5. Maimonides M, Halkin A: Crisis and Leadership: Epistles of Maimonides. Philadelphia, Jewish Publication Society, 1985, p 292

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The Use of Iconic Symbols to Represent Our Experience

TO THE EDITOR: The sculpture in the Images in Psychiatry article by Peter M. Bulow, M.D., M.F.A., as depicted in the March 2008 issue of the *Journal,* is a remarkable piece of art that succeeds in evoking a sense of pathos. Dr. Bulow stated that in conceiving the sculpture he was drawing upon "our common cultural heritage" (1, p. 334), and he refers to images of the Pietà and the Great Mother.

However, it should be noted that there are iconic sources both prior to the Pietà and relatively recent that are drawn upon. I refer first to *Roman Charity*, the story of Pero, who breast fed her father Cimon on her visits to his prison cell where he was condemned to starve to death. This story has been represented in many works of art throughout the centuries. The more recent story that could be referenced by Dr. Bulow's sculpture is that of Rose of Sharon Joad, who suffered a miscarriage and breast fed a starving, dying man in *The Grapes of Wrath*, by John Steinbeck.

The sources that Dr. Bulow drew upon to create his sculpture contribute to a sense of timelessness in experiencing a depiction of those who move through, share, and lose time.

Reference

1. Bulow PM: Alzheimer's Madonna. Am J Psychiatry 2008; 165: 334

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Father-to-Son Transmission of 6;17 Translocation in Tourette's Syndrome

TO THE EDITOR: Genetic inheritance of Tourette's syndrome was observed as early as the 19th century (1). To date, no clear susceptibility genes for Tourette's syndrome have been identified. However, a recent linkage study that utilized a whole-genome screen found evidence of linkage of Tourette's syndrome to a region on chromosome 2p (2). The identification of chromosomal structural aberrations that coincide with a particular phenotype could provide insight into the genesis of this disease. We report the case of a father and son who were clinically diagnosed with Tourette's syndrome and had a shared chromosomal alteration.

"Mr. A" was a 47-year-old married man who suffered from motor tics, such as head jerking, teeth grinding, and touching walls. Additionally, he experienced symptoms of coprolalia. Apart from the tics, no abnormality was detected in his clinical examination. His family history was positive, with a 9-year-old son who also suffered from complex motor and single vocal tics as well as a learning disability. Both father and son were diagnosed with Tourette's syndrome in accordance with DSM-IV criteria (3). Mr. A's 12-year-old daughter did not show any symptoms of the disease. Peripheral blood samples were obtained from Mr. A and both children and their mother, with informed consent from the parents and the children. G-banding by Trypsin-Giemsa analysis and karyotyping of metaphase chromosomes were undertaken via peripheral blood lymphocytes in accordance with standard cytogenetic protocols. Both the affected father and the affected son exhibited the same balanced translocation t(6;17)(q21;p11). The mother and daughter showed a normal karyotype. Since the children's grandparents were deceased, cytogenetic examination of the grandparents, who had been healthy and not consanguineous, was not possible. Thus, a de novo translocation could not be excluded.

In our case, a father-to-son transmission of a balanced translocation involving chromosomes 6q21 and 17p11, segregating with Tourette's syndrome, was observed. Chromosome 17p11 was identified for suggestive linkage in a linkage analysis of affected sibling pairs with attention deficit hyperactivity disorder—also a Tourette's syndrome spectrum disorder (4)—as well as for linkage in schizophrenia patients (5). The biological and physiological parallels of Tourette's syndrome and schizophrenia have been described previously (6).

The translocation found could have emerged in a former generation, and a coincidentally occurring segregation is still a possibility. Even if this is the case, isolation of the genomic region at the site of the translocation breakpoints on chromosomes 6 and 17 might be useful in the search for candidate Tourette's syndrome genes.

References

1. Charcot JM: Poliklinische Vorträge, Band I–II. Wien, Deuticke, 1894
2. Tourette Syndrome Association International Consortium for Genetics: Genome scan for Tourette disorder in affected-sibling-pair and multigenerational families. Am J Hum Genet 2007; 80:265–272
3. American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 4th Ed. Washington, DC, American Psychiatric Press, 2000
4. Ogdie MN, Fisher SE, Yang M, Ishii J, Francks C, Loo SK, Cantor RM, McCracken JT, McGough JJ, Smalley SL, Nelson SF: Attention deficit hyperactivity disorder: fine mapping supports linkage to 5p13, 6q12, 16p13, and 17p11. Am J Hum Genet 2004; 75: 661–668
5. Williams NM, Norton N, Williams H, Ekholm B, Hamshere ML, Lindblom Y, Chowdari KV, Cardno AG, Zammit S, Jones LA, Murphy KC, Sanders RD, McCarthy G, Gray MY, Jones G, Holmans P, Nimgaonkar V, Adolfson R, Osby U, Terenius L, Sedvall G, O'Donovan MC, Owen MJ: A systematic genomewide linkage study in 353 sib pairs with schizophrenia. Am J Hum Genet 2003; 73:1355–1367
6. Muller N, Riedel M, Zawta P, Gunther W, Straube A: Comorbidity of Tourette's syndrome and schizophrenia: biological and

physiological parallels. Prog Neuropsychopharmacol Biol Psychiatry 2002; 26:1245–1252

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Takotsubo Cardiomyopathy in a Patient With Emotional Stress

TO THE EDITOR: Takotsubo cardiomyopathy is a condition that manifests as a segmental wall motion abnormality that typically undergoes spontaneous reversal. This condition has also been referred to as the "broken heart syndrome" because of its appearance following acute emotional stress (1). We report the case of a patient who experienced Takotsubo cardiomyopathy that was relieved after sedative medication was prescribed.

"Ms. A" was a 35-year-old woman who was admitted to our hospital with typical anginal pain that began following the unexpected death of her brother. She had experienced chest pain for a duration of 3 hours and had no prior history of cardiovascular disease.

On physical examination, the patient was awake and alert. Her blood pressure was 120/90 mmHg; her respiratory rate was 28 breaths per minute; and her heart rate was normal, with no arrhythmia. An electrocardiogram (ECG) showed ST segment elevation in the anterior leads. The patient was initially diagnosed with acute myocardial infarction, and her laboratory evaluation showed a small increase in cardiac enzymes and troponin (in serial measurements). However, a coronary angiogram revealed normal coronary arteries.

Because of her persistent chest pain, a transthoracic echocardiography was performed, which revealed left ventricular akinesia of the apical segment and hyperkinesis of the basal segment. Consequently, a left ventriculography was also performed because Takotsubo cardiomyopathy was suspected.

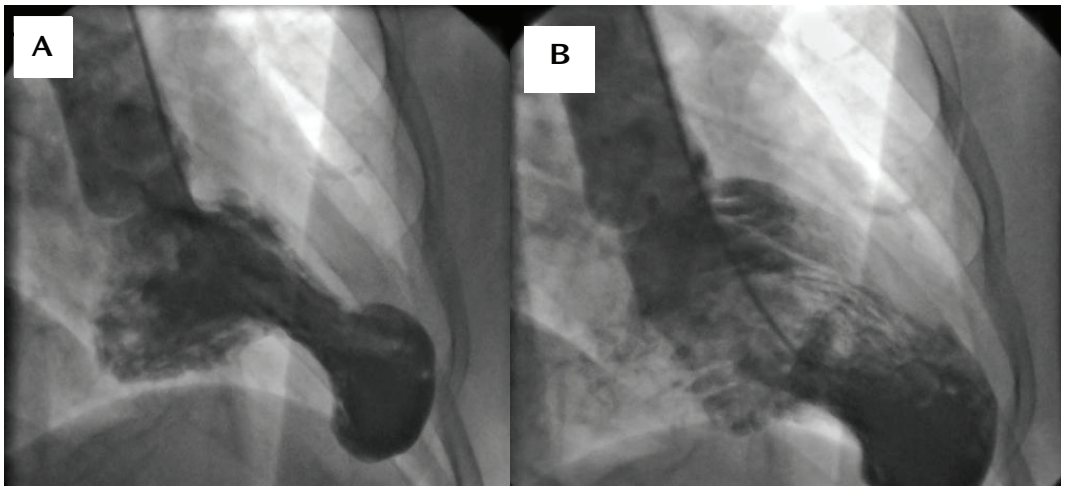
After she received a sedative medication, Ms. A experienced relief from the chest pain, and she experienced no further angina symptoms during the remainder of her hospital stay. After her discharge, a repeat echocardiography performed on day 10 revealed no wall motion abnormalities.

Although there is no specific treatment for Takotsubo cardiomyopathy, the prognosis for individuals who are diagnosed with the condition is usually good, and recurrence is rare.

The most likely underlying mechanism in the pathogenesis of Takotsubo cardiomyopathy is a sudden excessive catecholamine surge and increased sympathetic activity (2). Symptoms include a sudden onset of chest pain, ECG abnormalities (similar to those of acute myocardial infarction), normal coronary arteries (revealed on cardiac catheterization), and a signature appearance (revealed on ventriculography and transthoracic echocardiography) (3).

Psychological distress has been shown to result in increased risk of myocardial infarction (4). This association appears to occur primarily in patients with known atherosclerotic heart disease, in whom stress may contribute to the pathogenesis of atherosclerotic plaques. In contrast, the role of emotional stress in the pathogenesis of Takotsubo cardiomyopathy and myocardial infarction in individuals with normal coronary arteries is less understood (4, 5).

FIGURE 1. Left Ventriculography[a]



[a] Illustration of the ballooning shape of the left ventricle in systole (A) compared with diastole (B).

References

1. Virani SS, Khan AN, Mendoza CE, Ferreira AC, de Marchena E: Takotsubo cardiomyopathy, or broken-heart syndrome. Tex Heart Inst J 2007; 34:76–79
2. Lyon AR, Rees PS, Prasad S, Poole-Wilson PA, Harding SE: Stress (Takotsubo) cardiomyopathy: a novel pathophysiological hypothesis to explain catecholamine-induced acute myocardial stunning. Nat Clin Pract Cardiovasc Med 2008; 5:22–29