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sudden progression from sinus rhythm with a normal baseline PR interval to sudden heart block at the time of cementing argues strongly for a primary cause from methylmethacrylate.

Quinidine, a class Ia antiarrhythmic, also has the potential to cause heart block below the AV node, thus measuring the baseline PR interval may not tell the "whole story" in determining quinidine potential for causing heart block. Since the His-Purkinje system contributes less than 70 ms to the PR interval, there could be significant effect on this fast-conducting tissue with normal PR interval. However, conduction toxicity with quinidine is followed adequately with measurement of the QRS duration. When there is a doubling of this measurement, there is high likelihood of toxicity. The fact that this patient had a baseline QRS duration of 80 ms, well within normal limits, suggests that there was not a doubling of QRS duration from quinidine, and thus it is unlikely to be toxic to the conduction system. The more serious arrhythmias with quinidine relates to re-entry and torsades de pointes, which usually accompanies long QT intervals.⁴

Subclinical hypothyroidism could have contributed to the heart block along with the digitalis, but because of the timing of the heart block and baseline PR interval, this too seems unlikely to have had more than a small impact on the clinical outcome.

We agree with the comments regarding "proper preoperative medical evaluation," which needs to be done before any anesthetic. In retrospect, we agree that other tests should have been performed. But, rather than focus on the blood level of digitalis, which might be useful, or quinidine, which is totally inappropriate, this patient might have benefited from preoperative cardiac evaluation of the systolic murmur and status of left ventricular function, in light of cardiomegaly on chest x-ray. Aortic stenosis may be severe prior to the onset of symptoms. An electrocardiogram in a patient receiving digitalis may obscure signs of left ventricular hypertrophy, thus an important measure of the left ventricle's response to strain may be misinterpreted. A preoperative echocardiogram evaluating the aortic valve and overall left ventricular function might have provided more

useful information than that provided by measuring serum digitalis or quinidine concentrations.

Charles B. Hantler, M.D.
Associate Professor

David W. Learned, M.D.
Clinical Instructor

Department of Anesthesiology
The University of Texas Health Science Center
at San Antonio
7703 Floyd Curl Drive
San Antonio, Texas 78284-7838

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Electrocardiographic Changes during Cesarean Section

To the Editor:—The article by Mathew *et al.*¹ describing ST segment depression during cesarean section and delivery in otherwise healthy young women purports to show that the ST segment changes observed are not artifactual. This may be true, but the authors nonetheless should have described the frequency band width of their ambulatory monitor to assure readers that in fact American Heart Association standards for the accurate recording of electrocardiographic signals were met²; and if not, then the less stringent but still appropriate standards of Lambert *et al.*³ were. A relatively narrow band width, common with earlier ambulatory monitors, excluded important harmonics of the electrocardiographic signal such that artifactual ST segment depression occurred.⁴

As the authors admit, the origin of the ST segment depression re-

mains obscure. However, it may be helpful to realize that electrocardiographic ST segment depression can be the result of two very different electrophysiologic mechanisms.^{5,6} First, ST segment depression can be produced by a current that flows only during the ST interval, thus causing a true ST segment shift. Second, it can be produced by an injury current that flows during the entire cardiac cycle except that it is interrupted during the ST interval. This causes a shift in the electrocardiographic baseline (but not the ST segment), thereby causing an apparent (not real) ST segment shift. For the most part, apparent ST segment depression is more commonly associated with injury currents and therefore ischemia.⁶ Unfortunately, the electrocardiogram cannot distinguish between these two types of ST segment shifts. And unfortunately, too, the one technique that can—

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the magnetocardiogram^{5,6}—would have been technically impossible to use given the constraints of their study (*i.e.*, a magnetically shielded room is necessary).

The fact that no regional wall motion abnormalities were noted in Mathew *et al.*'s study supports the notion that the majority of the ST segment shifts were true ST segment shifts and therefore probably not ischemic but rather the result of unknown factors causing regional myocardial electrophysiologic differences in action potential waveforms. The major point being, however, that the ST segment shifts as described in the study probably do not reflect ischemia.

Bruce Kleinman, M.D.
Attending in Anesthesiology
Illinois Masonic Medical Center
836 Wellington Avenue
Chicago, Illinois 60657

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In Reply:—Kleinman's letter points out important concerns regarding the use of electrocardiography to diagnose myocardial ischemia. Accurate registration of the ST segment requires information from the lower frequencies, with the current American Heart Association standard for 12-lead recordings being 0.05 Hz. The ambulatory electrocardiogram recorder (Spacelabs model 90205) used in our study has a frequency response of 0.05–40 Hz. Due to the limitations of tape playback, the higher frequency response of our recorder is below the 100 Hz standard for 12-lead recordings. This should, however, affect only reproduction of the QRS peak and not affect the ST segment shift.

Kleinman discusses the magnetocardiogram as a technique that distinguishes between real and apparent ST segment shift. The magnetocardiogram is limited in that it can be affected by noncardiac currents mostly in the gastrointestinal tract and was found to be useful in only a third (4/10) of the patients studied.¹ Although we agree with Kleinman that the electrocardiographic changes observed may not be significant in the vast majority of women undergoing cesarean

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delivery, these ST changes do not all appear to be merely artifacts of parturition.

Joseph P. Mathew, M.D.
Lee A. Fleisher, M.D.
Department of Anesthesiology
Yale University School of Medicine
333 Cedar Street
P.O. Box 3333
New Haven, Connecticut 06510

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