

tion. I believe the abnormalities are actually due to severe generalized epicardial injury and ischemia; they are not imitators.

The ST-segment and T-wave abnormalities are caused by an abrupt elevation of serum catecholamine levels.²⁻⁵ An excess of catecholamines can damage myocytes directly and can also lead to generalized spasm of the coronary arteries. Actual cardiac infarction can result, especially if coronary atherosclerosis is present. This type of myocardial damage may be seen in patients who inject cocaine, those who are receiving intravenous norepinephrine, and those who have pheochromocytoma, startle reaction (“deer hunter’s heart attack”), or a severe emotional crisis.²⁻⁵

The word “mimicking” in the title of the article is misleading, and the words “erroneous diagnosis” at the end of the article are not correct. The abnormalities on the electrocardiogram are real: they are the result of epicardial injury and ischemia due to abruptly elevated catecholamine levels.

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THE AUTHORS REPLY: We appreciate Dr. Hurst’s comments regarding the potential cause of the marked

ST-segment and T-wave abnormalities in our 19-year-old patient, but we do not believe that the electrocardiographic pattern of acute injury was the result of severe generalized epicardial injury and ischemia. The electrocardiographic abnormality persisted for more than 24 hours, yet the echocardiogram revealed normal systolic and diastolic left ventricular function. Four serial measurements of cardiac troponin I (a sensitive biomarker of myocardial necrosis) were obtained because of the abnormal electrocardiographic finding, and all were within normal limits — a finding similar to that in a report by others who used the less specific creatine kinase MB test.^{1,2} Furthermore, normal autopsy findings in patients with intracranial hemorrhage and marked ST-segment and T-wave abnormalities have been reported.^{3,4}

Myocardial ischemia or infarction can certainly occur in patients with an intracranial hemorrhage, as Dr. Hurst points out, but we believe the electrocardiographic abnormalities in our patient are more likely explained by cardiac autonomic dysfunction as a result of the left temporal intracranial hemorrhage.

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Molecular Epidemiology of SARS — From Amoy Gardens to Taiwan

TO THE EDITOR: The outbreak of severe acute respiratory syndrome (SARS) in Taiwan described by Hsieh (Aug. 14 issue)¹ grew rapidly from mid-April 2003. We note the chronologic relation between this rapid wave of infections and the visit of a resident of Amoy Gardens, Hong Kong, to Taiwan on March 26.² He rode the train with a female passenger who was linked to a series of local cases.

We recently sequenced the complete genome of the SARS coronavirus associated with the outbreak in Amoy Gardens.³ A comparison of the sequence with those of all other SARS-coronavirus genomes accessible at GenBank (<http://www.ncbi.nlm.nih.gov>) in mid-May revealed two polymorphisms (at nucleotides 3852 and 11493, according to the sequence of GenBank accession number AY274119)

that are found only in the isolates from Amoy Gardens.³ On September 23, another comprehensive comparison with GenBank sequences was performed. Among all the complete genomes of the SARS coronavirus that have been made available publicly to date, only 10 recently submitted Taiwanese isolates share the two-polymorphism fingerprint of the Amoy Gardens isolates; these Taiwanese isolates are designated as TC1, TC2, TC3, TWH, TWJ, TWK, TWS, TWY, TWC2, and TWC3. These molecular data demonstrate that the same strain of the SARS coronavirus was involved in the Amoy Gardens outbreak and the late outbreak in Taiwan. These data provide objective support for the epidemiologic investigations of the World Health Organ-

ization and further demonstrate the usefulness of molecular epidemiology.³

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