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Acute CHF Exacerbations

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Objectives

After reading this issue, you should be able to:

- Identify the common causes of an acute exacerbation of congestive heart failure in patients who present to the emergency department.
- Explain the difficulty in making the diagnosis with certainty.
- Describe the rational use of diagnostic and treatment modalities for congestive heart failure in the emergency department and how to use them more effectively.
- Discuss a comprehensive differential diagnosis for etiologies of congestive heart failure exacerbations.

Introduction

Congestive heart failure, or CHF, continues to strain our already overburdened health care system, accounting for approximately 900,000 hospital admissions a year[1] and costing in excess of $7 billion dollars.[2] It most frequently affects the elderly-75% of patients afflicted with the disease are over the age of 65, and our population is aging rapidly.[3] More than 5 million patients in this country currently have CHF, and the problem will continue to grow. Emergency physicians should be able not only to make the diagnosis, but also to identify and treat the underlying cause of a patient's acute decompensation. Because the condition can deteriorate suddenly, emergency physicians must be prepared to treat heart failure appropriately and aggressively.

The challenge lies in differentiating between CHF and other causes of acute dyspnea. With the addition of B-type natriuretic peptide, or BNP, to many emergency department laboratory panels, physicians possess a tool that can potentially help them make this important distinction. However, a patient's BNP level has inherent limitations, and emergency physicians must fully understand these limitations in order to use this test effectively. A comprehensive understanding of the diagnostic limitations of chest x-ray, laboratory studies, and physical examination findings, combined with a thorough history, will help physicians make this diagnosis.

Once the diagnosis of CHF has been established, the cause of both the underlying pathology and the acute exacerbation must be sought. Ischemic cardiomyopathy due to coronary artery disease is the most common underlying pathology, but other pathologies-nonischemic cardiomyopathies (hypertensive, alcoholic, idiopathic), valve abnormalities (aortic stenosis, other obstructive causes), even separate entities such as volume overload in renal failure patients-can also contribute. Another factor is diastolic dysfunction, which accounts for up to 20% to 40% of patients with congestive heart failure.[4] As with many conditions, understanding the pathology of a CHF episode is important because it can dramatically change its management.

Yet, the etiology for heart failure is not always determined in the emergency department. The emergency physician must rapidly and aggressively begin to treat the patient's symptoms on arrival. By adequately dosing and quickly titrating appropriate medications, the physician will prevent unnecessary morbidity and mortality. If an underlying cause can be determined, then treatment can be tailored accordingly.

On Sight - 1

Bob Park, a 70-year-old man with a history of CHF and COPD, arrives at Farmington Medical Center on a very busy Monday morning. He had called 911 shortly after waking when he began to experience worsening shortness of breath. Mr. Park told the paramedics that he started having trouble breathing 3 days earlier after he ran out of his medications. He had put off going to the pharmacy because he was "in a lot of pain and not able to get around" after twisting his ankle. The paramedics give their report to the nurse, telling her that the patient has a history of CHF and COPD and is noncompliant on his medications. Mr. Park's past medical history is significant for hypertension, diabetes, and osteoarthritis. He takes aspirin, furosemide, atenolol, glucophage, glyburide, benazepril, digoxin, flovent, nitroglycerin, and albuterol prn. He has no known drug allergies. He underwent surgical repair of an inguinal hernia 10 years earlier, and CABG 10 months earlier after suffering a myocardial infarction.

Robert Boyd, MD, the emergency physician on duty, notes that Mr. Park is a tachypneic, diaphoretic elderly man who is barely able to answer questions without gasping for air. He is pale, slightly cyanotic, and aggressively using his accessory muscles to breathe. He is unable to give a detailed history but does admit to not taking his medications for the past 3 days. Triage vital signs are noted as blood pressure 150/100, pulse rate 110, respiratory rate 40, temperature 99.9°F, and oxygen saturation 83% on room air; his SaO_2 has improved to 89% with the administration of oxygen. Physical examination reveals elevated jugular venous pulsations at approximately 9 cm H_2O, a hepatojugular reflex, and bilateral pitting edema that is worse on the right. Examination of the chest reveals distant breath sounds with slight crackles at the bases-not what Dr. Boyd expects to hear with an acute CHF exacerbation. Cardiac sounds include a regular tachycardia with distant S_1 and S_2 but no S_3, S_4, or murmurs.

After examining Mr. Park, Dr. Boyd orders a CBC, electrolytes, BUN, creatinine, glucose, troponin, CK, chest x-ray, and an ECG. He also decides to order a BNP level to try to differentiate between CHF and COPD as the cause of Mr. Park's dyspnea. The initial ECG exhibits a sinus tachycardia at 110 bpm with a right bundle branch block; there is no previous ECG available for comparison. Chest x-ray reveals cardiomegaly with mild right pleural effusion and some atelectasis. While waiting for the laboratory results, Dr. Boyd decides to give Mr. Park an aspirin and try gentle diuresis, leaning toward the diagnosis of a CHF exacerbation. The aspirin is administered, in addition to furosemide 40 mg IV, nitropaste 1 inch to the chest wall, and continued oxygen by nonrebreather mask. Mr. Park begins to tire. Dr. Boyd, still unclear on the diagnosis, wonders whether he should intubate or start more aggressive treatment.

Sixty minutes later, the lab reports a BNP level of 240 pg/mL, which Dr. Boyd interprets as positive for a CHF exacerbation. He starts a nitroglycerin drip and orders more furosemide. Mr. Park's condition improves only minimally; he continues to struggle to breathe, and his mental status declines. Dr. Boyd intubates and sends Mr. Park to the ICU.

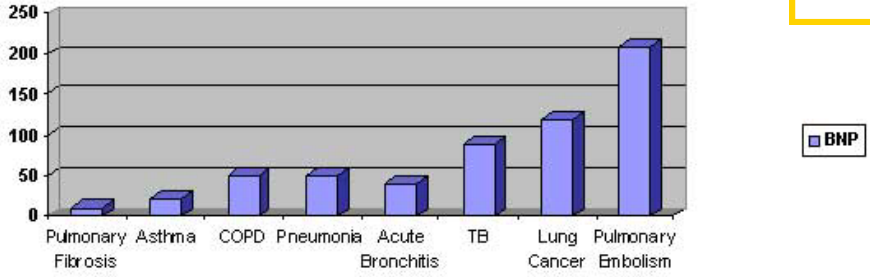
While in the ICU, Mr. Park becomes hypotensive. Echocardiography reveals failure of the inferior vena cava to collapse during inspiration as well as right ventricular dilation and hypokinesis-the diagnostic criteria for pulmonary embolism. Heparin and thrombolytic agents are started immediately. Mr. Park's condition stabilizes, but he has a prolonged hospital course. He eventually sues the hospital and Dr. Boyd for misdiagnosis and for delaying appropriate care.

Insight - 1

Differentiating between an exacerbation of CHF and COPD is difficult in the emergency department, without additionally having to consider other diagnoses. However, emergency physicians must be astute in assessing patients with acute dyspnea and aware of the limitations of using BNP as a diagnostic tool.

B-type natriuretic peptide is released from the ventricular myocardium in response to elevations of end-diastolic pressure and volume. It will be elevated during CHF exacerbations as a result of stress on the left ventricle. However, BNP levels also will be elevated in pulmonary embolism, cor pulmonale, and lung cancer-other conditions that increase the end-diastolic pressure of either the left or right ventricle.[5] These elevations are generally not as high as elevations caused by CHF, but the "cut-off" value that would distinguish CHF from these other conditions has yet to be determined (Figure 1).[5] In fact, BNP levels in the setting of large pulmonary embolisms have been reported from 200 to 300 pg/mL.[5]

Figure 1. Mean BNP levels seen in patients with various types of lung disease.*



*From Morrison LK, Harrison A, Krishnaswamy P, et al. Utility of a rapid B-natriuretic peptide assay in differentiating congestive heart failure from lung disease in patients presenting with dyspnea. *J Am Coll Cardiol.* 2002;39(2):202-209.

A recent prospective study that included more than 1,500 patients presenting with acute dyspnea showed that BNP had a negative predictive value of 96% with a cut-off of 50 pg/mL, but a positive predictive value of only 83.4% with a level of 150 pg/mL or greater.[6] Measurement of BNP level appears to be more helpful if used as a tool for excluding the diagnosis rather than for making it. Also, BNP has been found to correlate with a patient's New York Heart Association (NYHA) classification (Table 1).[6]

Table 1. Median BNP levels among patients in each of the four heart failure classes.*

Class	Patient Symptoms	BNP Level (pg/ml)
Class I (Mild)	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnea.	244 (+/- 286)
Class II (Mild)	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnea.	389 (+/- 374)
Class III (Moderate)	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitation, or dyspnea.	640 (+/- 447)
Class IV (Severe)	Unable to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency at rest. If any physical activity is undertaken, discomfort is increased.	817 (+/- 435)

*From data in Maisel AS, Krishnaswamy P, Nowak RM, et al. Rapid measurement of B-type natriuretic peptide in the emergency diagnosis of heart failure. *N Engl J Med.* 2002;347(3):161-167.

Overall, BNP levels should be interpreted with caution in any patient who has a history of underlying CHF.

Emergency physicians should understand not only the limitations of BNP, but also its utility in helping to differentiate cardiac from pulmonary causes of acute dyspnea. At the extreme ends of the spectrum, BNP can help make a difficult diagnosis. However, in the intermediate range, it should be interpreted with great caution while aggressively searching for other causes. Given that other modalities, such as

echocardiography and Swan-Ganz catheterization, are difficult to obtain in the emergency department, it is easy to see why some view BNP as a useful adjunct to excluding the diagnosis of CHF. It is not, however, an absolute indicator for making or excluding the diagnosis.

Hindsight - 1

Mr. Park presented with physical findings that indicated right heart failure, but not necessarily left heart failure. The presence of elevated jugular venous pressure, lower extremity edema, and hepatojugular reflex with only mild crackles and infiltrates on chest x-ray should have been the clue Robert Boyd needed to search for a cause of right heart failure. It could have been the result of a COPD exacerbation, but Mr. Park's history of immobility with asymmetrical leg swelling made consideration of pulmonary embolism reasonable. The persistent sinus tachycardia and right bundle branch block on ECG, although not specific, also suggested right heart strain. Robert Boyd instead considered a mildly elevated BNP level to be evidence of CHF, despite signs and symptoms that suggested otherwise, and despite the lack of an accepted cut-off value in the literature.
The plaintiff's attorney claimed that Robert Boyd's misdiagnosis delayed appropriate treatment and led to Mr. Park's prolonged and complicated hospital course and much suffering. The plaintiff's expert witness argued that Dr. Boyd should have realized the limitations of an intermediate BNP level and searched for other causes of the patient's acute dyspnea, especially in light of Mr. Park's history of immobility and the physical finding of asymmetric leg swelling. The expert witness expressed surprise that Dr. Boyd didn't consider pulmonary embolism or pursue the diagnosis with a CT pulmonary angiogram or a D-dimer. The defense team for the hospital and Dr. Boyd decided that its case was weak and recommended an out-of-court settlement.

- Interpret BNP levels with caution, especially those in the intermediate range. Pulmonary embolism, pulmonary hypertension, right heart failure, lung cancer, and other pulmonary diseases can cause mild elevations of BNP.
- Search for other causes when test results don't correlate with physical examination findings.
- BNP can be a helpful tool for differentiating CHF from other pathologies when it's considered within the context of the history, physical examination findings, and other test results.

On Sight - 2

On the Sunday night after the Super Bowl, 65-year-old Tony Vincent comes to the emergency department of Donovan Hospital complaining of shortness of breath. His wife tells the triage nurse that he has a history of CHF and coronary artery disease. She has his medications in her purse-aspirin, metoprolol, ramipril, and spironolactone-and tells the nurse that he is allergic to penicillin. He underwent a CABG 7 years earlier and "has these episodes every now and then, but this one is a lot worse." Mrs. Vincent is more than a little irritated with her husband because he spent the evening celebrating the Eagles' victory with his friends and "eating popcorn and chips and all that other stuff he knows he's not supposed to have." Within minutes of arrival, Mr. Vincent becomes more cyanotic. His oxygen saturation is 85% on room air, and his breathing difficulty worsens.
The triage nurse calls James Staley, MD, the emergency physician on duty, and asks him to see Mr. Vincent immediately. Dr. Staley has Mr. Vincent moved to the resuscitation area and instructs the nurse to administer oxygen by a nonrebreather mask. He notes elevated jugular venous pulsations to the angle of the mandible and hears loud diffuse crackles on examination of the chest. Cardiac auscultation reveals S_3 and sinus tachycardia. Dr. Staley also makes

note of lower extremity edema and the patient's obesity. His SaO_2 has risen to 91%. Dr. Staley decides that Mr. Vincent is experiencing an acute CHF exacerbation as a result of a high-salt diet.
Dr. Staley immediately starts a nitroglycerin drip at 10 µg/min and orders aspirin 325 mg, furosemide 40 mg IV, and placement of a Foley catheter to monitor urine output. When Mr. Vincent's breathing doesn't improve, the nurse asks Dr. Staley if the nitroglycerin drip can be increased. Initially Dr. Staley says no, that they shouldn't increase it by more than 5 to 20 µg every 10 minutes, but as Mr. Vincent continues to decompensate, Dr. Staley decides to increase the nitroglycerin drip to 30 µg/min. Oxygen saturation remains around 90% on the nonrebreather mask, but the patient is becoming tired. Minutes later his mental status begins to wane and his SaO_2 drops to 87%. Dr. Staley attempts intubation but is deterred by the patient's short, obese neck and an inability to visualize the cords. Bag-mask ventilation also is difficult. Mr. Vincent's SaO_2 level drops into the 70s, and Dr. Staley decides that cricothyrotomy is necessary. Although the procedure is made difficult by obscure landmarks, Dr. Staley successfully places a tracheostomy tube in the trachea through the cricothyroid membrane. The patient is finally oxygenated and placed on a ventilator but is only minimally responsive to painful stimuli and requires little sedation.
Dr. Staley admits Mr. Vincent to the ICU with the diagnosis of acute CHF exacerbation. He is only minimally responsive for several days, even after his hemodynamic status improves, and his wife is worried that he'll never wake up. Eventually Mr. Vincent begins to recover. He is taken off the ventilator on day 12 of his hospital stay and slowly makes a full recovery. He promises his wife that he'll stay away from salty foods.
Although Mrs. Vincent is approached by several attorneys who believe she has a "good case" against Donovan Hospital and Dr. Staley, she decides not to sue. Instead, she blames her husband for his bad habits and says that Dr. Staley "was just doing his job."

Insight - 2

Standard treatment for an acute CHF exacerbation includes oxygen, diuresis, and vasodilatation to decrease preload, as well as morphine for pain and anxiety and, to a lesser extent, vasodilatation. A patient who presents with severe hypoxia should be placed on a nonrebreather mask. Diuresis usually is accomplished with intravenous furosemide. Nitrates are the standard for vasodilatation and can be administered sublingually, transdermally, or intravenously.
The question arises as to which is more effective, diuresis or vasodilatation, although both are used concurrently. A recent study of 110 patients attempted to answer this question.[7] Patients presenting with signs of CHF were treated with oxygen 10 L/min, furosemide 40 mg IV, and morphine 3 mg bolus. Patients were then randomly assigned to two groups: group A received isosorbide dinitrate (3 mg bolus IV q5min), and group B received furosemide (80 mg bolus IV q15min) plus isosorbide dinitrate (16.7 µg/min increased q10min by 16.7 µg/min). Mechanical ventilation was required in 13% of patients in group A, and 40% of patients in group B ($P = 0.0041$). Myocardial infarction occurred in 17% and 37% of patients, respectively ($P = 0.047$). One patient in group A and three in group B died ($P = 0.61$). This study indicates that high-dose isosorbide dinitrate, given as repeated intravenous boluses after low-dose intravenous furosemide, is safe and effective in controlling severe pulmonary edema. This treatment regimen is more effective than high-dose furosemide with low-dose isosorbide nitrate in terms of need for mechanical ventilation and frequency of myocardial infarction.
Some literature supports the use of noninvasive ventilation in patients with CHF.[8,9] Noninvasive ventilation includes CPAP (continuous positive airway pressure) and BiPAP (bilevel positive airway pressure). CPAP raises intrathoracic pressure, decreasing arteriovenous shunting and therefore improving oxygenation.[10] It also decreases the work of breathing, allowing the patient to fatigue less rapidly. By decreasing left ventricular afterload, CPAP also improves cardiac output.[11] Three randomized studies have compared the effects of CPAP with standard therapy on patients hospitalized for acute cardiogenic pulmonary edema (ACPE).[10,12,13] One of these revealed improved respiratory and cardiovascular dynamics in ICU patients treated with CPAP.10 A meta-analysis of all three studies revealed a decreased need for intubation (41% to 16%) and a trend toward improved mortality.[14]

The evidence to support the use of BiPAP for ACPE is less impressive. A meta-analysis looking at BiPAP added to standard care in patients with respiratory failure revealed that there is a decreased need for intubation and improved survival only in those patients with COPD.[15] There are no randomized trials comparing BiPAP to standard medical therapy for ACPE. A randomized, controlled trial comparing the use of CPAP with BiPAP in 27 ACPE patients was stopped prematurely because of an extremely high rate of acute myocardial infarction in the BiPAP group (71% vs 31% vs 38% in historical controls). BiPAP did result in improved pulmonary parameters.[16] One study prospectively evaluated prehospital BiPAP in 62 ACPE patients and found no difference in treatment times, death, hospitalization length, or intubation.[17] Finally, a prehospital randomized trial of high-dose intravenous isosorbide dinitrate versus BiPAP demonstrated that the nitrate arm resulted in lower mortality, lower mechanical ventilation rate, and rate of acute myocardial infarction.[18]
A drawback of CPAP is that patients must be alert enough to cooperate with the device and follow instructions. Once a patient's mental status begins to wane, CPAP is usually ineffective and intubation is indicated. Therefore, CPAP should be attempted as early as possible in the management of acute CHF exacerbations. In summary, current evidence suggests there is no proven benefit to the use of BiPAP, but there might be some benefit to the use of CPAP early in the treatment of patients with ACPE.
Many patients with acute pulmonary edema caused by an acute CHF exacerbation receive one or several sprays of nitroglycerin in the field before they arrive at the hospital. The sublingual spray is 400 µg per bolus given every 5 minutes, which is equivalent to an 80 µg/min drip. Emergency physicians should consider starting a nitroglycerin drip at 50 to 100 µg/min as long as the patient's blood pressure is stable. They may also continue to give sublingual nitroglycerin while the drip is being prepared. Drips can then be aggressively titrated upward by at least 30 to 50 µg/min every 5 to 10 minutes until the patient's symptoms improve. In the study by Cotter,[7] patients in the high-dose isosorbide group received 3 mg boluses of nitroglycerin every 5 minutes, which is equivalent to a 600 µg/min nitroglycerin drip. The patients not only tolerated this regimen, but also required fewer intubations than those on a lower-dosed drip.
Together, positive pressure ventilation and aggressive treatment with nitrates might prevent unnecessary intubations in patients who suffer from a quickly reversible disease process

Hindsight - 2

James Staley and his department director were relieved that Mr. Vincent recovered, and equally relieved that his family didn't pursue legal action. In their review of the case, they agreed that, although Dr. Staley made the correct diagnosis, he might not have been aggressive enough in his treatment. Specifically, they agreed that if he had attempted CPAP initially when Mr. Vincent was awake and cooperative, he might have been able to avoid performing the cricothyrotomy.
After the hospital's QA committee reviewed the case, the department instituted CHF treatment guidelines to encourage the early use of CPAP and aggressive use of nitrates. The new guideline calls for initial dosing with three sublingual nitroglycerin sprays followed by a 50 to 100 µg/min drip titrated rapidly by 30 to 50 µg/min until the patient's condition improves. Inservice training was provided to ensure that the emergency department staff and respiratory therapists were educated on the use of CPAP and aggressive use of nitrates.

- Use nitrates aggressively in acute CHF exacerbations, starting drips at 50 to 100 µg/min and titrating quickly upward.
- Consider using CPAP early in the treatment of CHF to avoid intubation.
- Acute exacerbations of CHF are often quickly reversible if treated appropriately

On Sight - 3

Ray Smith brings his wife, Valerie, to the Sylvan Hospital emergency department. She is in obvious respiratory distress. Mr. Smith tells the triage nurse that his wife had a tooth pulled a few days earlier and "has been having trouble breathing ever since." He is especially concerned because she had a heart valve replaced 4 years earlier to treat mitral regurgitation, and her recovery was complicated by an episode of CHF. He couldn't remember the name of her cardiologist and decided to "just come on to the ER." As Mrs. Smith is taken to a treatment room, her distraught husband confides to the triage nurse, "She's been sort of depressed lately, and I'm pretty sure she's not taking any of her medicines." The triage nurse then adds this information to her notes.

When Walter Thompson, MD, comes into the treatment room, he notes that the patient is tachypneic and in obvious respiratory distress. Vital signs are blood pressure 100/70, pulse rate 120, respiratory rate 35, temperature 98ºF, and oxygen saturation 90% on room air. Following administration of oxygen, the oxygen saturation level rises to 95%. Dr. Thompson proceeds with the physical examination, noting elevated jugular venous pulsations at the angle of the mandible, bilateral crackles filling both lung fields, and a holosystolic murmur that radiates to the axilla. He is unable to hear the click of the prosthetic mitral valve but is unsure because the heart sounds are very distant. He also notes the presence of bilateral lower extremity edema.

Dr. Thompson speaks with Mrs. Smith briefly to follow up on the triage nurse's notes. Mrs. Smith admits that she's been suffering from depression lately but assures him that she's been taking her medications with the exception of the warfarin, which she stopped taking 3 days before having her tooth pulled according to the dentist's instructions. Unsure as to whether to believe Mrs. Smith or her husband, Dr. Thompson considers what might have triggered this acute episode of CHF, then decides to begin treatment while waiting for test results. He orders aspirin, furosemide 40 mg IV, and a nitroglycerin drip at 50 µg/min. Mrs. Smith's condition improves minimally. During the next 90 minutes that she waits in the emergency department for admission, her oxygen saturation level remains in the low 90s on a nonrebreather mask. Laboratory test results include a normal CBC count, creatinine slightly increased at 1.6, HCO_3^- of 16, and INR 1.8. Dr. Thompson realizes that the INR is slightly less than the desired 2.5 to 3.5 target for mitral valves recommended by the ACC/AHA guidelines, but he attributes it to the temporary lack of warfarin. He orders an oral dose of warfarin 5 mg and admits Mrs. Smith to a telemetry bed for diuresis and management of her CHF.

Shortly after being moved upstairs, Mrs. Smith becomes hypotensive and hypoxic. The cardiologist on call performs echocardiography and discovers that her mechanical valve has thrombosed and is nonfunctional. She now has wide-open mitral regurgitation. Despite administration of thrombolytics, Mrs. Smith becomes severely hypotensive, goes into cardiac arrest, and dies.

Mrs. Smith's family eventually sues Dr. Thompson for failure to recognize the cause of her CHF exacerbation, and the cardiologist for delay in treatment.

Insight - 3

Discovering the etiology of an acute CHF exacerbation can be difficult, if not impossible, in the emergency department. However, the emergency physician should still search diligently for the factors causing such acute decompensation. When assessing a patient who has a prosthetic valve and is in acute respiratory distress, the emergency physician should presume that the valve has thrombosed or is malfunctioning until proved otherwise. This difficult diagnosis is even harder to make in the emergency setting given the limited availability of echocardiography, especially in the middle of the night. However, physical examination findings of a prolonged, holosystolic, "pathologic" murmur and no discernible "click" from a properly functioning mechanical valve should alert the emergency physician to the correct diagnosis. The physician should still pursue echocardiography, as it remains the gold standard for determining valve patency.

In a recent case series, patients with thrombosed valves presented with either acute pulmonary edema or cardiogenic shock.[19] Both of these conditions will result in presentation to the emergency department, where it will be imperative for the emergency physician to make the correct diagnosis. The interval between implantation and dysfunction in these cases ranged from 1 month to 5 years. Therefore, time from implantation is not helpful in determining valve patency. However, systemic anticoagulation was inadequate in 80% of these patients (prothrombin time <2 times control). Valve thrombosis must be a concern in all patients who are not adequately anticoagulated.

Thrombosed valves are treated with either thrombolytic therapy or surgery for valve replacement or thrombectomy. Although several case reports have demonstrated success with thrombolytic therapy,[20-22] early surgical intervention has been shown to reduce both morbidity and mortality.[23] Once a thrombosed valve is suspected, both cardiology and cardiothoracic surgery should be contacted immediately.

Valve thrombosis in a patient with a prosthetic valve is an unusual cause of acute CHF, but the emergency physician must keep a wide differential diagnosis in mind when considering etiologies of CHF (Figure 2).

Figure 2. Causes of acute heart failure.

Myocardial infarction	Pericardial tamponade
Cardiomyopathy	Constrictive pericarditis
Hypertension	Myocarditis
Valve abnormalities, prosthetic or inherent	Toxins (alcohol, drugs, digitalis, beta-blockers)
Arrhythmias	Shunts
Hypoxemia	Anemia
Pulmonary embolism	Hyperkalemia
Endocarditis	Hyperthyroidism
Pregnancy	Congenital heart problems
Medication noncompliance	Dietary indiscretion

Hindsight - 3

In the case against Walter Thompson, the plaintiff claimed that the failure to diagnose Mrs. Smith's thrombosed valve led to her death. The expert witness argued that Dr. Thompson should have presumed any patient with CHF and a prosthetic valve has an acute thrombus until that diagnosis is excluded. The expert contended that Dr. Thompson should have immediately called both cardiology and cardiothoracic surgery for help in both diagnosis and treatment. The plaintiff's witness also argued that the cardiologist should be held responsible for not calling cardiothoracic surgery once the diagnosis was made, contending that he should have been aware that the outcomes are thought to improve with surgical replacement or thrombectomy rather than thrombolytic therapy. The cardiologist argued that he didn't have time to call in a cardiothoracic surgeon, and that he provided the best treatment possible in the setting of such rapid decompensation. Despite the plaintiff's expert witness's claim that he should have performed echocardiography immediately after Mrs. Smith was moved to a monitored bed, the cardiologist was not faulted by the court for the care he provided. Dr. Thompson, however, did not fare as well in the eyes of the court. Mrs. Smith's husband was awarded a large sum of money for the pain and suffering he incurred.

- Assume that a patient with CHF and a prosthetic valve has valve thrombosis until proved otherwise.
- Cardiology and cardiothoracic surgery should both be involved in the management of valve malfunctions.
- A patient with a subtherapeutic INR (<2) is at high risk for valve thrombus.

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