



From the Desk of Dr. Stephen Sinatra

## Ventricular Tachyarrhythmia and Psychological Stress

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Reprinted from JAMA Vol. 258, No. 6, August 14, 1987 the second enema, he began to complain of weakness and his wife noted that his gait had become unsteady. He quickly became profoundly weak and paramedics were summoned.

Resuscitation was attempted, but the patient remained asystolic. Results of initial laboratory tests were still pending when intravenous calcium was administered, in the belief that calcium might have been bound by phosphate. To our astonishment, electrical activity occurred. A second bolus of calcium was given and the patient had a pulse! Within minutes, the patient stabilized, with a sinus rhythm and normal blood pressure. He was transferred to the intensive care unit, where he regained consciousness. I do not know what happened to him thereafter, but his initial blood test showed a greatly decreased calcium level.

Comment.—That story has always stayed with me. Knowledge of medicine is a continuum, and as we collect data, sometimes we find pieces of the puzzle that have been missing for years. In the meantime, we have enough observations to warrant suspicion. At the very least, supportive data such as this can only help a fledgling teacher. It certainly decreases the eye rolling one gets from residents when one asks if there's a history of taking an enema.

Ricardo Martinez, MD Stanford (Calif) University

 Martin RR, Lisehora GR, Braxton M Jr, et al: Fatal poisoning from sodium phosphate enema: Case report and experimental study. JAMA 1987;257:2190-2192.

## Ventricular Tachyarrhythmia and Psychological Stress

To the Editor.—Brodsky et al' should be congratulated for a fine presentation revealing the connection between psychological stress and life-threatening arrhythmia. They raise a crucial issue: some individuals with complex ventricular arrhythmia in the setting of emotional stress and high sympathetic tone are at risk for sudden cardiac death regardless of the presence of coronary artery disease or the status of their left ventricular function. β-Blockers appear to be cardioprotective for patients in this subset. The reasons why some individuals respond to psychological stress with malignant arrhythmia are both unclear and provocative. There is a connection between emotional stress and cardiac vulnerability. Behavioral response to stress and subsequent neuroendocrine response to emotion will eventually occur in susceptible individuals, but sensitivity to emotion is variable. Some individuals prone to cardiovascular disorders probably have

in their character an oversensitivity to emotional arousal, psychological conflict, or personal loss. Such arousal can be manifested by physiological overreactivity<sup>2</sup> or neurohormonal biochemical oversensitivity.<sup>3</sup>

It would have been useful in determining whether any of the patients were overreactors if Brodsky et al had measured total peripheral resistance, mean arterial blood pressure, and cardiac output. Such physiological overreactivity in the laboratory may have also correlated with arrhythmia sensitivity. Moreover, biochemical assessments of norepinephrine, epinephrine, and urinary 17-hydroxycorticosteroid levels would have yielded biochemical information regarding patients' arousal in response to anger, anxiety, and vigilance.2 The discovery of elevated baseline levels of catecholamines, for instance, may have also given a sounder reason for the use and efficacy of βblockers.

The authors concluded that the impact of psychological stress and depression, particularly in Asian immigrants, may have also been a factor in the development of life-threatening ventricular tachyarrhythmia.

I would like to suggest the term heartbreak for an additional factor in the occurrence of such myocardial vulnerability. All patients in the study had heartbreak, manifested by the loss of a vital connection.

Cannon studied the relationship of emotional stress and "voodoo death." Although fear and terror were contributing factors in the "Curse-Death syndrome," Cannon concluded that the deaths of the ostracized victims were commonly preceded by intense loneliness, despondency, and social isolation. Ruberman et al<sup>5</sup> discussed the negative effects of social isolation on subsequent mortality in survivors of myocardial infarction. In contemporary society, such loss of family, relationships, and culture can result in loss of support, emotional arousal, and perhaps cardiac vulnerability. The pain of longing for a loved person who is unavailable is heartbreaking. There are many instances of sudden cardiac death after the loss of a loved one. The loss of a vital connection (spouse, parent, family member, pregnancy, or culture) and inability to establish a new connection, the feeling of heartbreak, or the feeling of being trapped can have serious consequences for the cardiovascular system.

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In Reply.-The letter by Dr Sinatra raises important issues regarding the association between psychological stress and cardiac arrhythmia. He details a number of potential physiological and biochemical factors that can be measured in individuals with this disorder. We did evaluate a number of these parameters in our patients at various times during their assessment. Two patients had labile hypertension. In the other patients, baseline cardiovascular physiological parameters, including total peripheral resistance, were normal. We measured urinary vanillylmandelic acid and catecholamine levels in two patients, and these were normal as well. During these periods, patients experienced the stress associated with hospitalization and invasive cardiac testing. However, at that time we did not subject the patients to a systematic program of induced psychological stress. We are currently using psychological stress testing in patients with these types of disorders.

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## CORRECTION

Incorrect References.—An error occurred in the original contribution entitled "A Multifactorial System for Equitable Selection of Cadaver Kidney Recipients," published in the June 12 issue of The Journal (1987;257:3073-3075). On page 3075, the references are incorrect. The following should have appeared as the references: 1. Terasaki PI, Himaya NS, Cecka M, et al: Overview, in Terasaki PI (ed): Clinical Transplants 1986. Los Angeles, UCLA Tissue Typing Laboratory, 1986, pp 387-392. 2. Lundgren G, Groth CG, Albrechtsen D, et al: HLA-matching and pretransplant blood transfusions in cadaveric renal transplantation: A changing picture with cyclosporine. Lancet 1986;2: 66-69. 3. Alexander UW, Vaughn WK, Pfaff WW: Local use of kidney with poor HLA match is as good as shared use with good matches in cyclosporine era: An analysis at one and two years. Transplant Proc 1987;19: 672-674.