been described as a dynamic orchestration involving coordinated reflex neural activity; abdominal, diaphragmatic, and other muscular contractions; motion of fascial planes; and the movement of more than 146 joints.<sup>8</sup> Within this complex orchestration exist more than 100 anatomic locations where restriction of motion can compromise function. Every anatomic structure in the chest, and many in the neck and abdomen, undergo motion on some level as the cycle of breathing occurs. Cervical motion is directly driven by cervical strap, scalene, and sternocleidomastoid musculature in addition to spinal articulations and the fascial connections throughout the pharynx, neck, and thorax. One study<sup>9</sup> in an intubated animal concluded that severing the ventrolateral cervical structures that mechanically link the thorax to the upper airway stops phasic fluctuations as well as results in an overall increase in upper airway resistance. Additionally, that study revealed that with denervation of upper airway dilatory muscles, paced diaphragmatic breathing still reduced upper airway resistance by 51% ± 11% (SEM; n = 7). A separate study proved that the changes in longitudinal tracheal tension occurring with respiration were the result not only of changes in intrathoracic pressure, but also from the pull of the diaphragm through mediastinal structures.<sup>10</sup>

In 1899, Andrew Taylor Still, MD,

DO, regarded lungs as "...surely the half-way place between life and death."<sup>11</sup> The cardinal focus here is the fundamental need to move air, which provides for O<sub>2</sub>/CO<sub>2</sub> exchange. Multiple osteopathic dysfunctions involving the thorax may place patients at a significant mechanical disadvantage from which they are unable to maintain adequate minute ventilation. In this case, the compensatory mechanisms of glottic closure and pursed-lip breathing were also impossible for the patient to achieve. As a result, our patient began to use accessory respiratory muscles and increase his respiratory rate. Diaphragmatic breathing has been shown to improve arterial blood gases, but such breathing requires greater inspiratory muscle loading.<sup>12</sup> Diaphragmatic breathing has also been shown to be mechanically inefficient and is associated with an increased sensation of dyspnea.<sup>13</sup>

A typical pattern might occur as follows: Patients are able to compensate for a period of time, after which they are unable to keep up with the increased metabolic demand. If they do not receive intervention, they fatigue, fail, become anoxic, and have cardiopulmonary arrest. The manipulative treatment given to this patient apparently relieved the accumulated somatic dysfunctions restricting his thorax, thereby resulting in clinical stabilization.

## Comments

Osteopathic manipulative treatment has been used to treat a wide variety of diseases in predominantly non-life-threatening settings. Yet, the potential also exists for OMT to be used for maintaining and protecting critical anatomic structures such as the airway. Nearly a century of medical research has taken place since A. T. Still described medical authors as "...unable to give intelligent explanations and having little to offer us."<sup>14</sup> That may have been true at the time, but real-time imaging, invasive critical-care monitoring technology, and electrodiagnostic tests have the potential to shed an objective and quantitative light on manual treatments, including OMT.

An example in our case is what a pulmonary artery pressure transducer would

have revealed pretreatment and post-treatment. It is reasonable to conclude that otherwise asymptomatic cervical and thoracic somatic dysfunctions can become critical factors in patients who have minimal remaining functional reserve. Emergent treatment of such dysfunctions may have a role, and should be considered, in stabilization of clinically deteriorating patients in whom there is palpable gross motion.

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