THOMSON
AMERICAN HEALTH CONSULTANTS

The Practical Journal for Emergency Physicians

Emergency Medicine Reports

Volume 24, Number 17 August 11, 2003

Noncardiac Causes of Chest Pain in the Emergency Department: Part I

Authors: **Eric Anderson, MD, MBA, FACEP**, Associate Director of Clinical Operations, Cleveland Clinic, OH; Assistant Professor of Emergency Medicine, The Ohio State University, Columbus; and **Jonathan Glauser, MD, MBA, FACEP**, Cleveland Clinic Foundation, Assistant Clinical Professor of Medicine, Case Western Reserve University; Faculty, Residency Training Program in Emergency Medicine, MetroHealth Medical Center, Cleveland, OH.

Peer Reviewers: **Charles V. Pollack, Jr., MA, MD, FACEP**, Chair, Department of Emergency Medicine, Pennsylvania Hospital, Associate Professor of Emergency Medicine, University of Pennsylvania School of Medicine, Philadelphia; and **William B. Ignatoff, MD, FACEP**, Emergency Physician, Carondelet Health Network, St. Joseph's and St. Mary's Hospitals, Tucson, AZ.

Chest pain and coronary ischemic symptoms (i.e., jaw pain, back pain, epigastric pain, shoulder pain, breathlessness, etc.) are common complaints in the emergency department (ED). The diagnosis of patients with chest pain is straightforward only occasionally, when there are diagnostic electrocardiogram (ECG) changes, radiographic findings, laboratory studies, or physical examination findings. In many cases, these findings are not present, and the differential diagnosis is broad. The emergency physician's primary responsibility is to exclude life-threatening disease, such as acute myocardial ischemia, pulmonary embolism, tension pneumothorax, aortic disasters, esophageal perforation, etc. Much has been written in the past several years (often driven by risk management concerns) about the evaluation of cardiac pain syndromes. This article focuses instead on the evaluation and initial management of the myriad causes of noncardiac chest pain (NCCP).

The evaluation of chest pain in the ED can provoke anxiety in the patient, the patient's family, and the caregivers. A systematic method of evaluating these patients is essential to assess for potentially life-threatening conditions. Often, a concern about coronary ischemia precipitates the ED visit. This article discusses noncardiac causes of chest pain. For the purpose of this article, cardiac causes are considered to be all causes within and including the pericardium. Several noncardiac causes are imminently life-threatening and will be discussed in this series. Imminent life-threatening causes include tension pneumothorax, pulmonary embolus, and aortic dissection. Other conditions will be addressed using an organ system approach, including gastrointestinal (GI), pulmonary, neurological, chest wall, and other disorders that can manifest with chest pain. Part I of this article will cover GI causes of chest pain and aortic dissection. Part II will focus on

pulmonary, psychiatric, and musculoskeletal causes of chest pain.

—The Editor

Epidemiology of Noncardiac Chest Pain

NCCP is a common occurrence, with a prevalence of 12% in the general population.[1] It has been estimated that there were 1.5 million cardiac catheterizations in 1989 to evaluate patients with chest pain of suspected coronary origin. Of those, approximately one-third (450,000) had normal coronary arteries and presumably a non-coronary cause of pain.[2] Many more patients had less invasive evaluations to assess the etiology of their chest pain.[3] Data from a teaching hospital ED recorded during a 10-month period indicated that 75% of patients evaluated for chest pain were felt to have a noncardiac cause of their chest pain.[4] Considering the catastrophic consequences of missed myocardial infarction (MI) or misdiagnosed myocardial ischemia, it is reasonable to rule out the most life-threatening diagnosis first. In many cases this can be done with reasonable certainty with a thorough history and physical exam, and in some cases basic studies (i.e., ECG, x-ray, and lab) will be helpful. In cases where there is doubt, it is advisable to evaluate for the diagnosis with the most lethal result first and then explore other diagnoses.

This article considers patients in the ED with chest pain who have been found, through prior cardiac workup, not to have a cardiac cause of their pain. Patients who continue to seek care and treatments for their chest pain each will spend approximately $3500 per year.[5,6] Estimates from the late 1980s indicate that more than $1.8 billion is spent per year in the United States on treatment of patients with NCCP. (*See Table 1.*) That amount may be considerably higher, considering inflation, increased patient volumes, and the ever-increasing cost of more sophisticated diagnostic technology seen today.

Patients with NCCP are known to have poor symptomatic, functional, and psychological outcomes.[6] Many desire additional clinical evaluations and feel that there may be significant disease that was missed.[7] Follow-up studies of these patients indicate that the seven-year mortality rate of patients with chest pain and normal coronary arteries is less than 1%.[8]

The etiology of NCCP varies from imminently life-threatening to musculoskeletal and psychiatric conditions. It has been estimated that gastroesophageal reflux disease (GERD) causes 20-40% of cases of NCCP.[1] Another study indicated that up to 50% of NCCP patients will have an "acid-induced abnormality" as the etiology of their chest pain.

Gastrointestinal Causes of Noncardiac Chest Pain

Pathophysiology. Esophageal and cardiac sensory afferent fibers enter the spinal cord at the same levels. It therefore is not surprising that esophageal pain can mimic cardiac pain in distribution and quality.[9]

Esophageal Perforation. Esophageal perforation (Boerhaave's syndrome) is the most lethal esophageal cause of NCCP and is the most serious perforation of the GI tract.[10] Typically this problem occurs in patients older than 50 years of age who have an underlying esophageal pathology such as esophageal cancer. Esophageal perforation also can be seen in post-endoscopy patients or in those with a retained esophageal foreign body. Symptoms include chest pain, fever, and painful respirations. The physical exam classically reveals Hamman's crunch and subcutaneous emphysema in the neck. A chest x-ray may reveal mediastinal emphysema, pneumothorax, widening of the mediastinum, and often a left pleural effusion.[11]

Survival rates are inversely related to the time it takes to make the diagnosis and initiate treatment. Esophagography usually will demonstrate extravasation of water-soluble contrast material into the mediastinum. ED treatment consists of fluid resuscitation, antibiotic administration, and prompt surgical consultation.

Gastroesophageal Reflux Disease. GERD exists when the

Emergency Medicine Reports™ (ISSN 0746-2506) is published biweekly by Thomson American Health Consultants, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Vice President/Group Publisher: Brenda Mooney
Editorial Group Head: Valerie Loner
Specialty Editor: Shelly Morrow Mark
Marketing Manager: Schandale Kornegay
GST Registration No.: R128870672

Periodicals postage paid at Atlanta, GA. **POSTMASTER:** Send address changes to ***Emergency Medicine Reports***, P.O. Box 740059, Atlanta, GA 30374.

Back issues: $31. Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

Multiple copy prices: One to nine additional copies, $359 each; 10 to 20 additional copies, $319 each.

Accreditation

Emergency Medicine Reports'™ continuing education materials are sponsored and supervised by Thomson American Health Consultants. Thomson American Health Consultants designates this continuing education activity for up to 60 hours in Category 1 credit toward the AMA Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

This CME activity was planned and produced in accordance with the ACCME Essentials.

Emergency Medicine Reports™ also is approved by the American College of Emergency Physicians for 60 hours of ACEP Category 1 credit and has been approved for 52 Category 2B credit hours by the American Osteopathic Association. Emergency Medicine Reports has been reviewed by the American Academy of Family Physicians as having educational content acceptable for Prescribed credit hours. This volume has been approved for up to 60 Prescribed credit hours. Term of approval covers issues published within one year from the beginning distribution date of 1/03. Credit may be claimed for one year from the date of this issue.

Thomson American Health Consultants (AHC) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

This is an educational publication designed to present scientific information and opinion to health professionals, to stimulate thought, and further investigation. It does not provide advice regarding medical diagnosis or treatment for any individual case. It is not intended for use by the layman. Opinions expressed are not necessarily those of this publication. Mention of products or services does not constitute endorsement. Clinical, legal, tax, and other comments are offered for general guidance only; professional counsel should be sought for specific situations.

Subscriber Information

Customer Service: 1-800-688-2421

Customer Service E-Mail: customerservice@ahcpub.com
Editorial E-Mail: shelly.morrow@ahcpub.com

World Wide Web page: http://www.ahcpub.com

Subscription Prices

1 year *with* 60 ACEP/60 AMA/60 AAFP Category 1/Prescribed credits (52 AOA Category 2B credits): $544
1 year *without* credit: $399
Resident's rate $199

All prices U.S. only.
U.S. possessions and Canada, add $30 plus applicable GST. Other international orders, add $30.

For Customer Service and CME questions,

Please call our customer service department at **(800) 688-2421.** For editorial questions or comments, please contact **Shelly Morrow Mark,** Specialty Editor, at shelly.morrow@ahcpub.com or (404) 262-5514.

THOMSON
AMERICAN HEALTH CONSULTANTS

Statement of Financial Disclosure

In order to reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Drs. Anderson, Glauser (authors), Pollack, and Ignatoff (peer reviewers) report no relationships with companies related to the field of study covered by this CME program. Dr. Bosker (editor) is on the speaker's bureau for Pfizer, Rhone-Poulenc Rorer, and Parke-Davis. Dr. Bosker also acknowledges that he receives royalties, commissions, and other compensation relating to the sale of textbooks, reprints of articles, and other written materials to the following pharmaceutical companies: Pfizer, Genentech, Aventis, Pharmacia, and Bayer.

Table 1. Epidemiology of Noncardiac Chest Pain[1,2,5,6,8]

Prevalence	12% of general population
Negative cardiac catheterizations	20-30% of all cardiac catheterizations
Negative cardiac catheterizations mortality	< 1% over 7 years
Annual NCCP patient care expense	$ 1.8 billion (1989)
Disability of NCCP patients	50% not working
Heart awareness programs	Increased ED evaluations for chest pain
Liability of missed myocardial infarction	Highest % of ED malpractice dollars paid

pH of the esophagus is persistently below 4.0. This results in macro- and micro-inflammatory changes to the esophagus that cause pain either by direct injury or by altering the pain sensation threshold in the area.[12] It originally was thought that GERD caused direct injury to the esophageal mucosa, resulting in chest pain. More recent investigators have found that chronic acid exposure of the lower esophagus seems to lower the pain threshold when compared to controls.[12-15]

The clinical presentation of GERD may be indistinguishable from acute coronary ischemia. Typically, there is a burning mid-chest pain that may be associated with nausea, diaphoresis, and shortness of breath. As mentioned earlier, these patients should have documented a normal coronary study before a diagnosis of GERD is pursued. A typical outpatient diagnostic strategy in GERD is ambulatory pH monitoring. Endoscopy is diagnostic of GERD in fewer than 10% of cases and rarely is used to diagnose the condition.[8,12]

The Omeprazole Challenge Test (also known as the acid suppression test) is omeprazole 20-40 mg in the morning and 20 mg in the evening for one week. Several studies have shown significant symptomatic relief with this regimen. The treatment group responded 78-81% vs. a placebo response of 6-14%.[3,12,17] The Omeprazole Challenge Test has been found to be both diagnostic and therapeutic in patients with suspected GERD.[12] One study demonstrated a cost savings of $573 per patient if the acid suppression test was used instead of typical diagnostic protocols. This would result in a 59% reduction in the number of evaluative procedures.[17] Patients who fail the acid suppression test should have further evaluation for the etiology of their NCCP. Further evaluation includes chest x-ray (CXR), ambulatory pH monitoring, esophageal manometry, endoscopy, esophogram upper GI, and chest computed tomography (CT).

Esophageal Motility Disorders. Esophageal motility disorders are a group of pathologic conditions characterized by abnormal peristaltic movements of the esophageal musculature in response to a food bolus. Esophageal motility disorders include achalasia, diffuse esophageal spasm, nutcracker esophagus, hypertensive lower esophageal sphincter (LES), and nonspecific esophageal motility disorder. Manometric definitions of these clinical entities are illustrated in Table 2. Achalasia, scleroderma, and Chagas disease have specific pathological findings that are identifiable histopathologically.[18] Nutcracker esophagus, hypertensive LES, and nonspecific esophageal motility disorder are diagnosed clinically based on manometric patterns at the LES. They have no reliably identifiable histopathologic findings.[18] In Chagas disease, achalasia, and diffuse esophageal spasm there is evidence of inflammatory cell infiltration, fibrosis, and damage to the intrinsic nervous system of the esophagus, especially in Auerbach's plexus.[18] Patients with various motility disorders demonstrate a higher rate of gastroesophageal reflux than do controls.[18,19]

A study in 1999 found "considerable overlap" in the abnormalities of esophageal motility in patients with documented coronary disease and esophageal dysmotility patients with documented normal coronary arteries. Both groups "differed significantly from controls."[20] This implies that a patient with documented motility disturbance may have a higher incidence of associated, clinically significant coronary disease. This reinforces the idea that coronary artery disease must be considered in these patients even if they have a documented esophageal motility disturbance but no documented evaluation of their cardiac status.

Treatment of patients with esophageal motility disorders is supported only by anecdotal reports. Pharmacologic treatment results have been inconsistent. Treatment has been attempted with calcium channel blockers, nitrates, and antimuscarinics. Diltiazem and nifedipine have been used for this purpose with mixed results.[1,9] Endoscopic treatment includes balloon dilation of the LES or Botulinum toxin injections into the LES. Surgical treatments include Heller myotomy or long myotomy. Emergency physicians should refer these patients for GI follow-up.

Syndrome X. Syndrome X is a medical condition in which the patient presents with chest pain that appears anginal in nature and is associated with objective signs of ischemia in exercise testing or myocardial scintography, but with normal coronary arteries.[2] The cause of this syndrome is not known. Researchers studied esophageal dysfunction in Syndrome X in 1998. They found that, of 20 patients studied, 67% experienced pain with esophageal balloon distension, 47% experienced pain with esophageal acid perfusion, and 37% had abnormal esophageal motility. Only five of the 20 reported typical symptoms associated with esophageal dysmotility.[21] These findings suggest the presence of visceral hypersensitivity, even in patients with abnormal exercise cardiac testing.[2]

Visceral Hyperalgesia. In visceral hyperalgesia, the patient is cognizant of noxious stimuli at lower pain thresholds than controls. The patient may have NCCP precipitated by esophageal balloon distension at lower balloon volumes than controls.[2] One study demonstrated that acid infusion in the lower esophagus could lower the pain threshold in the upper esophagus and the anterior chest wall. This area of hypersensitivity was well outside the area of acid exposure and was felt to be due to a change in sensory processing within the central nervous system.[13] The basic principles behind visceral hyperalgesia involve allodynia and hyperalgesia. Allodynia is the painful perception of a previously innocuous stimulus. Hyperalgesia is exaggeration of painful sen-

Table 2. Modern Classification of Esophageal Motility Disorders

1. ACHALASIA
- a. Absence of peristalsis in esophageal body
- b. Incomplete lower esophageal sphincter (LES) relaxation
- c. Elevated LES pressure (normal levels depend on individual labs)
- d. Increased intraesophageal baseline pressure relative to gastric baseline

2. DIFFUSE ESOPHAGEAL SPASM (DES)
- a. Simultaneous (non-peristaltic) contractions occurring after > 10% of wet swallows
- b. Periods of normal peristalsis
- c. May see spontaneous and/or repetitive contractions
- d. Contractions may be of increased amplitude or duration
- e. LES pressure may be elevated (one-third of patients)

3. NUTCRACKER ESOPHAGUS
- a. Mean peristaltic amplitude in distal esophagus ≥ 180 mmHg
- b. Normal peristaltic sequence preserved
- c. May have prolonged duration (≥ 6.0 seconds)

4. HYPERTENSIVE LES
- a. LES pressure ≥ 2 standard deviations above normal mean with normal relaxation
- b. Normal peristalsis

5. NON-SPECIFIC ESOPHAGEAL MOTILITY DISORDER
- a. Reduced amplitude of esophageal peristalsis
- b. Triple peaked contraction waves
- c. Simultaneous contractions (≤ 10% of wet swallows)
- d. Spontaneous contractions
- e. Isolated prolonged duration contractions

Adapted from: Pirtniecks A, Smith LF, Thorpe JA. Autonomic dysfunction in non-specific disorders of eosophageal motility. *Eur J Cardiothoracic Surg* 2000;17:101-105.

Table 3. Treatment of GI Causes of Chest Pain

GERD
- Omeprazole, 20 mg bid (4-8 wks)
- Omeprazole (high dose), 40 mg in a.m., 20 mg in p.m.

ESOPHAGEAL DYSMOTILITY DISORDERS
- Nitrates: Nitroglycerine, 0.4 mg SL prn pain *OR* Isosorbide dinitrate, 10-30 mg bid
- Calcium channel blockers: Nifidipine, 10-30 mg *OR* Diltiazem, 90 mg, both qid immediately before meals or as needed for pain. Watch for hypotension.

VISCERAL HYPERALGESIA
- Imipramine, 50 mg at bedtime
- Trazodone, 100-150 mg daily

BOERHAAVE'S SYNDROME
- Prompt diagnosis
- IV fluids
- Antibiotics
- Prompt surgical referral

sation. Both result from the aberrant processing of visceral stimuli. Repeated irritation of the lower esophagus secondary to GERD can lead to both allodynia and hyperalgesia. Esophageal balloon distension can yield similar results.[2,9]

Pharmacologic treatment is aimed at blunting the visceral sensations.[2] Tricyclic antidepressants at low doses are known to decrease pain of visceral or somatic origin. Imipramine 50 mg at bedtime has been found to be an effective treatment for visceral pain.[2,14,22] (*See Table 3.*) Trazodone (100-150 mg per day) can yield significant improvement in six weeks.[2,14] Promising drugs still under study include octreotide, a synthetic analogue of somatostatin; alosetron, a potent selective 5-HT3 antagonist; and fedotozine, a kappa opioid agonist.[2]

Aortic Dissection

Aortic dissection is the most common and most lethal emergency involving the aorta and is approximately 2-3 times more common than ruptured abdominal aortic aneurysm. The reported incidence is approximately 10-20 per million population annually, with an estimated 4500 new cases per year.[23,24] While aortic dissection is not common relative to acute myocardial infarction, it is an entity that emergency physicians are likely to encounter during their careers. One autopsy study indicated that acute dissections were the cause of sudden deaths in 4.2% of cases reviewed over a three-year period.[25]

To put the lethality and urgency of acute aortic dissection in perspective, the estimated mortality untreated is 28% within 24 hours, 50% at 48 hours, 70% within one week, and 90% within three months.[25,26]

More than perhaps any other disease entity, aortic dissection may be considered the great imitator. Depending upon which branch vessels are compromised, aortic dissection may present with myocardial infarction, pericardial tamponade, acute central nervous system events including syncope or stroke, intestinal or spinal cord ischemia, or renal insufficiency.[27]

The term "aneurysme dissequant" first was coined by Laennec in 1819.[28] It is unfortunate that this terminology has persisted in some circles, since aortic dissection is not an aneurysm, and the aorta rarely is aneurysmal. Aortic dissection is a separation of the layers of the aortic wall with variable proximal and distal extension.[29]

Acute aortic dissection is defined if it has been present less than two weeks. Chronic aortic dissection, by definition, has been present for more than two weeks.

Classification. There are two widely used classifications, the DeBakey and the Stanford. The DeBakey system first was proposed in 1955. Those who feel that prognosis and treatment is based upon whether the dissection is proximal or distal may prefer to use the Stanford classification.

DeBakey:

Type I: Dissection originates in the ascending aorta and extends beyond the ascending aorta;

Type II: Dissection confined to the ascending aorta;

Type III A: Origin in the descending thoracic aorta, dissection does not extend past the diaphragm;

Type III B: Origin in the descending thoracic aorta, dissection extends beyond the diaphragm;

Stanford:

Type A: All dissections of the ascending aorta (DeBakey Types I and II): 60-65% of cases;

Type B: All dissections distally (DeBakey III): 30-35% of cases.[30]

Risk Factors and Epidemiology of Aortic Dissection. Chief among risk factors for aortic dissection is hypertension. Conversely, aortic dissection constitutes a true hypertensive emergency. It shows a male predominance of approximately 3:1, most commonly affecting patients in the fifth through seventh decades of life. Importantly for ED patients, cocaine use[31] and activities that may cause sudden rises in blood pressure such as weight lifting have been implicated as triggers for aortic dissection.

Connective tissue disorders such as Ehlers-Danlos or Marfan's syndrome are risk factors. Other aortic disorders, such as giant cell arteritis or systemic lupus, may predispose to dissection. Congenital disorders of the aortic valve, such as coarctation, unicommisural, or bicuspid valves, represent risk factors as well.

Patients with Type A dissections tend to be younger and to have a higher incidence of connective tissue disorders or of congenital disorders of the aortic valve. Patients with Type B dissections tend to be older, and to have more factors associated with circulatory ailments such as smoking history, hypertension, generalized atherosclerotic disease, and chronic pulmonary disease.

Symptoms of Aortic Dissection. The classic description of pain associated with aortic dissection is that of tearing chest pain, sudden in onset, of a ripping or unbearable quality. It is maximal in severity at onset, and often is described as "knifelike." Proximal dissections may cause radiation of pain to the anterior chest, neck, jaw, and arms. Distal dissections may present with intrascapular pain or back pain. With propagation of the dissection, the pain may migrate.

Since dissections may cause obstruction of any branch vessel from the aorta, other symptoms may predominate. GI symptoms may result from mesenteric ischemia. Hematemesis may result from rupture into the esophagus. Dyspnea may be due to aortic insufficiency, and pulmonary edema may result from compression of the trachea or main stem bronchi, or from erosion into the tracheobronchial tree with hemoptysis.

Neurologic symptoms may predominate in up to 20% of aortic dissections. These symptoms may wax and wane due to spinal cord or peripheral nerve ischemia, or due to stroke. If the patient has altered mentation, the pain history may be unclear.

In general, the patient with aortic dissection is anxious. Pulsus paradoxus may be present from pericardial tamponade. Blood pressure may be high (11%), normal (66%), or low (23%). If hypotension is present, it most commonly is due to aortic insufficiency, pericardial tamponade, aortic rupture, or pseudohypotension due to subclavian artery obstruction. A murmur of aortic insufficiency is present in approximately 50%. Since branch vessels may be occluded by the false lumen or by emboli, blood pressure differences of 15-20 mmHg between arms may be present and should be elicited. Pulmonary edema may occur from aortic insufficiency.

Differential Diagnosis. Because aortic dissection may cause a variety of symptoms related to the extent of dissection, the differential diagnosis is quite broad. It is helpful to separate these into broad categories. (*See Table 4.*) It is particularly useful, however, to recall two points. First, aortic dissection may cause myocardial infarction (MI) through coronary artery involvement in the dissecting process (especially the right coronary artery). Thrombolysis for apparent MI in this setting is dangerous. Second, there is little else that causes chest pain and acute focal neurologic deficit other than aortic dissection.

Diagnostic Testing. Tests instituted in the ED evaluation of chest pain may include CXR and ECG. Although patients with aortic dissection have some abnormality on the CXR 80-90% of the time, these findings often are non-specific. Mediastinal widening or separation of intimal calcium of greater than 6 mm from the aortic wall may be present. There may be a disparity in size between the ascending and descending aorta, blurring of the aortic knob, or depression of the left main stem bronchus. If rupture has occurred, there may be a left apical cap or hemothorax.

The ECG may show left ventricular hypertrophy from long-

Table 4. Differential Diagnosis of Aortic Dissection

CARDIAC
• MI, ischemia
• Pericarditis
• Acute pulmonary edema
• Acute valvular dysfunction
PULMONARY
• Pulmonary embolus
• Pneumonia
• Pneumothorax
• Bronchitis, "pleurisy"
NEUROLOGIC
• CVA
• TIA
• Seizures
• Spinal cord compression
GASTROINTESTINAL
• Mesenteric ischemia
• Pancreatitis
• Esophageal spasm/rupture, esophagitis
• Peptic ulcer disease
• Biliary disease

Key:
MI = Myocardial infarction; CVA = Cardiovascular accident; TIA = Transient ischemic attack

Table 5. Diagnostic Testing for Aortic Dissection

VARIABLE	AORTOGRAPHY	CT	MRI	TEE
Sensitivity	++	+++	+++	+++
Specificity	+++	+++	+++	++/+++
Site of intimal tear	++	+	+++	++
Presence of thrombus	+++	++	+++	+
Presence of aortic insufficiency	+++	–	+	+++
Pericardial effusion	–	++	+++	+++
Branch vessel involvement	+++	+	++	+
Noninvasive	No	Yes	Yes	+/-
Rapid	No	Yes	+/-	Yes
No IV contrast	No	No	Yes	yes
Typically available to the ED	No	Yes	No	+/-

standing hypertension. Since 1% of dissections cause an MI, and aortic dissections involve the proximal coronary arteries in 10-20%, there may be acute changes of myocardial injury.

Basic laboratory testing is non-diagnostic. If aortic dissection is suspected, a rapid and sensitive test must be ordered. The most rapid and safe means for screening and detection of aortic dissection will be institution-dependent, but there are four imaging modalities useful in making the diagnosis of dissection: aortography, CT, magnetic resonance imaging (MRI), and transesophageal echo. Each is considered modality is considered below.

Angiography has been the traditional gold standard for diagnosis of aortic dissection, primarily because prior to the use of CT in the 1970s, it was the only test available. It identifies an intimal tear, aortic regurgitation, and branch vessel involvement from the false lumen. It also has been rendered largely obsolete for the detection and diagnosis of aortic dissection.[32]

Modern CT has the ability to image the entire aorta in a single volumetric breath hold scan. Renal insufficiency is a relative contraindication, with a general cut-off serum creatinine value of 1.8-2.0 mg/dL. It is widely available and non-invasive except for intravenous contrast injection.

MRI relies on the mobile hydrogen concentration of the blood to generate an image, and takes approximately 3-4 minutes to perform. Gadolinium-based contrast enhanced MR angiography can measure the cross-sectional size of the aorta and define the extent and origin of aortic dissection as well as branch vessel involvement.[33] Disadvantages to MR as a screening test include cost, longer examination times relative to CT, and lack of timely availability.

Transesophageal echocardiography (TEE) is the only imaging modality that can be brought to the patient's bedside. TEE provides a major role in the evaluation of aortic valvular disease. Disadvantages include the need to sedate the patient, especially if respiratory failure is present, risk of perforation of the piriform sinus, and the fact that the patient preferably should have nothing to eat or drink for four hours prior to the procedure. A routine TEE takes approximately 15 minutes, and up to 3% of patients do not tolerate the procedure. Like MRI, it may not be readily available to the emergency physician.

A 1996 study found that the sensitivity of MRI, CT, and TEE for diagnosis of aortic dissection all approached 100%, with specificities of 94%, 100%, and 94% respectively.[34] The relative diagnostic advantages of each diagnostic modality are summarized. (*See Table 5.*)

Treatment. Traditionally, Type A dissections have required emergency surgery, while distal dissections have the option of medical therapy. Type B dissections may cause limb ischemia, acute renal failure, or mesenteric ischemia. Type A (proximal dissections) may cause intrapericardial rupture and pericardial tamponade, aortic regurgitation and left ventricular failure, or myocardial infarction from coronary artery dissection. The complications of Type A dissections tend to be fatal more rapidly.

Stabilization in the ED starts with airway management and ventilation. If the patient is hypotensive, fluid resuscitation should be provided. Narcotic analgesics should be given for pain, and the blood pressure controlled with sodium nitroprusside, but only in conjunction with beta-adrenergic blockade. Beta blockade will reduce the shearing force on the aorta. Esmolol, propranolol, or labetalol are all acceptable agents.

Surgical techniques may include use of Dacron prostheses, human fibrin glue,[35] intimal tear resection with primary anastamosis of the aorta, total aortic replacement, or composite valve replacement or valve repair. In recent years, endovascular procedures with stenting of branch vessel occlusions and of the aortic true lumen has gained favor.[36-38] Operative mortality for Type A dissections historically has been in the 7-30% range, with overall five-year survival of 75-90% when all therapies are considered.

References

1. Richter JE. Noncardiac (Unexplained) chest pain. *Curr Treat Options Gastroenterol* 2000;3:329-334.
2. Achem SR, DeVault KR. Recent developments in chest pain of undetermined origin. *Curr Gastroenterol Rep* 2000;2:201-209.
3. Katz PO, Castell DO. Approach to the patient with unexplained chest pain. *Am J Gastroenterol* 2000:95:S4-S8.
4. Katz PO, Codario R, Castello O. Approach to the patient with unexplained Chest pain. *Compr Ther* 1997;23:249-253.
5. Mayou RA Bass CM Bryant BM. Management of noncardiac chest pain from research to clinical practice. *Heart* 1999;81:387-392.
6. Varia I, Logue E, O'Connor C, et al. Randomized trial of sertraline in patients with unexplained chest pain of noncardiac origin. *Am Heart J* 2000;140:367-372.
7. Potts SG, Bass CM. Psychological outcome and use of medical resources in patients with chest pain and normal or near normal coronary arteries: A long term follow up study. *QJ Med* 1993;86: 583-593.
8. Katz PO. Approach to the patient with unexplained chest pain. *Semin Gastrointest Dis* 2001;12:38-45.
9. Paterson WG. Canadian Association of Gastroenterology Practice Guidelines: Management of noncardiac chest pain. *Can J Gastroen-*

terol 1998;12:401-407.
10. Berry BE, Ochsner JL. Perforation of the esophagus: A thirty year review. *J Thoracic Cardiovasc Surg* 1973;65:1-7.
11. Jouriles NJ. Atypical ches pain. *Emerg Med Clin North Am* 1998; 16:717-740.
12. Castell DO, Katz PO. The acid suppression test for unexplained chest pain. *Gastroenterology* 1998;115:222-224.
13. Sarkar S, Aziz Q, Woolf CJ, et al. Contribution of central sensitisation to the development of non-cardiac chest pain. *Lancet* 2000;356: 1154-1159.
14. Beedassy A, Katz PO,Gruber A, et al. Prior sensitization of esophageal mucosa by acid reflux predisposes to reflux-induced chest pain. *J Clin Gastroenterol* 2000;31:121-124.
15. Tougas G, Spaziani R, Hollerbach S, et al. Cardiac autonomic function and oesophageal acid sensitivity in patients with non-cardiac chest pain. *Gut* 2001;49:706-712.
16. Hookman P, Barkin JS. The diagnosis and management of unexplained chest pain - is less more? *Am J Gastroenterol* 1999;94: 2310-2313.
17. Fass R, Fennerty MB, Ofman JJ, et al. The clinical and economic value of a short course of Omepramezole in patients with noncardiac chest pain. *Gastroenterology* 1998;115:42-49.
18. Pirtniecks A, Smith LF, Thorpe JA. Autonomic dysfunction in nonspecific disorders of oesophageal motility. *Eur J Cardiothorac Surg* 2000;17:101-105.
19. Anggianasah A, Marshall RE, Aggianasah CL, et al. Nonspecific oesophageal motility disorders and its relationship with gastroesophageal reflux. *Neurogastroenterol Motil* 1998;10:58.
20. Adamek RJ, Roth B, Zymanski CH, et al. Esophageal motility patterns in patients with and without coronary heart disease and healthy controls. *Hepatogastroenterology* 1999;46:1759-1764.
21. Borjesson M, Albertson P, Dellborg M, et al. Esophageal dysfunction in Syndrome X. *Am J Cardiol* 1998;82:1187-1191.
22. Chambers J, Bass C, Mayou R. Non-cardiac chest pain: Assessment and management. *Heart* 1999;82:656-657.
23. Pate JW, Richardson RL, Eastridge CE. Acute aortic dissections. *Am Surgeon* 1976;42:395-404.
24. Bickerstaff LK, Pairolero PC, Hollier LH, et al. Thoracic aortic aneurysms: A population-based study. *Surgery* 1982;92:1103-1108.
25. Gore I, Siewert VJ. Dissecting aneurysm of the aorta. Pathologic aspect and analysis of 85 fatal cases. *Arch Pathol* 1952;53:121.
26. Hirst AE Jr, Johns VJ Jr, Kime SW Jr. Dissecting aneurysm of the aorta. A review of 505 cases. *Medicine* 1958;37:217.
27. Ponraj P, Pepper J. Aortic dissection. *Br J Clin Pract* 1992;46: 127-131.
28. Laennec RTH. *De L'Auscultations Mediate, ou Traite de Diagnostic des Maladies des Poumons et du Couer Fonde Principaliment sur ce Nouveau Moyen d'Exploration*. Paris: Brossen & Chaude;1819:2.
29. Ring WS. Congenital Heart Surgery Nomenclature and Database Project: Aortic aneurysm, sinus of Valsalva aneurysm, and aortic dissection. *Ann Thorac Surg* 2000; 69:S147-163.
30. Torossov M, Singh A, Fein SA. Clinical presentation, diagnosis, and hospital outcome of patients with documented aortic dissection: The Albany Medical Center Experience, 1986 to1996. *Am Heart J* 1999;137:154-161.
31. Madu EC, Shala B, Baugh D. Crack-cocaine associated aortic dissection in early pregnancy- a case report. *Angiology* 1999;50: 163-168.
32. Urban BA, Bluemke DA, Johnson KM, Fishman EK. Imaging of thoracic aortic disease. *Cardiol Clin* 1999;17:659-682.
33. Prince MR, Narasimham DL, Jacoby WT, et al. Three-dimensional gadolinium-enhanced MR angiography of the thoracic aorta. *AJR Am J Roentgenol* 1996;166:1387-1397.
34. Sommer T, Fehske W, Holzknecht N, et al. Aortic dissection: A comparative study of diagnosis with spiral CT, multiplanar transesophageal echocardiography, and MR imaging. *Radiology* 1996; 199:347-352.
35. Neri E, Massetti M, Capannini G, et al. Glue containment and anastamosis reinforcement in repair of aortic dissection. *Ann Thorac Surg* 1999;67:1510-1511.
36. Dake MD, Kato N, Mitchell RS, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. *N Engl J Med* 1999;340:1546-1552.
37. Mitchell RS. Endovascular solutions for diseases of the thoracic aorta. *Cardiol Clin* 1999;17:815-825.

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

38. Marin ML, Lyon RT, Hollier LH, et al. Experience with endovascular grafts in the treatment of infrarenal aortic aneurysms associated with proximal aortic dissection. *Am J Surg* 1999;177:102-106.

Physician CME Questions

161. Which of the following statements is true regarding survival in esophageal perforation?
    A. It is causally related to lung hyperinflation.
    B. It is increased by the Omeprazole Challenge Test.
    C. It is inversely related to the time it takes to make the diagnosis and initiate treatment.
    D. It is decreased by the use of trazodone.

162. Which of the following statements is true of visceral hyperalgesia?
    A. It occurs when a previously innocuous stimulus becomes painful.
    B. It occurs when a painful stimulus becomes exaggerated.
    C. It is the endoscopic finding of patients with GERD.
    D. It is treated with nonsteroidal anti-inflammatory agents.

163. The Omeprazole Challenge Test is used diagnostically and therapeutically in which clinical condition?
    A. Gastroesophageal reflux disease
    B. Visceral hyperalgesia
    C. Pulmonary embolism
    D. Herpes Zoster

164. Which of the following is the most lethal esophageal cause of NCCP?
    A. GERD
    B. Esophageal perforation
    C. Nutcracker esophagus
    D. Diffuse esophageal spasm

165. The clinical presentation of GERD may be indistinguishable from acute coronary ischemia.
    A. True
    B. False

166. In the Stanford classification of aortic dissection, which of the following is true?
    A. Type A involves the ascending aorta.
    B. Type C involves the ascending and descending aorta.
    C. Ambulatory pH monitoring is required to classify the patient as Type I or II.
    D. There is a 90% mortality rate within the first 24 hours.

167. Which of the following conditions has evidence of inflammatory cell infiltration, fibrosis, and damage to the intrinsic nervous system of the esophagus?
    A. Nutcracker esophagus
    B. Nonspecific esophageal motility disorder
    C. Hypertensive LES
    D. Achalasia

168. Depending on which branch vessels are affected, aortic dissection may present with which of the following?
    A. Myocardial infarction
    B. Stroke
    C. Intestinal or spinal cord ischemia
    D. Renal insufficiency
    E. All of the above

169. Which of the following statements is true regarding aortic dissection?
    A. It shows a female predominance of 3:1.
    B. Patients with Type B dissection tend to be younger and have a higher incidence of connective tissue disorders.
    C. Patients with Type A tend to be older and have more factors associated with circulatory ailments.
    D. A chief risk factor for the condition is hypertension.

170. The following four imaging techniques are useful in making the diagnosis of aortic dissection: aortography, CT, MRI, and TEE.
    A. True
    B. False

In Future Issues:

Noncardiac Chest Pain, Part II

Emergency Medicine Reports CME Objectives

To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur.

Answer Key

161. C	166. A
162. B	167. D
163. A	168. E
164. B	169. D
165. A	170. A

Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Noncardiac Chest Pain, Part I

Epidemiology of Noncardiac Chest Pain

Prevalence	12% of general population
Negative cardiac catheterizations	20-30% of all cardiac catheterizations
Negative cardiac catheterizations mortality	< 1% over 7 years
Annual NCCP patient care expense	$ 1.8 billion (1989)
Disability of NCCP patients	50% not working
Heart awareness programs	Increased ED evaluations for chest pain
Liability of missed myocardial infarction	Highest % of ED malpractice dollars paid

Modern Classification of Esophageal Motility Disorders

1. ACHALASIA

a. Absence of peristalsis in esophageal body
b. Incomplete lower esophageal sphincter (LES) relaxation
c. Elevated LES pressure (normal levels depend on individual labs)
d. Increased intraesophageal baseline pressure relative to gastric baseline

2. DIFFUSE ESOPHAGEAL SPASM (DES)

a. Simultaneous (non-peristaltic) contractions occurring after > 10% of wet swallows
b. Periods of normal peristalsis
c. May see spontaneous and/or repetitive contractions
d. Contractions may be of increased amplitude or duration
e. LES pressure may be elevated (one-third of patients)

3. NUTCRACKER ESOPHAGUS

a. Mean peristaltic amplitude in distal esophagus ≥ 180 mmHg
b. Normal peristaltic sequence preserved
c. May have prolonged duration (≥ 6.0 seconds)

4. HYPERTENSIVE LES

a. LES pressure ≥ 2 standard deviations above normal mean with normal relaxation
b. Normal peristalsis

5. NON-SPECIFIC ESOPHAGEAL MOTILITY DISORDER

a. Reduced amplitude of esophageal peristalsis
b. Triple peaked contraction waves
c. Simultaneous contractions (≤ 10% of wet swallows)
d. Spontaneous contractions
e. Isolated prolonged duration contractions

Adapted from: Pirtniecks A, Smith LF, Thorpe JA. Autonomic dysfunction in non-specific disorders of eosophageal motility. *Eur J Cardiothoracic Surg* 2000;17:101-105.

Treatment of GI Causes of Chest Pain

GERD

- Omeprazole, 20 mg bid (4-8 wks)
- Omeprazole (high dose), 40 mg in a.m., 20 mg in p.m.

ESOPHAGEAL DYSMOTILITY DISORDERS

- Nitrates: Nitroglycerine, 0.4 mg SL prn pain *OR* Isosorbide dinitrate, 10-30 mg bid
- Calcium channel blockers: Nifidipine, 10-30 mg *OR* Diltiazem, 90 mg, both qid immediately before meals or as needed for pain. Watch for hypotension.

VISCERAL HYPERALGESIA

- Imipramine, 50 mg at bedtime
- Trazodone, 100-150 mg daily

BOERHAAVE'S SYNDROME

- Prompt diagnosis
- IV fluids
- Antibiotics
- Prompt surgical referral

Differential Diagnosis of Aortic Dissection

CARDIAC

- MI, ischemia
- Pericarditis
- Acute pulmonary edema
- Acute valvular dysfunction

PULMONARY

- Pulmonary embolus
- Pneumonia
- Pneumothorax
- Bronchitis, "pleurisy"

NEUROLOGIC

- CVA
- TIA
- Seizures
- Spinal cord compression

GASTROINTESTINAL

- Mesenteric ischemia
- Pancreatitis
- Esophageal spasm/rupture, esophagitis
- Peptic ulcer disease
- Biliary disease

Key:
MI = Myocardial infarction; CVA = Cardiovascular accident; TIA = Transient ischemic attack

Diagnostic Testing for Aortic Dissection

VARIABLE	AORTOGRAPHY	CT	MRI	TEE
Sensitivity	++	+++	+++	+++
Specificity	+++	+++	+++	++/+++
Site of intimal tear	++	+	+++	++
Presence of thrombus	+++	++	+++	+
Presence of aortic insufficiency	+++	–	+	+++
Pericardial effusion	–	++	+++	+++
Branch vessel involvement	+++	+	++	+
Noninvasive	No	Yes	Yes	+/-
Rapid	No	Yes	+/-	Yes
No IV contrast	No	No	Yes	yes
Typically available to the ED	No	Yes	No	+/-

Supplement to *Emergency Medicine Reports*, August 11, 2003: "Noncardiac Causes of Chest Pain in the Emergency Department: Part I."
Authors: **Eric Anderson, MD, MBA, FACEP,** Associate Director of Clinical Operations, Cleveland Clinic, OH; Assistant Professor of Emergency Medicine, The Ohio State University, Columbus; **Jonathan Glauser, MD, MBA, FACEP,** Cleveland Clinic Foundation; Assistant Clinical Professor of Medicine, Case Western Reserve University; Faculty, Residency Training Program in Emergency Medicine, MetroHealth Medical Center, Cleveland, OH.