



Literature Reviews

Pathophysiological processes underlying emotional triggering of acute cardiac events.

Strike PC, Magid K, Whitehead DL, Brydon L, Bhattacharyya MR, Steptoe A. *Proc Natl Acad Sci USA* 2006 Mar 14;103(11):4322-7.

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Abstract: Every time comedian Redd Foxx became angry or disappointed, he would histrionically clutch his chest and announce that he was joining his departed wife. The humor depended on the grim plausibility of a lethal reaction to emotional stress. Though negative emotions are long suspected to prompt acute coronary events, the phenomenon is difficult to examine with high scientific control. Researchers from University College London interviewed 34 men after they had survived a myocardial infarction or a bout of unstable angina. Fourteen of the patients reported acute negative emotion in the 2-hour period before onset of symptoms. Examples included "arguments with neighbors, family conflict, anniversaries of bereavements," and, a good one, "frustrating commuting." About a year later, in comparison with the rest of the patients, the fourteen exhibited markedly increased platelet aggregation when blood was sampled soon after deliberate psychological stress.

There were two experimental stresses. One was a challenge to identify target color words printed over incongruous colors on a computer screen. This challenge of trying to push the right buttons on the computer increased systolic blood pressure and cardiac index (calculated from finger pulse pressure recordings) in both groups of patients. There is a lesson there. The computer stressor was followed by a public speaking challenge that further increased blood pressure and cardiac index. Those cardiovascular indices were slower to recover after the "emotional" group was stressed.

Peripheral venous blood samples were citrated and incubated with color-tagged antibodies against various cell surface antigens. Flow cytometry then quantified leukocyte-platelet, monocyte-platelet, and neutrophil-platelet aggregates. The results are striking. The patients who did not identify an emotional trigger for their coronary event did not exhibit an increase in platelet aggregates in response to experimental stress. The patients for whom an emotional stress may have triggered a coronary event exhibited a significant rise in circulating aggregates during experimental stress, and the rise peaked at 30 minutes after the experimental stress.

The authors conclude that "some patients with coronary artery disease may be particularly susceptible to emotional triggering of acute coronary syndrome because of heightened platelet activation in response to psychological stress, coupled with impaired hemodynamic poststress recovery."

Comments: The difficulty in achieving perfect isolation of variables in a clinical study of this subject is obvious. When possible, aspirin and beta-blockers were withheld for experimental testing. However, both drugs were continued more often in the emotion-triggered patients. This limitation of the study does not seem to quantitatively explain the impressive platelet differences found between the two groups.

Leukocyte-platelet aggregates form when platelets are stimulated to move leukocyte-binding P-selectin molecules from intracellular alpha granules to the platelet surface. This measure of platelet activation is plausibly connected to coronary thrombosis, but the connection is, of course, hypothetical in this study.

Interestingly, our hospitals are no doubt emotionally stressful for our cardiovascular patients.