

1. Gorlin RJ, Cohen MM Jr, Condon LM, Burke BA, Bannayan-Riley-Ruvalcaba syndrome. *Am J Med Genet* 1992;44:307-14.
2. Jones KL. *Smith's recognizable patterns of human malformation*. 5th ed. Philadelphia: W.B. Saunders, 1997.

The Electrocardiogram in Acute Myocardial Infarction

TO THE EDITOR: The review article by Zimetbaum and Josephson (March 6 issue)¹ on the usefulness of the electrocardiogram in the diagnosis of acute myocardial infarction is essential reading for anyone who cares for patients with a suspected acute myocardial infarction. Some qualifications regarding the diagnosis of acute myocardial infarction in patients with left bundle-branch block are in order. Although proposed criteria² facilitate the diagnosis, an occasional problem arises when reliance on the criterion of an ST-segment elevation of more than 5.0 mm in leads with primarily negative QRS complexes leads to a false positive diagnosis of acute myocardial infarction. This criterion is non-specific for acute myocardial infarction, and some patients who have left bundle-branch block without an acute myocardial infarction, but with left ventricular hypertrophy or dilatation, have electrocardiograms with ST-segment elevations that are much larger than 5.0 mm.^{3,4} An association between large ST-segment elevations and large QRS complexes has been reported.³ Repeating electrocardiography may also be of value, since a change in the amplitude of the ST-segment elevation suggests an acute myocardial infarction.⁵ Finally, since measurements of ST-segment elevations are being used for the diagnosis of acute myocardial infarction in patients with left bundle-branch block, it is prudent to mark the thorax in order to ensure reproducible serial electrocardiograms.⁵

John E. Madias, M.D.

Mount Sinai School of Medicine
New York, NY 10029
madiasj@nychhc.org

1. Zimetbaum PJ, Josephson ME. Use of the electrocardiogram in acute myocardial infarction. *N Engl J Med* 2003;348:933-40.
2. Sgarbossa EB, Pinski SL, Barbagelata A, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the

presence of left bundle-branch block. *N Engl J Med* 1996;334:481-7. [Erratum, *N Engl J Med* 1996;334:931.]

3. Madias JE, Sinha A, Ashtiani R, Agarwal H, Win M, Narayan VK. A critique of the new ST-segment criteria for the diagnosis of acute myocardial infarction in patients with left bundle-branch block. *Clin Cardiol* 2001;24:652-5.

4. Madias JE, Sinha A, Agarwal H, Ashtiani R. ST-segment elevation in leads V1-V3 in patients with LBBB. *J Electrocardiol* 2001;34:87-8.

5. Madias JE. Serial ECG recordings via marked chest wall landmarks: an essential requirement for the diagnosis of myocardial infarction in the presence of left bundle branch block. *J Electrocardiol* 2002;35:299-302.

THE AUTHORS REPLY: We agree entirely with the comments of Dr. Madias. The criteria for a diagnosis of myocardial infarction in a patient with left bundle-branch block are flawed. Not only is it difficult to interpret ST-segment changes in a patient with left bundle-branch block and preexisting left ventricular hypertrophy or dilatation, but also the coexistence of multivessel coronary artery disease or left ventricular aneurysm will most likely limit the ability to diagnose acute myocardial infarction electrocardiographically. We chose to present data from the largest series of patients with documented acute myocardial infarction.¹ The editorial by Wellens that accompanied the report on that series further highlighted the difficulties involved in assessing acute myocardial infarction in the presence of left bundle-branch block.²

Peter Zimetbaum, M.D.

Mark E. Josephson, M.D.

Beth Israel Deaconess Medical Center
Boston, MA 02215
pzimetba@bidmc.harvard.edu

1. Sgarbossa EB, Pinski SL, Barbagelata A, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. *N Engl J Med* 1996;334:481-7. [Erratum, *N Engl J Med* 1996;334:931.]

2. Wellens HJJ. Acute myocardial infarction and left bundle-branch block — can we lift the veil? *N Engl J Med* 1996;334:528-9.

Vestibular Neuritis

TO THE EDITOR: Droperidol (2.5 mg to 10 mg) was mentioned as a treatment option in the Clinical Practice article on vestibular neuritis by Baloh (March 13 issue),¹ with “liver or kidney disease” mentioned as a precaution. In December 2001, the Food and Drug Administration (FDA) strengthened its warnings about the use of droperidol, specifically add-

ing a “black box” warning about deaths associated with prolongation of the QT interval and torsade de pointes. It was recommended that droperidol be reserved for the treatment of patients whose condition was unresponsive to other therapies and that it be used only if the benefit outweighed the risk; it was also recommended that monitoring of vital

signs and electrocardiography be routinely performed during therapy.

At that time, through the Freedom of Information Act, I reviewed actual reports submitted to the FDA and found that, although most reports were from Europe and involved higher-than-usual doses (25 mg), some adverse cardiac events were reported at the lower end of the range of doses (1.25 mg). I believe that this information is relevant to clinicians who are planning to use droperidol to treat vestibular neuritis.

E. Jane Orr, Pharm.D.

Wellmont Holston Valley Medical Center
Kingsport, TN 37662
e_jane_orr@wellmont.org

1. Baloh RW. Vestibular neuritis. *N Engl J Med* 2003;348:1027-32.

DR. BALOH REPLIES: Dr. Orr points out a serious potential side effect of droperidol. Although I have little personal experience with this drug, there have been multiple studies describing good results in treating acute vertigo with droperidol, with no serious side effects.¹ However, given this potential adverse effect, I would not use droperidol as a primary agent for the treatment of vestibular neuritis.

Robert W. Baloh, M.D.

UCLA School of Medicine
Los Angeles, CA 90095-1769
rwbaloh@ucla.edu

1. Irving C, Richman P, Kaiafas C, Eskin B, Allegra J. Intramuscular droperidol versus intramuscular dimenhydrinate for the treatment of acute peripheral vertigo in the emergency department: a randomized clinical trial. *Acad Emerg Med* 2002;9:650-3.

Postmenopausal Hormone Therapy

TO THE EDITOR: The assertion by Grodstein et al. (Feb. 13 issue)¹ in their Sounding Board article that confounding due to socioeconomic status is unlikely to explain the different reported associations between postmenopausal hormone therapy and cardiovascular disease in observational studies and in randomized clinical trials rests on flawed assumptions. Among these are the assumptions that the Nurses' Health Study "controlled for educational level and occupation by including only registered nurses" (with some adjustment for husband's educational level) and that the Leisure World study "adjusted for income by recruiting exclusively from a middle-class retirement community."

These three socioeconomic variables — education, occupation, and a surrogate for income — are at best partial measures, with occupation particularly ill-suited for analyses of women's health.² Evidence, including some from the Nurses' Health Study,³ indicates that the risk of cardiovascular disease among women (and men) is shaped by lifetime socioeconomic status, beginning in utero⁴; lifetime socioeconomic factors may also affect age at menopause.⁵ Greater precision in socioeconomic as well as biologic variables is needed in order to resolve discrepancies in research about postmenopausal hormone therapy.

Nancy Krieger, Ph.D.

Harvard School of Public Health
Boston, MA 02115

1. Grodstein F, Clarkson TB, Manson JE. Understanding the divergent data on postmenopausal hormone therapy. *N Engl J Med* 2003; 348:645-50.

2. Krieger N, Williams DR, Moss NE. Measuring social class in US public health research: concepts, methodologies, and guidelines. *Annu Rev Public Health* 1997;18:341-78.

3. Gliksman MD, Kawachi I, Hunter D, et al. Childhood socioeconomic status and risk of cardiovascular disease in middle aged US women: a prospective study. *J Epidemiol Community Health* 1995; 49:10-5.

4. Davey Smith G, Gunnell D, Ben-Shlomo Y. Lifecourse approaches to socio-economic differentials in cause-specific adult mortality. In: Leon D, Walt G, eds. *Poverty, inequality, and health: an international perspective*. Oxford, England: Oxford University Press, 2000:88-124.

5. Wise LA, Krieger N, Zierler S, Harlow BL. Lifetime socioeconomic position in relation to onset of perimenopause. *J Epidemiol Community Health* 2002;56:851-60.

THE AUTHORS REPLY: We explored the issue of confounding by socioeconomic status in epidemiologic studies of postmenopausal hormone therapy and coronary heart disease; we did not seek to review the complex relation between socioeconomic status and coronary heart disease. In many observational studies, postmenopausal hormone users are of higher socioeconomic status than nonusers,¹ which may lead to lower rates of coronary heart disease that are attributable to improved access to health care and healthier lifestyles. Use of cohorts such as that in the Nurses' Health Study inherently provides adjustment (although not complete adjustment) for such potential confounding, since all participants are registered nurses and have access to health care and knowledge about health. For example, in the Nurses' Health Study, the rates of cholesterol and blood-pressure screening and the intake of saturated fats are similar among hormone users and nonusers; adjustment for husband's education, childhood socioeconomic status, or house-