CME CREDIT

TAKE-HOME POINTS FROM EDUCATIONAL PRESENTATIONS BY CLEVELAND CLINIC FACULTY AND VISITING PROFESSORS

Is stress linked to heart disease? The evidence grows stronger

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ABSTRACT

Acute stress caused by strong emotions such as fear can sometimes cause sudden death in people with underlying coronary artery disease (CAD). Chronic mental stress may also promote the long-term development of coronary disease, although the distinction between Type A and Type B personalities appears overly simplistic. Stress-management interventions measurably improve CAD patients' performance on cardiac function tests, and should be incorporated more often in standard cardiac rehabilitation programs.

RECENT EVIDENCE confirms the popular folklore that mental stress has harmful effects on coronary health. In the short term, acute stress can trigger lethal cardiac events in susceptible patients, and in the long term, it appears to promote coronary artery disease.

In this article, I outline some of the mechanisms that relate mental stress to coronary disease and discuss recent research showing that stress-management programs may help prevent CAD.

HOW STRESS INFLUENCES CORONARY HEALTH

Coronary disease has three pathophysiological "dimensions": obstruction, vasoreactivity, and clotting. Mental stress can influence all three of these processes directly or indirectly by:

- Increasing blood pressure
- Increasing atherogenic lipid levels
- Decreasing endothelial-dependent vasodilation
- Increasing the risk of thrombosis
- Disposing patients to habits such as smoking, which compound coronary risk.[1,2]

CAN STRESS CAUSE SUDDEN DEATH?

Even though mental stress is popularly believed to cause heart attacks and sudden death, the phenomenon was not studied rigorously until recently. Recent studies indicate that stress precipitates heart attacks mainly in those who have predisposing factors, such as lipid-laden plaques or myocardial ischemia.[3]

One study showed an abrupt spike in the number of heart attacks in Tel Aviv at the beginning of the Persian Gulf War in January 1991, with surges in the incidence of heart attacks after each missile attack.[4]

A similar study revealed a spike in the number of sudden deaths related to atherosclerotic cardiovascular disease on the day of the Los Angeles earthquake of January 17, 1994.[5] On that day, 24 such deaths occurred, compared with a daily mean of 4.6 in the preceding week. Notably, the mean number of sudden cardiac deaths in the week after the earthquake was significantly lower than baseline: only 2.7 deaths per day. This suggests that the earthquake hastened the deaths of people who were already at risk of dying.

Remember, however, that stress-induced sudden cardiac deaths are rare, even in the worst situations. The 24 people who died of cardiac disease on the day of the earthquake constitute only a tiny fraction of the more than 8 million people who live in Los Angeles.

Stress decreases endothelial-dependent vasodilation

STRESS EXACERBATES ACUTE ISCHEMIA

To test the effect of mental stress on heart function, researchers have developed several "mental stress tests" that can be performed on volunteers in a laboratory.

The mental arithmetic stress test requires a subject to subtract numbers from a four-digit number while the evaluator urges him or her to work faster and faster. In the Stroop color-word task, subjects are shown the name of a color printed in ink of another color. For example, the word "green" might be printed in red. After the word is taken away, the subject is asked to recall the color of the ink.

Using such tests, several researchers demonstrated reproducible causal relationships between acute mental stress and myocardial ischemia, as indicated by ST-segment deviation and wall-motion abnormalities.[1,6] Mental stress tests can also distinguish between subjects with a normal hemodynamic response to stress and those who exhibit an exaggerated response. These latter "hot responders" are more likely to have occult myocardial ischemia during normal daily activities and during mental stress.[7]

In cardiac rehab, stress management reduces morbidity and mortality

Mental stress-induced ischemia is associated with significantly higher rates of subsequent cardiac events, both fatal and non-fatal, than is exercise-induced ischemia.[8]

In addition, Mittleman[9] showed that an outburst of anger can trigger acute myocardial infarction, but that aspirin may reduce the risk.

PERSONALITY TYPE AND CARDIOVASCULAR DISEASE

Beginning in the 1960s, the relationship between personality types and disease became the focus of speculation. Competitive, aggressive, impatient people were described as having the Type A personality and were thought to be prone to cardiovascular disease, while the calmer Type Bs were considered at lower risk.[10]

Unexpectedly, long-term follow-up studies of Type A and Type B cohorts revealed that the Type As actually had lower mortality from cardiac problems.[11]

Findings such as these led to speculation that there may be more than one physiological reaction to stress. Some people might love stress and choose professions (such as emergency medicine) and hobbies (such as skydiving) that give them frequent adrenaline boosts. Many of these people would be classically described as Type A personalities and considered at risk of stress-related illness. But it is possible that these people, who feel "in control" when faced with stress, have different neurohumoral and hemodynamic responses than those who feel defeated or fearful in far less intense situations.

STRESS MANAGEMENT FOR TREATING HEART DISEASE

Stress management is not a standard part of cardiac rehabilitation, but it probably should be.

In a striking prospective study, researchers at Duke University randomly assigned coronary disease patients to a 4-month course of either aerobic exercise, stress-management training, or standard medical therapy.[12]

The stress-management patients received instruction about heart disease and stress, underwent biofeedback training, and learned progressive muscle relaxation exercises. In group therapy and cognitive behavior therapy sessions, they learned to monitor irrational thoughts and generate less stressful alternatives.

Two years later, compared with the medical therapy group, the stress-management group had had 74% fewer heart attacks or other adverse heart events ($P = .03$). Although the exercise patients lost weight and increased their aerobic fitness, the difference between their risk of cardiac events and that of the usual-care patients was not significant (relative risk = 0.68, $P = .41$).

A recent meta-analysis of 23 randomized controlled trials[13] found that adding psychosocial treatments to standard cardiac rehabilitation programs reduces mortality and morbidity, particularly during the first 2 years after treatment. Psychosocial treatments also reduce psychological distress and ameliorate some of the biological risk factors for myocardial infarction.

More studies are needed, but the existing data should be enough to convince cardiolo-

gists to include psychosocial interventions in prevention and rehabilitation programs.

REFERENCES

1. **Rozanski A, Bairey CN, Krantz DS, et al.** Mental stress and the induction of silent myocardial ischemia in patients with coronary artery disease. N Engl J Med 1988; 318:1005–1012.
2. **Muller J, Abela G, Nestro R, Tofler G.** Triggers, acute risk factors and vulnerable plaques: The lexicon of a new frontier. JACC 1994; 23:809–813.
3. **Lecomte D, Fornes P, Nicolas G.** Stressful events as a trigger of sudden death: a study of 43 medico-legal autopsy cases. Forensic Sci Int 1996;79:1–10.
4. **Meisel SR, Kutz I, Dayan KI, et al.** Effect of Iraqi missile war on incidence of acute myocardial infarction and sudden death in Israeli civilians. Lancet 1991; 338:660–661.
5. **Leor J, Poole WK, Kloner RA.** Sudden cardiac death triggered by an earthquake. N Engl J Med 1996; 334:413–419.
6. **Blumenthal JA, Jiang W, Waugh RA, et al.** Mental stress-induced ischemia in the laboratory and ambulatory ischemia during daily life. Association and hemodynamic features. Circulation 1995; 92:2102–2108.
7. **Gottdiener JS, Krantz DS, Howell RH, et al.** Induction of silent myocardial ischemia with mental stress testing: relation to the triggers of ischemia during daily life activities and to ischemic functional severity. JACC 1994; 24:1645–1651.
8. **Jiang W, Babyak M, Krantz DS, et al.** Mental stress-induced myocardial ischemia and cardiac events. JAMA 1996; 275:1651–1656.
9. **Mittleman MA, Maclure M. Sherwood JB, et al.** Triggering of acute myocardial infarction onset by episodes of anger. Determinants of Myocardial Infarction Onset Study Investigators. Circulation 1995; 92:1720–1725.
10. **Friedman M, Rosenman R.** Association of specific overt behavior pattern with blood and cardiovascular findings: blood cholesterol level, blood clotting time, incidence of arcus senilis, and clinical coronary artery disease. JAMA 1959; 169:1286–1296.
11. **Ragland DR, Brand RJ.** Type A behavior and mortality from coronary heart disease. N Engl J Med 1988; 318:65–69.
12. **Blumenthal J, Jiang W, Babyak M, et al.** Stress management and exercise training in cardiac patients with myocardial ischemia. Arch Intern Med 1997; 157:2213–2223.
13. **Linden W, Stossel C, Maurice J.** Psychosocial interventions for patients with coronary artery disease. Arch Intern Med 1996; 156:745–752.

CME ANSWERS

CME 2 CREDIT HOURS

Answers to the CREDIT TEST on page 127 of this issue

1 **E** 2 **C** 3 **D** 4 **D** 5 **C** 6 **A** 7 **A** 8 **A** 9 **B** 10 **D** 11 **B** 12 **E**