The role of osteopathic manipulative therapy in the treatment of coronary heart disease

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An imbalance between the supply and demand for oxygen in the myocardium is the basic physiologic factor in both angina pectoris and myocardial infarction, and infarction sometimes occurs when coronary arteriograms show no abnormality. The possible role of visceral-somatic and somatic-visceral mechanisms involving facilitation has been suggested, and observations on arterial spasm have been reported. These observations provide a rationale for the use of osteopathic manipulative therapy for patients with heart disease, and may explain the measurable improvement in electrocardiograms recorded after exercise.

A review of the osteopathic literature reveals that osteopathic manipulative therapy (OMT) has been demonstrated to be of benefit to some patients with angina pectoris and coronary insufficiency. While few studies have elucidated the biologic basis for the objective results obtained or the manipulative techniques utilized, we believe that significant evidence exists for postulating a biochemical and neurologic basis for the value of OMT as a tool in the therapeutic management of cardiac disease. Two case histories will be presented to support the most recent biologic explanations of the change in homeostatic control in some patients with angina pectoris and coronary artery disease and the theoretic basis for OMT.

Background

Previous experiments utilizing OMT for patients with coronary insufficiency have shown by objective criteria that OMT can be of significant value in the treatment of these patients. In one series, manipulation was followed by significant improvement in cardiovascular function, shown by changes in the electrocardiogram (EKG) after exercise before and after manipulation. The manipulative techniques employed, however, were not discussed. In another paper on cardiac patients tested with OMT, no objective criteria for improvement were reported, but various techniques were discussed. The author described cautious elevation of the second left rib with relaxing technique to the sternal end of the second rib. Frymann recently described a combination of techniques employing rib release, thoracolumbar manipulation, fascial release, and occipito-atlantal decompression. Techniques based on Chapman’s reflexes also have been described for both the diagnosis of cardiac disease by locating a tender nodule in the third or fourth left parasternal intercostal space and treatment by application of sustained pressure to the same nodule. (This has been observed by us to be of more therapeutic than diagnostic value.)

These reports were highly suggestive of a possible therapeutic role of OMT in the treatment of cardiac disease. Since “Truth is the confirmation of the mind with reality, and the reverse is fantasy,” it is necessary to observe the reality that patients with angina pectoris and coronary insufficiency do demonstrate improvement after proper manipulative approaches, directed either to relaxation and mobilization of certain rib structures or to pressure utilizing reflex arcs. Such validation goes beyond the purpose of this study. The purpose of this paper is to review recent knowledge of abnormal myocardial metabolism, and to supply a theoretic basis for the application of osteopathic principles.

Pathophysiology

The true nature of the cause of pain in angina pectoris has been the subject of speculation for many
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years, and the cause of both angina pectoris and myocardial infarction remains controversial. Keefer and Resnik\(^5\) stated that the basic physiologic factor involved is a state of imbalance between the myocardial demand for oxygen and the supply of oxygen, and Gross and Sternberg\(^6\) asserted:

This imbalance, depending on its severity and its duration, may lead to different phenomena, varying from transitory anginal pain to extreme myocardial damage. In its severest form it may lead to sudden death, with or without myocardial infarction.

Those authors reviewed studies that challenged the belief that coronary artery disease is the sole factor in producing this imbalance between the supply and the demand for oxygen. Other factors in nutrition to the myocardium also are involved. These include mechanical factors, hematologic changes, and reflex and humoral factors. Gross and Sternberg\(^6\) listed these as follows:

Factors Affecting Myocardial Nutrition

I. Mechanical Factors
   1. Fall in aortic blood pressure, especially in peripheral shock
   2. Phasic variations in coronary flow:
      Aortic insufficiency; arteriovenous fistula
      Aortic stenosis
      Hypertension
   3. Cardiac hypertrophy
   4. Tachycardia
   5. Failure of adequate collateral circulation; anomalies of coronary arteries

II. Changes in Blood
   1. Anemia
   2. Polycythemia

III. Reflex Factors
   (A) Coronary Vasomotor Reflexes
       Anoxemia due to failure of compensatory dilatation due to
       1. Inadequate reflex response
       2. Sclerosis of vascular walls
       (b) Marked vasoconstriction
       These reflexes may be classified as
       1. Reflexes originating in heart and associated structures
          (a) Proprioceptive reflexes adapting coronary flow to increased work, etc.
          (b) Carotic sinus reflexes
       2. Reflexes originating in different parts of body, especially
          (a) Those arising in abdominal viscera
          (b) Pulmonary reflex
       3. Reflexes originating centrally
   (B) Extracardiac Reflexes Affecting Coronary Flow
      Failure of adequate emptying of depot organs (liver, spleen, skin, etc.)
      1. Digestive state
      2. Variations in temperature

   3. Relaxed abdominal musculature

IV. Humoral Factors
   1. Epinephrine
   2. Histamine
   3. Acetylcholine
   4. Vasopressin, etc.

Evaluation of reflex factors must include visceral-somatic and somatic-visceral mechanisms involving facilitation, or lowering of the threshold, of motor pathways in lesioned segments. In such segments, said Korr\(^7\):

...the anterior horn cells (and probably the interneurons which synapse upon them) are maintained in a state of... lowered reflex thresholds. That is, they are hyperresponsive to impulses reaching them from any part of the body or from any part of the brain. It requires relatively few impulses, from any source, to initiate and sustain activity in these neurons, and their responses are exaggerated... Chronic segmental facilitation... extends to the other category of efferent neurons of the spinal cord, the lateral horn cells... Facilitation of these neurons results in increased impulse traffic to viscera and to blood vessels of virtually all tissues.

Autonomic nervous system

It is an oversimplification to describe the action of the autonomic nervous system in terms only of changes in the cardiac rate or coronary artery flow. Autonomic control extends right to the myocardial cell at the ultramicroscopic level where the sarcoplasmatic reticulum provides pathways for preferential diffusion of metabolites and intracellular spread of excitation.\(^8\)

Sympathetic innervation to the myocardium plays a vital role in contractility, nutrition, and metabolism. Braunwald\(^9\) stated:

Nerve terminals storing the sympathetic neurotransmitter norepinephrine are found throughout the heart in the sinoatrial and atrioventricular nodes, in the Purkinje system, and in the atrial and ventricular myocardium. The heart... can readily extract norepinephrine from the blood stream. Within the past few years it has been demonstrated experimentally... that the heart itself and its constituent sympathetic nerve endings can synthesize the neurotransmitter from its precursor amino acid tyrosine and need not be dependent on extraction. It now appears that fully 90% of the norepinephrine present in the heart is manufactured there. In this sense one can think of the heart as an endocrine gland that synthesizes and releases a hormone, norepinephrine, as needed to allow the circulation to respond appropriately to changing metabolic demands of body tissues.

He reported that a decreased amount of norepinephrine in the tissues of the heart in failure
has been demonstrated. Raab\textsuperscript{10} said that sympathetic activity is governed by the hypothalamus, with participation of the peripheral sympathetic ganglia and nerve endings. This activity, he said, is subject to reflex stimulation by stress from various sources, among which he listed physical exercise, emotional excitement or tension, sensory stimulation, use of nicotine, trauma, and infection. The effect of stimulation of the left stellate ganglion is similar to the action of catecholamines, which Nickerson\textsuperscript{11} said "increase the positive overshoot of the action potential and tend to increase its rate of rise." He said this occurs primarily through facilitation of the sodium-carrier mechanism of the cell membrane. The cardiotoxic action of the sympathogenic catecholamines reduces the myocardial glycogen stores, phosphocreatine, adenosine triphosphate, and potassium, thus creating alterations in myocardial intermediate metabolism that are identical with those seen in a patient with advanced degenerative heart disease with infarction and/or congestive failure.

Raab\textsuperscript{10} reported production of disseminated focal necroses in the subendocardial layers of the left ventricle by injections of epinephrine or norepinephrine and stated:

\ldots prolonged electrical stimulation of the norepinephrine-discharging cardiac sympathetic nerves \ldots has been found to elicit electrocardiographic signs of hypoxic myocardial damage and subendocardial hemorrhages and necroses.

The parasympathetic system also has a vital role in cardiac function. In early experiments Hall, Ettinger, and Banting\textsuperscript{12} showed that damage to the myocardium and coronary artery resulted from long continued daily administration of acetylcholine to unanesthetized dogs. In a further illumination of the mechanism of this action, Manning, Hall, and Banting\textsuperscript{13} utilized continuous stimulation of the vagus nerve in anesthetized and unanesthetized dogs. In lightly anesthetized dogs microscopic examination of the heart showed capillary congestion and hemorrhage with some early hyaline degeneration of the cardiac muscle. In unanesthetized dogs microscopic examination showed numerous areas of hyaline degeneration. One of the sections showed hemorrhagic areas around the small blood vessels, while others showed fibrous areas in the myocardium. Scattered areas of hemorrhage with infarcts were observed in the apex of the heart of one animal. Such myocardial damage did not occur in an animal given atropine but was increased in one given Eserine.

**Coronary blood flow**

Milnor\textsuperscript{14} provided the following description:

Myocardial metabolism is predominantly aerobic, so it is not surprising that cardiac muscle should need an increased blood supply when called upon to deliver an increased cardiac output, but the basic cause of this vasodilatation is not clear. \ldots Any procedure that increases the work or metabolic activity of the heart leads to an increase in coronary blood flow, and this indirect effect complicates investigation of the specific action of nerve impulses or other factors on the coronary vessels.

The pericoronary innervation is extraordinarily rich, full of cholinergic and adrenergic fibers. (The cholinergic fibers are more abundant around vessels of small caliber than around larger vessels.) Silber and Katz\textsuperscript{15} reported:

Both alpha and beta adrenergic receptors have been demonstrated in the coronary vascular bed \ldots and they subserve vasoconstriction and vasodilatation, respectively; stimulation of parasympathetic nerve fibers to the heart reduces coronary blood flow, whereas stimulation of sympathetic fibers increases the flow. These results have generally not been regarded as primary but rather as secondary to metabolic effects on the myocardium following catechol or nerve stimulation. Thus, parasympathetic stimulation, by reducing the heart rate and the atrial contribution, reduces the level of myocardial metabolism and thereby diminishes coronary blood flow; sympathetic stimulation, on the other hand, increases heart rate and myocardial contractile force with the result that coronary blood flow increases in response to the enhanced local metabolic demand.

The effect of epinephrine is the same as that of sympathetic stimulation.\textsuperscript{16} Paradoxically, acetylcholine possesses the same action as epinephrine. Vasopressin (pitressin) produces marked vasoconstriction.

**Spasm of coronary artery**

The occurrence of angina pectoris and/or myocardial infarction in the absence of demonstrable coronary thrombosis or total occlusion of a coronary
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artery has been well documented, and as a result interest in the theory that spasm of a coronary artery is a major factor in induction of episodes of either angina or infarction has been renewed. Kattus reported the dramatic case of a 55-year-old woman with intense substernal nocturnal angina who underwent cardiac surgery. At the time of operation the surgeon observed intense spasm of the right coronary artery, during which the formerly soft, pliable vessel was converted into a firm cord much reduced in diameter and with the consistency of stout twine. The spasm was relieved in about 1 minute after a nitroglycerin tablet was placed under the patient's tongue and 1 percent lidocaine solution was sprayed over the exposed vessel.

Most studies of spasm of a coronary artery as a cause of angina have referred to the large portion of the artery seen during coronary angiography of human beings or at surgery. In exploring the possibility that stasis of a coronary artery may produce secondary thrombosis, Hellstrom measured blood flow in a coronary artery in 17 dogs by electromagnetic flow techniques from 4 to 7 days after infarction was produced by occlusion of a coronary artery for 1 hour. In 15 of 16 dogs available for study he reported "marked reduction of flow in the anatomically patent but previously occluded left circumflex artery." He said the stasis appeared to be due to spasm of small intramyocardial arterial branches, because "flow patterns were 'spastic' and stasis was reversed by ischemia, a potent coronary artery vasodilator."

Prinzmetal's variant angina is a form of angina that was first believed to be due to severe vascular disease, but reports on coronary angiograms in a number of cases showed less than 50 percent stenosis of coronary arteries, normal coronary arteries, and, in some patients, coronary arterial spasm. Prinzmetal's variant angina, in contrast to effort angina, occurs almost exclusively during rest, is associated with transient elevation in the S-T segment, and frequently is accompanied with complete atrioventricular block or ventricular tachycardia.

Case reports

The following two cases are presented to show the dynamic nature of coronary heart disease. For too long medicine has been under the influence of pathologists who view the heart only in its static, postmortem condition, from which they attempt to deduce the sequence of events leading to death. Case 1 is an example of unquestionable evidence of coronary heart disease with minor changes observed in the coronary arteries at angiography.

Case 1

A 41-year-old white woman was seen on January 27, 1976, with a history of "sudden onset of weakness" experienced in October 1975. At that time she had been helping her husband pour concrete, when she rapidly became extremely weak, diaphoretic, dyspneic, and nauseated. Her symptoms lasted approximately 15 minutes. A similar episode had occurred on December 26, 1975, when she was shoveling snow. On January 20, 1976, the same symptoms had been experienced in association with discomfort below the xiphoid process. No radiation of the discomfort was experienced, on December 26, 1975, cian prescribed a diet suitable for hypoglycemia, but fatigue increased.

There was no history of previous dyspnea, orthopnea, syncope, coital discomfort, nocturia, or murmurs. Pretibial edema had been associated with estrogen ingestion for the past 3 years, and there had been frequent leg cramps. There was no history of rheumatic fever, growing pains, scarlet fever, chorea, or hypertension. She had undergone surgery only for a vocal nodule. Her last menstrual period had begun 1 week ago. She had undergone surgery for a vocal nodule. Her last menstrual period had begun 1 week ago. She had two living children and one child had died in infancy because of respiratory insufficiency. She had no drug allergies, and at present was using Hormonin No. 2 and Tranxene.

She had smoked one pack of cigarettes per day for
20 years and took one alcoholic drink before dinner daily. Her use of salt was of Class 2.* She used under-arm and household sprays.

Physical examination showed her blood pressure to be 120/72 mm Hg in the right arm. Her thyroid gland was not enlarged, and the cervical veins were not distended when she was in the supine position. The carotid arteries were normal to palpation. The heart rate was 61 beats per minute, with regular rhythm, and no thrills were palpable. There was a Grade 1 or 2 systolic murmur at the left sternal border. There were no clicks when she assumed the sitting position after ten sit-ups. There were no masses in the breasts. The lung fields were clear to auscultation. The liver and spleen were not palpable, and no bruits were audible. There was no evidence of pretibial edema. Pulses in the femur and dorsum pedis were equal and full.

A phonomechanocardiogram recorded the first and second heart sounds to be normal. There was an early and midsystolic murmur, most noticeable at the left sternal border. There were no third or fourth sounds or opening snap. Studies of left ventricular function showed an ejection time of 0.305 second corrected at a rate of 68 per minute, which is 103 percent of normal. The pre-ejection phase was 0.115 second. The systolic time interval was 0.38 (normal: 0.35 + 0.04) second. The systolic quotient was 2.65 (normal: 2.5 to 5.0). The isosystolic time was 0.45 (normal: 0.015 to 0.045) second. The end diastolic index was A/EO = 18 percent (normal: 25 percent or less), a/EO = 4 percent (normal: 15 percent or less).

A multiple-stage treadmill stress electrocardiogram (EKG) was made after a resting test showed a sinus rate of 72 per minute and a borderline normal EKG (Fig. 1). There was no change after hyperventilation. The EKG during stress utilizing the Bruce protocol showed ischemic changes at a heart rate of 172 per minute (target rate, 160). The test was interrupted at Stage III, at 2.8 minutes (total time, 6.8 minutes) because of fatigue and dyspnea. Severe chest pain and nausea developed approximately 1 minute after stress. The EKG immediately after stress showed sinus tachycardia at a rate of 120 per minute, with marked anterolateral subendocardial injury believed to be reciprocal from inferior subepicardial injury (Fig. 2). The EKG 1 minute after stress showed marked inferior subepicardial injury characteristic of recent infarction (Fig. 3). The EKG 10 minutes after stress resembled the tracing during rest. Nitroglycerin was given sublingually, oxygen nasally, and Demerol intramuscularly within 1 minute of the onset of chest pain. Intravenous infusion of 500 ml. of 20 percent dextrose solution and water with potassium chloride, 20 mEq., and regular insulin, 15 units added, was begun immediately. The EKG returned to the resting level within 10 minutes, but because of the possibility that acute inferior myocardial infarction had developed, the patient was admitted to the intensive care unit. She continued to have mild anterior chest discomfort, which receded during the next hour. The EKG remained similar to the resting tracing. During hospitalization, enzyme values remained within normal limits.

On February 2, 1976, left heart catheterization with coronary angiography was performed by the Sones technique. The left ventricular pressure was 160/0 mm Hg, and after injection, the end-diastolic pressure was 12 mm Hg (normal, less than 12 mm.). The aortic pressure was 160/96 mm Hg and the mean pressure 100. The patient tolerated the procedure well with no complications.

Figures 4A and 4B show minor irregularities in the right coronary artery. Figures 5A and 5B show the left coronary artery. The left anterior oblique view shows 20 percent stenosis of the middle third, which is not significant. Figure 6 shows the left ventricle in systolic phase, with satisfactory contractility.

*Classification for use of salt:
4+—Salts food before tasting
3+—Salts food with shaker after tasting
2+—Does not use salt shaker at the table, but is not careful about buying or cooking foods low in salt
1+—Buys and cooks with care to avoid salt and other sodium compounds
Fig. 1. Borderline normal electrocardiogram in case 1, recorded before stress test.

Fig. 2. Electrocardiogram in case 1 after stress test. The atrial T wave in lead 2 was considered normal, but there was evidence of reciprocal subendocardial injury in leads V4, V5, and V6.
Fig. 3. Electrocardiogram in case 1 recorded 1 minute after end of stress test, showing marked elevation in S-T segment in lead 3.
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in comparison with the diastolic phase (Fig. 7). Final impression was of angina pectoris with essentially normal coronary arteries.

Case 2

A 55-year-old white woman was admitted to the hospital on February 28, 1976, with the complaint of sharp substernal chest pain which usually followed activity and radiated down the left arm, associated with dyspnea and diaphoresis. The duration of pain was approximately 5 minutes, and it seemed to be relieved with rest. Symptoms had begun approximately 3 years before, and had become increasingly frequent. Occasionally they awakened her at night. Dyspnea was experienced when she climbed approximately one flight of stairs. There was no orthopnea. Fatigue had increased over the past year. There was no pretibial edema. Nocturia was present, about once per night. The patient stated that dyspnea occurred with coitus but did not interrupt coitus. She had no history of a previous murmur, rheumatic fever, scarlet fever, glomerulonephritis, or chorea and did not have hypertension, a chronic cough, or hemoptysis. She had frequent dysphagia, related to a hiatal hernia. She did not experience odynophagia, hematemesis, or melena but had chronic diarrhea alternating with chronic constipation. She had smoked for approximately 1 year 25 years before, did not use alcohol, and was of Class 4 in the use of salt.

Her surgical history included five curettages, one tubal ligation, appendectomy, tonsillectomy and adenoidectomy, hysterectomy with oophorectomy, bilateral bunionectomy, and hemorrhoidectomy. She had no allergies.

During history taking the patient cried occasionally. At physical examination the thyroid gland was not palpable, and the cervical veins were not distended when she was supine. The carotid arteries were normal to palpation bilaterally, without bruits. Bilateral auscultation showed the lungs to be essentially clear. The apical heart rate was 80 beats per minute with regular rhythm. No murmurs or extra sounds were heard. There were no masses in the breasts. The abdomen was soft, without masses or organomegaly. Pulses in the femur and dorsum pedis were present and equal bilaterally. There was no pretibial edema. The Master two-step test gave a result on the borderline of abnormality.

The clinical impression was of angina pectoris due to arteriosclerotic heart disease, and sliding hiatal hernia (from the history).

On March 22, 1976, left heart catheterization with coronary angiography was performed by the Sones technique.

The left ventricular pressure was 156 mm. Hg, with end-diastolic pressure of 12 mm. Hg. After injection the end-diastolic pressure was 14 mm. Hg (normal less than 12 mm.). The aortic pressure was 156/100 mm. Hg and the mean pressure 124.

The right coronary artery showed marked spasm of the proximal third (Fig. 8). Figure 9 shows partial relaxation of the spasm following sublingual nitroglycerin, and Figure 10, taken later, shows a normal coronary artery. The left coronary artery appeared normal in its entirety (Figs. 11A and 11B).

The clinical impression was of angina pectoris due to spasm of the coronary artery.

It should be noted that angiography in many cardiac laboratories, nitroglycerin is given routinely before the first injection of contrast medium. This was not done in the present case, and there is a possibility that when prophylactic administration of nitroglycerin is not used spasm may be seen with increased frequency.

Comment

Osteopathic manipulative therapy has been demonstrated to be of significant value in some patients with coronary insufficiency. This paper reviews the physiologic mechanisms involved in normal and compromised cardiac metabolism and provides a theoretical explanation for the observed improvement in cardiovascular function following osteopathic manipulation. The role of abnormal myocardial metabolism in the production of coronary insufficiency has received much attention in recent years. This concept challenges the idea that atherosclerotic phenomena are responsible for both angina pectoris and myocardial infarction. The
Figs. 4A and 4B. Angiograms of right coronary artery, showing minor irregularities (case 1).

Figs. 5A and 5B. Angiograms of left coronary artery, showing insignificant lesion of anterior descending branch (case 1).

Fig. 6. Left ventriculogram in systolic phase in case 1, showing satisfactory contractility. Fig. 7. Left ventriculogram in diastolic phase in case 1.
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Fig. 8. Angiogram of right coronary artery in case 2, showing spasm in proximal portion. Fig. 9. Angiogram of right coronary artery in case 2 after sublingual administration of nitroglycerin, showing partial relaxation of spasm. Fig. 10. Later angiogram of right coronary artery in case 2, showing no abnormality of artery.

Figs. 11A and 11B. Angiograms of left coronary artery in case 2, showing no abnormality.
pathophysiologic basis of abnormal myocardial metabolism in the absence of coronary artery disease is as yet controversial, but involves changes in cellular mechanisms as well as in autoregulatory control of coronary artery flow.

Dilatation of the coronary arteries and their branches is the normal response to an increase in the metabolic needs of the myocardium. This response is believed to be neurohumoral, mediated partly through the autonomic nervous system. In ischemic heart disease there is a disturbance in the normal response of coronary vasodilatation to meet increased oxygen demands, and in some cases spasm of a coronary artery occurs. This is an exaggerated paradoxical reaction. The two cases presented here illustrate the occurrence of unquestioned ischemic heart disease with essentially no abnormality on coronary angiograms and the presence of angina pectoris with transient spasm of a coronary artery.

It is logical to assume that manipulative treatment, by normalizing the action of the autonomic nervous system, might influence both cellular metabolism and the vasomotor dynamics of the coronary arteries.

2. Johnson, F.E.: Some observations on the use of osteopathic therapy in the care of patients with cardiac disease. JAOA 71:799-804, May 72
3. In cardiac patients: Osteopathic manipulation held to aid heart function. Clin Trends Osteop Med 2:1, 5, Mar-Apr 76
4. Wolfe, H.: Personal communication