COILS

Patient with a
Myocardial Infarction

Prepared by: AACOM’s Educational Council on Osteopathic Principles
Description

This Clinical Osteopathically Integrated Learning Scenario (COILS) focuses primarily on the palpatory evaluation and supportive osteopathic manipulative treatment for a patient with myocardial infarction.

The COILS is divided into two sections:

Section One
The Roundtable Discussion Workshop includes a discussion and evaluation of the patient’s case history, diagnosis, pathophysiology, osteopathic principles involved, functional anatomy, treatment options, contraindications, and (if time permits) a demonstration of manipulative treatment techniques applicable to the patient’s homeostatic needs.

Section Two
The Patient-Based Application Workshop is the supervised application of manipulative treatment techniques for a patient with this diagnosis. The workshop is designed to evaluate the student’s or physician’s diagnostic and psychomotor skills when providing an osteopathic manipulative treatment for an actual (or simulated) patient.

If time permits, the instructor may deliver this entire two-section program at one time. Ideally, however, Section One should be taught several days before Section Two to allow time for the student or physician to review and practice appropriate techniques. If an actual patient is not available for Section Two, a simulated patient may be used.
Section One: Roundtable Discussion

I. Description

This section is a roundtable-type presentation and discussion on the osteopathic approach to the treatment of a patient with a myocardial infarction (MI).

II. Cognitive Components

A. Case Presentation

A 48-year-old Native American male with a history of MI and congestive heart failure (CHF) presents with complaints of slight chest pressure and slight nausea. He first noticed the pressure in his chest while shoveling snow a week ago. The pain then spread to his left arm and was accompanied by nausea. Initially, the symptoms responded to sublingual nitroglycerin, but the pain has become more frequent and has not subsided with nitroglycerine. The symptoms lessen with rest. The patient waited 16 hours before presenting to the emergency room. He was initially treated with aspirin, a beta blocker, and IV nitroglycerin and then was admitted to the hospital.

The patient has a history of hypertension, and hypercholesterolemia. He had a MI 3 years ago. He has known CHF, coronary artery disease, as well as angina upon exertion. He refuses invasive studies or treatments but has agreed to take medications for chest pain, hypertension, and hypercholesterolemia. Present medications include Losartan 100 mg PO daily and Simvastatin 20mg PO daily. The patient has a cholecystectomy about 5 years ago.

Physical Examination

Vital signs: Temperature, 99° F; Blood Pressure, 154/92; Respiratory Rate, 22; Heart Rate, 108; Height: 6’4”; Weight, 285 lbs

General: Anxious; pale; slightly diaphoretic

Skin: Warm, dry, without lesions or rashes; skin testing on the hand, forearm, and abdomen remains tented at 10 seconds

Head: Normocephalic; atraumatic

Eyes: Pupils equal, round, reactive to light, and accommodation; external ocular muscles intact.

Nose: Nares without discharge; turbinates engorged; no sinus pressure tenderness; no epistaxis

Throat: No adenopathy; thyroid not enlarged; trachea midline and moveable; no masses

Cardiac: Rate and rhythm rapid and regular at 108 bpm; S3 gallop noted

Lungs: Scattered rales noted
Abdomen: Well-healed scar noted in the right upper quadrant of abdomen consistent with surgical history; bowel sounds within normal limits; abdomen soft, protuberant; no masses, tenderness, or rebound

Genitourinary: No scrotal edema

Rectal exam: Sphincter tone normal; occult blood negative

Extremity: 2+ pitting edema noted

Osteopathic Structural Examination

- OA FSLRR
- AA rotated left
- Left first rib elevated
- T1 ERLLS
- T2–T4 NSRRL, with tissue texture abnormalities in paraspinal soft tissues along T2–T4 left, inclusive of rib angles
- T7–T10 NSLRR
- Diaphragm motion restriction on the left, with ribs 7–10 exhalation restriction (inhalation somatic dysfunction)

Diagnostics

- EKG shows ST segment elevation in leads 2, 3, aVF
- Troponin-1 – 5.1 (Normal Value: <0.4 ng/ml)
- Chest x-ray showed no acute pulmonary disease

Diagnosis

1. Stable angina
2. Another form of acute coronary syndrome (unstable angina or nstemi)
3. Acute pericarditis
4. Pulmonary embolism
5. Pneumonia
6. Pneumothorax
7. Esophageal spasm
8. Acute gastritis
9. Cholecystitis
10. Pancreatitis
11. Musculoskeletal chest pain
B. Pathophysiology

1. Acute MI is a clinical event associated with myocardial cell injury or death.
2. The most common mechanism for MI, coronary thrombosis, is secondary to platelet adhesion and narrowing of arterial vessels.
3. Diagnosis of MI is made based on elevation of cardiac biomarker values with associated symptoms of ischemia, EKG changes (Q wave, ST elevations, new LBBB), abnormal angiography or imaging. (Thygesen k)
4. The MI can involve systolic and diastolic failure.
5. Compensatory responses include increased sympathetic tone, blood pressure, heart rate, myocardial contractility, and myocardial workload.
6. Compensatory responses contribute to a “supply and demand” imbalance for perfusion of the myocardium.
7. Arrhythmias are commonly associated with MI and other types of heart disease. Sympathicotonia encourages tachyarrhythmias. Inappropriate increased parasympathetic tone encourages bradyarrhythmias and heart blocks.
8. Visceral facilitation of the spinal cord from the visceral afferents of the myocardium in the region of the MI facilitates its spinal cord segments and produces the deep severe pressure in the chest and arm. This situation corresponds to innervation from the somatic afferents and enhances the palpable tissue texture changes.

C. Functional Anatomy

Includes knowledge of structure and physiology necessary to properly carry out the osteopathic manipulative treatment support.

1. Chronic viscero-somatic tissue changes are palpable in the paraspinal deep soft tissues of related spinal cord segments (e.g., T1–T5). Chronic segmental somatic dysfunction acting over time can produce a hyperexcitable or “facilitated” spinal segment. This irritable segment responds abnormally, usually excessively, to minimal stimuli and an increase in the sympathetic outflow to related visceral organs (e.g., the heart, coronary arteries).
2. Research studies have documented viscero-somatic reflexes in the left upper thoracic area of patients with MI.
3. Osteopathic physicians have describe the cardiac reflex as side bent left and rotated right (group mechanics) in the upper thoracic spine. This long-term positional change is produced by chronic hypertonicity of the left upper thoracic muscles.
4. Somatic dysfunction at T1–T2 are often associated with patients who develop tachyarrhythmias.
5. A right pectoralis major trigger point has been associated with selected cardiac arrhythmias, many of which are resistant to anti-arrhythmic drugs.
6. Dr. K Sato identified dorsal root ganglion cells with a visceral projection to the heart and a somatic projection to the left arm. A single cell has two projections: one to the periphery and one to the viscera. The dorsal root ganglion cells report to the spinal cord. The central nervous system (CNS) is not accustomed to the nociceptive input from the heart, so the pain is interpreted as coming from the arm.

7. Initial pain from an MI is visceral in nature and is described as a severe, deep-pressure feeling. As the pressure dissipates, it is replaced with a severe, sharp chest, arm, and neck pain, as the viscero-somatic reflex takes over.

D. Goals for Osteopathic Manipulative Management

Includes a review of treatment pearls; a general plan for manipulative treatment of the patient; and a discussion of treatment options, contraindications, and plans for follow-up evaluation and treatment.

Initial Management

1. Address the underlying cause, MI, before initiating OMM/OMT.
2. Treat viscero-somatic reflexes with inhibitory pressure or release techniques directed toward upper thoracic and OA somatic dysfunctions to reduce viscero-somatic chest pain.
3. Improvement of chest pain relieves anxiety, which reduces CNS facilitation.
4. Treat the musculoskeletal (e.g., OA, cervical, upper thoracic, rib) components associated with arrhythmias. This treatment reduces detrimental somatic influence to the facilitated segment and the heart.
5. For tachyarrhythmias, treat the upper thoracic somatic dysfunctions that encourage inappropriate sympathetic outflow to the heart. These areas are usually located at T1–T2 and their corresponding ribs.
6. For bradyarrhythmias and heart block, normalize the vagal response. This process includes treatment of OA/AA and cervical region somatic dysfunctions. Use suboccipital inhibition (Foundations, pp.781–782); an indirect method to the cervical somatic dysfunctions (Foundations, pp. 802–803).
Long-Term Management

1. Treat chronic motion restrictions of the upper thoracic region, if present. Perform this type of treatment once the patient is ambulatory.
2. Treat as necessary to maintain proper diaphragmatic function. Technique selection may involve direct or indirect methods; myofascial release; and thoraco-abdominal diaphragm release using indirect methods.
3. Treat cervical spine suboccipital inhibition, and relieve any mid-cervical somatic dysfunction. This treatment may help with diaphragm function via the phrenic nerve.
4. Perform thoracolumbar soft tissue release, articulatory treatment, or myofascial release, all of which can improve diaphragm function.
5. Normalize fascias at the thoracic inlet, which may redome a flattened thoracoabdominal diaphragm.

E. Contraindications and Cautions Regarding Treatment

See contraindications to treatment, Foundations, pp. 1015–1024.

1. Do not treat the patient in the supine position or treatment positions that restrict respiratory efforts.
2. Do not treat with forceful direct method treatments.
3. Do not overtreat.
4. Note that liver pump, liver flip, and classic thoracic pumps are all too vigorous. The liver and spleen may be friable, so be careful to avoid undue sudden compression or decompression changes in abdomen or undue abdominal pressure.
5. Continue to treat the patient to provide optimal lymphatic flow to reduce the amount of scarring from the healing process.

F. Instructor’s Notes

Personal clinical pearls and lessons learned from previous COILS presentations.

1. Patients with significant heart problems have chronic upper left thoracic changes and group upper left thoracic curve (Type I).
2. Within 24 hours, cardiac viscero-somatic reflex develops, sharpening and tightening the chest pressure. Cardiac reflex develops (TART) at the T3–T5 region unless the infarct is posterior. In a posterior MI, the reflex is usually at T5 and spills over into the abdomen area, producing GI symptoms. This viscero-somatic visceral reflex begins to affect upper GI sympathetic innervation. Upper GI symptoms are also influenced through the C2 connection with the vagus nerve. Nausea, vomiting, or other upper GI complaints are a common. "Fatal gastritis" is often an undiagnosed posterior wall MI.
3. There are two basic rhythm problems:

- Bradyarrhythmias and heart block are likely if vagal nerve irritation dominates. Somatic dysfunction is usually found in the neck. Right-sided OA and C2 somatic dysfunctions are more likely to initiate or predate cardiac bradyarrhythmias. Left-sided OA and C2 somatic dysfunction are more likely to predate cardiac bradyarrhythmias or heart blocks.
- Tachyarrhythmias are likely if sympathicotonia dominates the clinical picture, and somatic dysfunction may be found in the T1–T2 region.

Initial OMM/OMT

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III. **Psychomotor Components**

*If time permits, this part can be carried out on a simulated patient.*

1. Practice palpatory diagnosis. (See techniques under Section D above.) Diagnoses procedures include OA, AA, and cervical spine; upper thoracics and ribs; thoracic inlet; anterior chest wall; thoraco-abdominal diaphragm; thoracic cage compliance; tissue texture palpation and evaluation; and Zink whole-body fascial pattern.

2. Demonstrate key treatment techniques in the body regions involved. These techniques include inhibitory paraspinal pressure, myofascial release for the upper thoracics and ribs, OA myofascial release/indirect, cervical, and the universal (Sutherland) rib.

3. Evaluate the plan for treating the patient in the appropriate position, localization of gentle forces, and activation.

IV. **References**


**V. Examination Questions**

*These multiple-choice questions involve the treatment of a patient with MI.*

(* denotes answer)

1. Inhibitory treatment of which area would be most likely to decrease excessive sympathetic tone to the heart?
   A. OA  
   B. C3–C5  
   C. T2–T4  
   D. T10–T12  
   E. L4–L5

VI. Treatment of which area would have the greatest effect on vagal tone?

A. OA  
B. C3–C5  
C. T2–T4  
D. T10–T12  
E. Sacroiliac

VII. Which should you do prior to beginning lymphatic treatments?

A. Treat paraspinal inhibition.  
B. Treat thoracic inlet.  
C. Treat OA somatic dysfunction.  
D. Perform the Kirksville “KRUNCH” (“supine multiplane thoracic thrust”) to the mid-thoracic spine.  
E. Rock the sacrum.

VIII. Which lymphatic technique would be the most appropriate treatment?

A. Liver pump  
B. Pectoral lift  
C. Classic thoracic pump  
D. Ischial rectal fossa technique  
E. Gentle pedal pump
5. Treatment of dysfunction in which single region would affect lymphatic drainage from lungs, kidneys, abdomen, and legs?
   A. OA
   B. C3–C5
   C. T1–T4
   D. Sacroiliac
   E. L4–L5

Section Two: Patient-Based Application Workshop

I. Description

This section includes the practical application of osteopathic treatment techniques to support the patient with a MI.

II. Psychomotor Components

(Refer to Section One for regions of the body that are involved.)

1. Examination of the patient using TART, including postural screen, palpation, segmental motion testing, and diagnosis of somatic dysfunction.
2. Application of philosophy and treatment technique.
3. Re-evaluation of the patient after treatment is completed to assess result. If a simulated patient is used, then the student or physician should verbalize length of treatment and future treatment goals.

III. Cognitive Components

1. Documentation in the medical record.
2. Post-treatment discussion.

Note. It is recommended to use the standardized outpatient form included in each of these chapters for documentation.
Critical Actions Evaluation Checklist of Osteopathic Principals
Applicable to a Patient with a Myocardial Infarction

<table>
<thead>
<tr>
<th>CRITICAL ACTION</th>
<th>COMPLETED</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Become familiar with the patient’s history physical examination findings, laboratory and other diagnostic findings.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perform an osteopathic structural examination.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Determine significant areas of somatic dysfunction.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Determine body region(s) to be treated with OMT.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apply OMT to at least the body region determined to be the most in need of treatment at present time.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treat other significant somatic dysfunctions if feasible.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Document treatment and immediately observable effects.</td>
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<td></td>
</tr>
</tbody>
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Trainer: ________________________________________________________________
Osteopathic Musculoskeletal Examination

**Required**

<table>
<thead>
<tr>
<th>Region Evaluated</th>
<th>Severity</th>
<th>Specific Major Somatic Dysfunctions</th>
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<tbody>
<tr>
<td></td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Head</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Neck</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Thoracic</td>
<td>T1-4</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>T5-9</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>T10-12</td>
<td>0</td>
</tr>
<tr>
<td>Lumbar</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Pelvis/Sacrum</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Pelvis/Innominate</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Extremity Lower</td>
<td>R</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>0</td>
</tr>
<tr>
<td>Extremity Upper</td>
<td>R</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>0</td>
</tr>
<tr>
<td>Ribs</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Other/Abdomen</td>
<td></td>
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</tr>
</tbody>
</table>

**Major Correlations with:**
- Traumatic
- Orthopedic
- Neurological
- Viscerosomatic
- Primary Musculoskeletal
- Cardiovascular
- Rheumatological
- EENT
- Cardiovascular
- Pulmonary
- Activities of Daily Living
- Genitourinary
- Other: 

**Scoliosis (Lateral Spine Curves)**
- None
- Functional
- Mild
- Moderate
- Severe

**Assessment Tools**
- T = Tenderness
- A = Asymmetry
- R = Restricted Motion
- Active
- Passive
- T = Tissue Texture Change

**Ant. Post. Spinal Curves:**

<table>
<thead>
<tr>
<th>Cervical Lordosis</th>
<th>Increased</th>
<th>Normal</th>
<th>Decreased</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoracic Kyphosis</td>
<td>Increased</td>
<td>Normal</td>
<td>Decreased</td>
</tr>
<tr>
<td>Lumbar Lordosis</td>
<td>Increased</td>
<td>Normal</td>
<td>Decreased</td>
</tr>
</tbody>
</table>

**Osteopathic Abbreviation Key**

OA: Occipito-Atlantal joint

Sympathetic Ganglia:
- C: Celiac
- S: Superior Mesenteric
- I: Inferior Mesenteric

TMJ: Temporomandibular joint

TMP: Temporal Bone

SBS: Sphenobasilar symphysis

**Severity Key**

0: No SD or background (BG) levels
1: Minor TART more than BG levels
2: TART obvious (R&T esp) +/- symptoms
3: Symptomatic, R and T very easily found “key lesion”

For coding purposes only.

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For [Image]