

## How Do You Mend a 'Broken' Heart?

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### Course Objectives

The purpose of this program is to inform nurses about the features of and treatments and nursing interventions for stress cardiomyopathy ("broken heart syndrome"). After studying the information presented here, you will be able to —

- Identify signs and symptoms of stress cardiomyopathy.
  - Distinguish stress cardiomyopathy from an acute myocardial infarction (MI).
  - Describe three nursing implications in the treatment of people with stress cardiomyopathy.
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*Joyce,\* a fourth-grade teacher, is an active, healthy person, but extremely shy. On her 60th birthday, her husband took her out to dinner to celebrate. Arriving at the restaurant, Joyce was overwhelmed by the 25 friends and family members gathered for a surprise birthday party. Soon after, she began to experience shortness of breath, weakness, substernal chest pain, and feelings of impending doom. Joyce had no previous cardiac history. When the paramedics arrived, she was pale and clearly distressed.*

*When Joyce arrived at the ED, she still had chest pain and shortness of breath; her vital signs revealed a blood pressure of 80/50, heart rate of 130, and respiratory rate of 24. Twelve-lead ECG showed T-wave inversion and a prolonged QT interval. Serum [troponin I levels](#), biochemical indicators of cardiac disease, were slightly elevated. (Troponin levels are significantly elevated when there is myocardial damage, such as with an acute MI.) Joyce was admitted to the coronary care unit for a cardiac evaluation.*

*On the unit, she received oxygen, bed rest, telemetry, a vasopressor for her BP, and treatment as an acute coronary syndrome patient. [Coronary angiography](#) revealed an ejection fraction of 20% (the percent of blood ejected with each heartbeat; normal is 65%) and apical hypokinesia (weakened contractions). Her initial symptoms resolved within three hours of admission. Joyce went home five days later with a diagnosis of stress cardiomyopathy, sometimes called "broken heart syndrome," and recovered completely within two weeks.*

Nurses need to learn about the recently defined syndrome of stress cardiomyopathy to meet the needs of patients like Joyce. When triaging people with cardiac symptoms, nurses should be aware of stress cardiomyopathy as a potential diagnosis and recognize the profile of those at risk. Patients will need a great deal of reassurance regarding their cardiac function and the cause of their symptoms, and a knowledgeable nurse can provide valuable education.

### How the heart 'breaks'

Stress cardiomyopathy is a rapidly reversible, left ventricular dysfunction precipitated by sudden emotional stress, such as that caused by the death of a loved one or other trauma. It's most commonly seen in postmenopausal women.<sup>1</sup> Unlike an MI stemming from blockage of one or more coronary arteries, stress cardiomyopathy is thought to be caused by a surge of [catecholamines](#) (e.g., epinephrine) that causes myocardial stunning — the myocardium cells' temporary loss of the ability to contract effectively — as a result of the acute stress. Myocardial stunning is a reversible process, while coronary artery disease and MI result in permanent damage.<sup>2,3</sup>

When correctly diagnosed, the patient with stress cardiomyopathy will not need the invasive testing so commonly used with irreversible myocardial damage. However, stress cardiomyopathy is such a newly recognized phenomenon that more invasive testing, such as cardiac angiography, is likely to be ordered to rule out more serious and irreversible cardiac disease.

While the cause of stress cardiomyopathy is understood to be a surge of catecholamines, the mechanism of action for this syndrome remains unknown. Several theories have been suggested to explain the pathophysiology. One theory points to an epicardial coronary artery spasm from increased sympathetic activity, which results in vasoconstriction of the coronary arteries and a temporary decrease in blood flow. An alternative mechanism to explain the disorder is sympathetic stimulation of the microcirculation that leads to microvascular spasm that decreases blood flow. A third explanation involves the direct cardiotoxic effects (due to increased calcium) of excessive catecholamine release to the heart muscle.<sup>2</sup>

### Fact or fable?

Tales of people being literally "scared to death" or "dying of a broken heart" are common throughout history.<sup>3</sup> But efforts to record this phenomenon medically have been sporadic until fairly recently.

In 1943, a U.S. scientist proposed a cause-and-effect relationship between spasm of the coronary arteries and intense emotion and speculated

that such spasms could cause tissue damage from ischemia or that a clot could result if the spasm was prolonged.<sup>4</sup> In the 1950s and 1960s, a nine-year study of more than 4,000 widowers in Britain found that mortality rate among the men was 40% higher in the first six months of bereavement than that of married men of the same age and that death was most often from cardiac dysfunction.<sup>5</sup> Animal research from the 1970s concluded that changes in autonomic innervation, especially increased sympathetic activity, played a role in stress-induced cardiomyopathy and heart rate changes.<sup>6</sup>

Heart muscle damage resulting from pheochromocytoma (a usually benign adrenal medullary tumor characterized by excessive secretion of catecholamines, resulting in hypertension) has been shown to be similar to the myocardium cell degeneration due to the catecholamine surge found in sudden, severe emotional stress.<sup>7</sup> Analysis of pericardial fluid in victims of violence in which cardiac injury was not the cause of death showed catecholamine elevations greater than in those people who did die of heart attack, suggesting that catecholamine-mediated injury or stress cardiomyopathy is part of the physiologic response to emotional as well as physical trauma.<sup>8</sup>

Although Japanese research as early as 1991 suggested the presence of myocardial stunning or stress cardiomyopathy, the syndrome has only recently been recognized in the U.S. as something more than a folktale.<sup>9-12</sup> Over the past decade, evidence-based data suggest that contractile abnormalities, congestive heart failure, and even death can be associated with sudden emotional stress.<sup>1,2</sup>

### **The brain-heart connection**

A two-way relationship exists between the heart and the mind. Emotions and stressful experiences affect the heart through the autonomic nervous system and neuroendocrine pathways. People can experience cardiac activity and function as somatic symptoms, such as fatigue, lethargy, insomnia, and loss of appetite or as more classic cardiac symptoms like chest pain and palpitations.<sup>4,6</sup> Psychotropic agents can affect the heart, and drugs used to treat heart disease can affect the brain. Some antipsychotic medications, for example, can lower blood pressure or increase heart rate while a cardiac drug used to strengthen heart contractility can result in nervousness or apprehension.

Currently, little evidence supports the idea that people who develop stress cardiomyopathy have preexisting emotional disorders. The condition seems to be more directly related to the acuity of the stress. Even though we now are aware of certain physiologic changes that occur when someone is "scared to death" or has "a broken heart," further study needs to be done on how psychological factors contribute to stress cardiomyopathy.<sup>4,6</sup>

### **Putting the pieces back together**

The myocardial stunning that results from acute stress is most often reversible when identified rapidly and treated appropriately.<sup>9</sup> Nurses can provide the following when caring for patients with stress cardiomyopathy:

**Awareness:** Nurses should be aware that while cardiac disease is the No.1 killer of both women and men, women are more likely than men to have stress cardiomyopathy. In addition, nurses who understand that more than one explanation can exist for acute cardiac symptoms or for heart failure can be more effective in evaluating people who present with chest pain, palpitations, a sense of impending doom, and other symptoms classically associated with an MI. This awareness can be instrumental in accurate diagnosis and prompt treatment. Most crucial is the awareness that, like MI, stress cardiomyopathy requires urgent treatment and symptoms should never be dismissed.

The phenomenon of older people dying soon after the death of a spouse has focused attention on the risk for cardiac events in this population. Nurses need to understand that during bereavement, stress cardiomyopathy is a risk, particularly in otherwise healthy people.

**Differentiation of stress cardiomyopathy from other conditions:** Adequate assessment is crucial in determining whether cardiac symptoms indicate transient impairment in cardiac function or permanent damage to the heart muscle. The presenting symptoms are identical in MI and stress cardiomyopathy. Defining characteristics may be the presence of slightly elevated troponin I levels (no elevation to mild elevation is usual in stress cardiomyopathy), differences in the ECG pattern (see sidebar), and presence or absence of coronary artery disease (absent or minimal in stress cardiomyopathy). In other words, MI usually represents significant cardiovascular disease while stress cardiomyopathy seldom has this underlying disease process.<sup>1,2</sup>

The physical symptoms of a panic attack can look very much like those of acute coronary syndrome or stress cardiomyopathy. A misfiring of the emotional center of the brain (the limbic system, most often the amygdala) causes the body to react as if it were under severe threat. The physical symptoms of a panic attack can include palpitations, chest pain, diaphoresis, tremor, nervousness, and a feeling of doom. Patients, especially those experiencing a first panic attack, may present to an ED afraid that they are having heart attacks. Panic attacks are usually self-limiting, resolving in an hour or less (which may seem like an eternity to the person affected). Evaluation, monitoring, and emotional support are still needed while symptoms are sorted out.

More chronic in nature than panic attacks, depression and anxiety increase cardiac risk factors and vice versa. People with depression or anxiety are at higher risk to develop cardiac disease and experience cardiac events while those with cardiac conditions are more prone to develop symptoms of anxiety and depression. No link has been established between mood or anxiety disorders and stress cardiomyopathy, but further research may reveal changes in the feedback systems between the adrenals, the pituitary, the thyroid, and the autonomic nervous system that could apply to both acute and chronic conditions.<sup>13</sup>

Support and interventions: Appropriate diagnostic tests and emotional support of the patient and family are critical regardless of final diagnosis. Apprehension and a sense of doom, as well as distressing physical symptoms, need to be dealt with in patients with either MI or stress cardiomyopathy. Nurses can provide information and support matched to the person's level of anxiety and physical distress. The higher the anxiety, the less able the person is to process information. The nurse can listen, acknowledge feelings of distress, and provide reassurance that everything necessary is being done to obtain adequate diagnostic information and to treat symptoms accordingly. Immediate physical discomfort can be addressed in the same way as for an MI: Loosen clothing, position for adequate respiratory effort, provide a warm blanket for comfort or towel for diaphoresis, and administer oxygen and other medications as indicated by the patient's condition. Above all, the nurse should stay with the patient if at all possible and provides explanations to family members as needed.

Evaluation and follow-up: The rapid resolution of symptoms, the absence of coronary artery disease, and an ECG pattern characteristic of acute cardiac stress cardiomyopathy point to transient rather than permanent damage. However, the patient still needs rest and supportive treatment while the poorly functioning myocardium and neurohormones return to normal levels.

The links between mind and body continue to offer new frontiers for exploration and research — and new ways for nurses to understand the pathophysiology underlying phenomena such as stress cardiomyopathy. We now know that certain neurotransmitters, particularly serotonin and norepinephrine, modulate both physical pain and depression.<sup>14</sup> A review of literature also suggests that the emotional responses of "social pain," or rejection, may follow the same neural pathways as physical pain, perhaps serving similar purposes of self-preservation and self-protection.<sup>15</sup>

"Broken heart syndrome" is a real condition. The incidence is unknown, and the pathogenesis is not completely understood. An accurate diagnosis and appropriate medical management are possible following a complete clinical history and minimally invasive testing. With appropriate diagnosis, treatment is likely to be successful with no long-term complications. Recognition of the condition and immediate, appropriate care can be instrumental to the recovery of these otherwise healthy people.

*\*Name has been changed*

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