Chest pain and the role of somatic dysfunction

CRAIG M. WAX, DO
DAVID S. ABEND, DO
ROBERT H. PEARSON, DO

The symptom of chest pain may be a result or manifestation of somatic dysfunction of the musculoskeletal system. Chest pain is a common chief complaint among patients in a family practice office or in the emergency room. Various intrathoracic and extrathoracic factors may be responsible. The authors describe their experience with one patient in whom osteopathic manipulative treatment was used along with accepted medical tests and therapy to diagnose and treat this patient. If chest pain is effectively diagnosed and promptly treated, OMT can provide the most thorough, cost-effective, and satisfying care available.

(Key words: Somatic dysfunction, chest pain, osteopathic manipulative treatment, osteopathic manipulative medicine, manipulation, thoracic cage, nerve facilitation)

In any emergency room (ER), a common inciting complaint for admission is chest pain. Many diverse causes exist that may fit into cardiac, vascular, pulmonary, gastrointestinal, musculoskeletal, and other categories (*Figure 1*). A commonly overlooked cause of chest pain is somatic dysfunction of the musculoskeletal system and soft tissues of the thoracic cage.¹ It may be the lone cause of the pain, in synergistic combination with other factors, or it may be a manifestation of autonomic disturbance.² Effectively diagnosed and treated, the patient will be restored to normal function with the pain resolved. The following case describes a relatively healthy-appearing patient with cardiac risk factors who presented with chest, back, and shoulder pain.

Report of case History

A 52-year-old white man presented to the ER with a chief complaint of epigastric and lower chest pain that persisted for two episodes, each lasting 15 to 20 minutes, during a 2-day period. The pain was characterized as sharp over the lower sternum and epigastrium. The patient rated the severity of the pain at 6 to 7 on a subjective pain scale of 10.

He also had right shoulder pain for the past year that was exacerbated with movement. He was unsure if the shoulder and epigastric pain were radiating from the chest pain or independent phenomena. At home, he assumed the pain might be heartburn and had tried antacids without results. Although resting seemed to make the pain better, nothing seemed to provoke the pain.

Nitroglycerin was administered sublingually and a 1-inch strip of nitroglycerin paste was applied to his anterior chest wall. Chest pain was minimal after treatment. He was also given 1 aspirin (325 mg) at that time. He denied recent

episodes of shortness of breath, headache, syncope, nausea, and vomiting.

Medical, surgical, social background

His medical history was significant for insulin-dependent diabetes mellitus. He had undergone a laparoscopic cholecystectomy 5 years earlier, and appendectomy and tonsillectomy and adenoidectomy many years before that. His social history was of interest as he was a former cigarette smoker (one pack daily for 20 years) who had quit 4 years previously. He used to drink 3 to 10 beers per day, but had quit 3 years ago. He denied use of any drugs except his insulin (10 μ U/mL regular and 34 μ U/mL NPH in the morning; 10 μ U/mL regular before dinner, and 28 μ U/mL NPH before bed). He reported drinking 15 cups of caffeinated beverages (coffee, tea, and cola) per day. For the past few years, he has been exercising by walking briskly for 1 hour a day. He denied any significant family medical history or predisposition to any illnesses. A review of his systems revealed only lower sternal chest pain—sharp in quality with questionable radiation to the epigastrium and right shoulder. His cardiac risk factors included formerly smoking cigarettes and drinking alcohol; being an insulin-dependent, middle-aged diabetic male; and imbibing large amounts of caffeine.

Physical examination

His vital signs were as follows: temperature, 97.4°F; pulse rate, 80 beats per minute; respirations, 18/min; and blood pressure, 147/87 mm Hg. The patient was well nourished, well developed, well hydrated, alert, and cooperative. He had an athletic build and seemed to be in no acute distress. His skin was warm and moist but not diaphoretic.

Head examination was atraumatic/normocephalic; pupils were equal and reactive to light and accommodation and extraocular muscles were intact bilaterally. No carotid bruits or thyroid abnormalities were present. His inner ears, nasal mucosa, and posterior oropharynx were without erythema and injection. His neck was supple. His heart

At the time this paper was written, Dr Wax was a senior resident in family medicine at Allegheny University Hospital—City Avenue Hospital (Philadelphia, Pa). Drs Pearson and Abend are in family practice together in Emerson, NJ.

Correspondence to Craig Wax, DO, 18 Leonardville Rd, Middletown, NJ 07748.

E-mail address: craigwax@mem.po.com.

rate and rhythm were regular without any murmurs, pericardial rubs, or gallops. His lung fields were clear bilaterally without wheezes, rales, or rhonchi. His abdomen was soft, nontender to palpation, and without organomegaly.

Bowel sounds were present in all four abdominal quadrants. The rectal examination was contraindicated to prevent vagal overstimulation, as myocardial infarction (MI) and angina pectoris were to be ruled out. Neurologically, his examination was nonfocal. His deep tendon reflexes and pulses were strong and equal bilaterally; there were no acute deficits.

The musculoskeletal examination revealed right shoulder girdle and thoracic paravertebral spasm and pain with palpation of deep soft tissues. No noticeable scoliosis was present nor were Freyette's type I (multiple segment) curves noted on the standing examination; however, multiple midthoracic type II (single segment) dysfunctions were noted, predominantly rotated left and side bent left. His right first rib was slightly elevated and restricted in its descent on expiration by spasmodic scalene muscles. The C2 vertebra (occipito-atlantal joint) was rotated right and the C7 vertebra was rotated right and side bent right. His sharp chest pain was reproducible upon firm palpation to the anterior aspect of the right 10th rib at the sternocostal junction. This rib was prominent anteriorly and tender to palpation. His right shoulder was limited in abduction to 100 degrees by discomfort.³ Laboratory data for this patient are shown in *Table 1*.

Treatment and hospital course

During the ER postphysical examination, the patient agreed to undergo OMT to treat a likely source of his pain. Because the pain was reproducible with firm palpation of the right anterior 10th rib, it was of high probability that his symptoms were amenable to two approaches: OMT alone or in combination with nonsteroidal anti-inflammatory drugs (NSAIDs) and muscle relaxants; and rest.

The treatment plan was to use OMT

Checklist

■ Cardiac causes

- ☐ Myocardial infarction
- ☐ Coronary artery disease
- ☐ Aortic stenosis
- ☐ Hypertrophic cardiomyopathy
- ☐ Pancarditis

■ Vascular causes

- ☐ Aortic dissection
- ☐ Pulmonary embolism
- ☐ Pulmonary hypertension
- ☐ Right ventricular strain

■ Pulmonary causes

- ☐ Pleuritis or pneumonia
- ☐ Tracheobronchitis
- ☐ Pneumothorax
- ☐ Tumor
- ☐ Mediastinitis or mediastinal emphysema

■ Gastrointestinal causes

- ☐ Esophageal reflux
- ☐ Esophageal spasm
- ☐ Mallory-Weiss tear
- ☐ Peptic ulcer disease
- ☐ Biliary disease
- ☐ Pancreatitis

■ Musculoskeletal causes

- ☐ Somatic dysfunction of thoracic cage
- ☐ Cervical disk disease
- ☐ Arthritis of shoulder or spine
- ☐ Costochondritis/chest wall pain (Tsetse's syndrome and Texidor's twinge)
- ☐ Intercostal muscle cramps
- ☐ Interscalene or hyperabduction syndromes
- ☐ Subacromial bursitis

■ Other causes

- ☐ Breast disorders
- ☐ Chest wall tumor
- ☐ Herpes zoster

to relieve somatic dysfunctions, consider pharmaceutical intervention if OMT incompletely relieved the symptoms, and admit the patient for observation and have the patient undergo an angina pectoris workup to rule out an MI. Although his primary complaint of chest pain was reproducible with digital pressure, the index of suspicion of angina remained high, despite his overall healthy appearance. Unstable angina pain and MI had to be ruled out in light of mildly elevated creatine kinase (CK) levels, although his CK-MB isoenzyme fraction and index were low for possible ischemia or MI (*Table 2*). His history and cardiac risk factors, previously mentioned, were also taken into consideration.

Muscle energy techniques (isometric resistance and reengagement of functional barrier) were used to treat the thoracic spine dysfunctions and followed up with high velocity/low amplitude (HVLA) thrusting technique. The occipito-atlantal joint and the C7 vertebrae were engaged and corrected using these same techniques. The 1st and 10th ribs on the right were no longer elevated and resumed normal functional mechanics after treatment. The patient reported that the sharp chest pain and shoulder and thoracic pain resolved after OMT.

He was admitted to the telemetry unit to the service of a cardiologist to rule out new onset of angina and for observation. Serial electrocardiograms (EKGs) and cardiac enzyme tests (*Table 2*) were ordered over an 18-hour period. His EKGs continued to demonstrate normal sinus rhythm. His chest, shoulder, and back pain did *not* return at any time during his 24-hour hospital stay. Having been diagnosed with "atypical chest pain," the patient was discharged in stable condition.

Discussion

Musculoskeletal disorders are responsible for more cases of chest pain than any other disease entities.⁴ Many times physicians are content to concentrate on the heart and lungs for the cause of chest pain. Certainly, the cardiac and pulmonary organs are among the most seri-

Figure 1. Some causes of chest discomfort. (Adapted from Wilson, et al: Harrison's Principles of Internal Medicine, ed 12. New York, McGraw-Hill, Inc, 1991.⁶)

ous and life-threatening causes of pain. Logically, then, cardiac and pulmonary causes must be ruled out first. When dealing with chest pain, physicians should always assume the worst; use the patient's history, physical examination, and diagnostic tests to rule out threats to life.⁵ However, just as "all that wheezes is not asthma," so it follows that "all chest pain is not angina." The chest is innervated by thoracic spinal segments T1-T6. Structures that receive sensory fibers from this region include bones, muscles, skin, lungs, heart, mediastinum, gallbladder, stomach, and pancreas.⁵ Both somatic and visceral means may be responsible for chest pain. In otherwise healthy and normal patients, somatic dysfunction of the musculoskeletal system is responsible for pain that may mimic angina pectoris but does not respond to other medical and surgical modalities.

Somatic factors discussed in this article can play an integral role in the differential diagnosis of perceived cardiac complaints. Anterior chest wall syndrome of noncardiac origin may present with somatic factors that can either coexist with cardiac disease or be construed as purely musculoskeletal once cardiac etiology is ruled out.

We have discussed some common noncardiac etiologies, such as cervical, thoracic, rib, and sternal somatic dysfunctions; costochondritis; intercostal myositis and neuritis; and myofascial trigger points related to greater pectoral and smaller sternal muscles or a rib fracture (obvious or stress). When cardiovascular diagnostic and therapeutic measures prove to be negative, osteopathic manipulative correction of these aforementioned somatic problems can relieve the patient's symptoms. Successful treatment of the specific palpatory spinal segmental facilitation reflex can be augmented with a combination of OMT, physical therapy, NSAIDs, local application of heat or cold, and other modalities, such as electric muscle stimulation and ultrasound.

The autonomic nervous system (ANS) is vital to the structure and function of the body in health and disease states.

Table 1
Case Report: Laboratory Data

Test	Values
White blood cell count	4.4×10 ⁹ /L
Hemoglobin	13.9 g/dL
Hematocrit	39.9 %
Platelets	199×10 ⁹ /L
Sodium	140 mEq/L
Potassium	3.5 mEq/L
Chloride	104 mEq/L
Carbon dioxide	24 mEq/L
Blood urea nitrogen	14 mg/dL
Chromium	0.6 mg/dL
Glucose	159 mg/dL
Total protein	6.1 g/dL
Albumin	3.9 g/dL
Globulin	2.2 g/dL
Albumin-to-globulin ratio	1.8
Calcium	8.8 mg/dL
Total bilirubin	1.4 mg/dL
Phosphate	3.2 mg/dL
Uric acid	4.4 mg/dL
Cholesterol	134 mg/dL
Triglycerides	140 mg/dL
Alkaline phosphate	86 U/L
Aspartate transaminase	22 U/L
Alanine transaminase	19 U/L
Lactic dehydrogenase	142 U/L
Creatine kinase	287 U/L
Creatine kinase isoenzymes, MB fraction	2.7 %
Creatinine kinase isoenzyme index	0.9
Prothrombin time, in seconds	12.2
International normalized ratio	1.0
Partial thromboplastin time, in seconds	24.4
Electrocardiogram, beats per minute (no acute ischemic changes)	77
Chest x-ray film	No acute pulmonary process or infiltrates

Clinical application of ANS concepts can help one to understand the pathophysiology and, hence, rationale for OMT of noncardiac and cardiac chest pain. The ANS is further subdivided into the sympathetic (SNS) and parasympathetic (PSNS) nervous systems. The SNS plays a role in modulating somatic tissues by providing outflow tracts through visceral afferent nerves. These impulses provide information to the central nervous system (CNS). The patient interprets this information as pain in the somatic tissues, such as muscles, ligaments, tendons, fascia, joints, and other soft tissues surrounding the chest wall and related to the cervicothoracic and rib region, known collectively as the thoracic cage. Palpation of these tissues can provide the physician with important information regarding the state of affairs in the patient's body. Diagnostically, the physician can use this information to understand how the SNS innervations and the SNS collateral ganglia can be affected adversely during noncardiac reflex pathways that transmit pain. Using certain manipulative skills, the osteopathic physician can help to reduce the increased sympathetic tone, which at first provides a negative painful "facilitated" response or reflex. In this manner, DOs have been able to improve the patient's body function, thus restoring homeostasis under conditions of somatic dysfunction, disease, or poor health. Although perceived by some as "alternative" or "complimentary," specifically applied OMT by a DO (or an MD) can augment traditional medical therapies, such as pharmacologic or surgical methods in a nonsurgical, conservative manner.

The SNS innervates all visceral organs including the blood vessels (*Figure 2*). Therefore, it affects the soma (bone, muscles, ligaments, fascia, tendons, and other connective tissue) and the CNS, peripheral nerves, and lymphatic vessels. Sympathetic ganglia are specific tissues in which a synapse occurs. These synapses can be located paraspinally or in collateral ganglia (celiac, superior, and inferior mesenteric ganglia), the latter of which are not related to the heart

Table 2
Serial Cardiac Enzymes Measured During 18-hour Period

Lactic dehydrogenase (LDH) (118-273 U/L)*	Creatine kinase (CK) (24-195 U/L)*	Creatine kinase isoenzymes MB fraction (CK-MB)	CK-MB index
142	287	2.7	0.9
146	224	3.3	1.5
136	217	2.4	1.1

*Reference laboratory normal range.

and lungs. Thoracolumbar sympathetic outflow to all parts of the body originates in the intermediolateral columns of the spinal cord at the T1-L2 levels. Impulses travel out from the spinal cord (an afferent system) through paraspinal chain ganglia deep to each vertebra. The vertebrae can be palpated effectively after undergoing skillful manipulative training such as that taught in the colleges of osteopathic medicine.

The main target of OMT in noncardiac and cardiac chest pain is to alleviate pain caused by sympathetic outflow from facilitated segments. The SNS nerves travel in mesh-like strands and follow the course of the arterial blood supply to the organs and tissues (viscera). The location where mesh-like sympathetic webs accumulate is called a plexus. In the thoracic region exists a cardiac plexus. This region receives postganglionic sympathetic nerve fibers from the chain of ganglia in and around the spinal regions T1-T6 and postganglionic cardiac nerves from each of the cervical sympathetic paraspinal chain ganglia. It also contains parasympathetic cardiac nerves from the right and left vagus nerve.

The visceral afferent system comprises nerves that carry afferent nerve impulses from the viscera to the spinal cord. This system uses the same pathway as that particular organ's sympathetic innervation. Less frequently, the parasympathetic system (such as lung alveoli) is used. In this manner, nervous impulses "reporting" an organ's dysfunction will

usually synapse in the spinal cord at the level of the particular sympathetic innervation between T1 and L2.

When sympathetic hyperactivity occurs, a disease-related nervous hyperactivity called "facilitation" takes place. This facilitation is produced by visceral afferent nervous bombardment of that cord level or by proprioceptor bombardment from a somatic dysfunction. The facilitated segment "fires" sympathetic nerve outbursts to the related organ and somatic structures when somatic or visceral impulses (viscerosomatic, somatovisceral, somatosomatic, viscerovisceral) just pass.

"Facilitated segments are chronically hyperirritable and hyperresponsive. Muscles in the region are maintained in a hypertonic state, which constricts the available spinal motion. All of the supportive structures and their innervations may become afferent sources of facilitation."⁶ There are pools of central and peripheral neurons that become facilitated and, thus, trophic and vasomotor influences produce observable and palpable changes in the tissue.⁴ This inappropriate sympathetic bombardment of visceral or somatic tissue has a detrimental effect on that tissue and, consequently, the body. This "cross-talk" between the somatic and visceral system at the spinal cord level gives the physician diagnostic musculoskeletal clues to the particular visceral dysfunction. The physician can then treat the musculoskeletal system at that particular spinal level with OMT and influence

the organ's function via its effect on the facilitated cord segment.

In this instance, especially with MI, this SNS facilitation increases stress on the myocardium. Minimizing this response with OMT decreases myocardial demand. A decrease in myocardial demand minimizes the extent of the MI and maximizes the cardioprotective effects of nitroglycerin, β -blockers, and other agents.

We can see sympathetic hyperactivity and its facilitation in the musculoskeletal system, especially with regard to noncardiac chest pain in the form of several common syndromes. Job-related stress and its resultant anger, frustration, occupational deadlines, phobias, anxiety, panic, and pain (acute and chronic) can exacerbate somatic dysfunction. Other lifestyle factors—nutrition, sleeping habits, exercise, body mechanics, and posture—all play a crucial role.

Digital palpation of the soft tissues, joints, and spinal segments gives practitioners important information. Increased or decreased neural activity and viability of these areas can be felt in the form of tissue texture, tone, temperature, moisture, and resiliency. Further palpable are motion characteristics of joints, soft tissues, and fascial planes, all of which are diagnosed as segmental somatic (musculoskeletal) dysfunction.

In many instances, it is unclear if the somatic dysfunction is "the chicken or the egg." In other words, it may be a manifestation of underlying visceral disease (viscerosomatic) or the cause of the visceral dysfunction (somatovisceral). It is also possible that it is an unrelated and independent phenomenon. In any scenario, it is most strongly suggested that palpatory diagnosis and treatment be applied for the benefit of the patient.

Thoracic cage somatic dysfunction may manifest itself in many ways, including restriction of motion and palpatory tissue texture changes. It may then be treated with good results using any of the osteopathic manipulative medicine (OMM) techniques. They include indirect techniques, soft tissue stretching, myofascial techniques, counterstrain,

facilitated positional release, muscle energy, and even HVLA. The goal of OMM is to restore homeostasis by normalizing joint and tissue motion, fluid flow, and neurologic sensitivity. The combinations of OMM techniques are endless and, therefore, hard to quantify and qualify, but all yield encouraging results when conscientiously applied with sound medical history and physical and diagnostic determinations.

One of the purposes of this article is to introduce criteria to standardize the diagnosis of musculoskeletal chest pain. It is also possible to use the following criteria to safely and accurately rule out cardiac abnormalities as part of the patient's workup:

- Pain is reproducible by palpation of the thoracic cage. Palpation is defined by asymmetry, restriction of motion, tissue texture changes, and tenderness (the ARTT of diagnosis).⁷
- Palpatory findings of somatic dysfunction may be ameliorated by OMT.
- No acute ischemic EKG changes exist.
- Laboratory results for three sets of cardiac enzymes are normal.

In the case reported here, OMT was a cost-effective and time-effective diagnostic and treatment modality. Once the cardiac tests determined our patient was stable, a trial of manipulation was indicated (Figure 3). When this standard becomes commonplace, patients will be treated more appropriately and not be likely to "bounce" back and forth from home to emergency room and back again.

In the era of managed care and abbreviated hospital stays, it is urgent that physicians consider the palpatory diagnosis and treatment of the musculoskeletal system coupled with a solid patient history. It is especially important when evaluating chest pain. It will save the patient countless repetitive and costly workups during the course of his or her life.

By taking time and following this approach, the physician will be rewarded with better patient and monetary outcomes. Recently, OMM billing codes were added to the Standard CPT codes to ensure reimbursement. Therefore,

Figure 2. Chest pain management algorithm.

Figure 3. Common sympathetic innervations. (Adapted from KM: Osteopathic Considerations in Systemic Dysfunction. Kirksville, Mo, Kirksville College of Osteopathic Medicine Press, 1991.²)

OMM diagnosis and treatment becomes a time- and cost-effective means of delivering care. It will further assure patients of receiving a higher quality of health-care.

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Case reports

Neuroleptic malignant syndrome: Case report and review

TIMOTHY J. DYKSTRA, DO

MICHAEL J. MENOLASINO III, DO

Neuroleptic malignant syndrome is a rare but potentially fatal disorder that has been associated with the use of antipsychotic medications. Because neuroleptic malignant syndrome is rare, clinicians often have a low index of suspicion for the disorder which may lead to delayed treatment and increased mortality. This article describes a case of neuroleptic malignant syndrome and briefly reviews current diagnostic criteria and treatment options.

(Keywords: Neuroleptic malignant syndrome, hyperthermia, antipsychotics)

Neuroleptic malignant syndrome (NMS) is a drug-induced hyperthermic disorder first described by Delay and associates¹ in 1960. Although discrepancy exists in the reported incidence of the disorder, most of the literature suggest that the disorder occurs in 0.07% to 2.2% of those patients treated with antipsychotic medications.² Recently, particular attention has been drawn to a possibly higher incidence of NMS in those patients treated with a combination of haloperidol and lithium carbonate.^{3,4}

When NMS occurs, it carries a mortality rate of 10% to 20%.^{2,5} Reduction in mortality appears to be directly related to the early recognition of symptoms and risk factors by healthcare workers.⁶ This article therefore presents a case of NMS, with a brief review of current diagnostic criteria and treatment options to familiarize clinicians with the disorder.

Report of case

A 49-year-old man was admitted to a community hospital from a local extended care veterans facility in August 1996

with periumbilical pain as his chief complaint. The patient's medical history was remarkable for schizophrenia, bipolar disorder, and Barrett's esophagus with gastroesophageal reflux disease. A known manifestation of his schizophrenia was psychogenic polydipsia, with resulting past bouts of abdominal discomfort and hyponatremia. His medications included the following: Omeprazole (Prilosec), 20 mg/d; lithium carbonate, 600 mg twice a day; clomipramine hydrochloride (Anafranil), 50 mg at bedtime; trifluoperazine hydrochloride (Stelazine), 20 mg at bedtime; and benztropine mesylate (Cogentin), 2 mg at bedtime. He was admitted to the service of an attending general surgeon.

Workup of the patient's abdominal pain revealed evidence of right colonic dilation of 18 cm in the greatest dimension. After a failed attempt at decompressive colonoscopy, the patient was taken to surgery and a successful right hemicolectomy was performed. The patient was allowed to have his medications at the outpatient dosages, with sips of water on the second postoperative day.

On the third postoperative day, the nurses reported that the patient was drinking large amounts of water despite their surveillance. A patient sitter was ordered,

From the Department of Internal Medicine, Metropolitan Hospital, Grand Rapids, Mich.

Correspondence to Timothy J. Dykstra, DO, Department of Internal Medicine, Metropolitan Hospital, 1919 Boston, SE, Grand Rapids, MI 49506.
E-mail address:timdykstra@aol.com.