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Heart Block after Methylmethacrylate Cementing

To the Editor:—Learned *et al.*¹ recently described an unfortunate case of progressive atrioventricular (AV) block (sinus bradycardia to second- to third-degree) during cementing for total hip arthroplasty using methylmethacrylate (MMA). Despite their excellent discussion of surgical techniques that may predispose to MMA absorption as well as nonsurgical factors that can contribute to potential reactions from MMA, the cause-and-effect relationship elaborated by the authors must be tempered by their lack of documentation of nontoxic serum levels of digitalis and quinidine in their particular patient.

The lack of preoperative serum level determinations of antiarrhythmics such as digitalis and quinidine goes well beyond academic interest in this case. Suffice it to say that a proper preoperative medical evaluation of this or any elderly patient with a prior cardiac history, hypothyroidism, a grade III/VI systolic ejection murmur (ventricular outflow obstruction?) and a resting sinus bradycardia of 45 beats/min should include not only serum antiarrhythmic levels but a thorough cardiac evaluation as well, none of which was mentioned in this report. The combination of digitalis and quinidine is especially pertinent due to the fact that either drug, alone or acting synergistically in toxic ranges, can lead to high-degree AV block. In addition, concomitant administration of digitalis and quinidine reliably produces two- to threefold increases in the plasma concentration of digitalis. Quinidine, depending upon its plasma concentration, appears to displace a percentage of digitalis from plasma proteins, thus increasing the free plasma levels of the later drug. Quinidine also decreases the renal clearance of digitalis. Since subtle cardiac signs of glycoside toxicity, such as significant sinus bradycardia or ectopy as in this case, may occur without other signs of toxicity and often precedes noncardiac toxic effects, the authors in question could not rule out digitalis toxicity by history alone.

It is certainly possible, as the authors contend, that the lethal progression of AV block occurring concomitant with MMA cementing took place in the presence of normal or even subtherapeutic antiarrhythmic levels, thus creating a potentially troublesome situation for the many similar patients who present for total joint replacement.

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In Reply:—Mirenda and Broyles are correct that, when quinidine is administered with digitalis, the possibility of digitalis toxicity should be of concern and looked for clinically *or* with serum digitalis levels.¹ They are also correct that sinus bradycardia and ectopic beats may be an early sign of digitalis intoxication. But, as they are no doubt aware, the baseline PR interval of 0.14 s is well within normal limits even when one considers the rather slow sinus rate, suggesting that the toxicity is *not* manifested on the AV node in our patient. As

But it is just as plausible to conjecture that any number of stimuli in their patient (*i.e.*, cementing, hypoxemia, hypercarbia, myocardial ischemia) may have converted glycoside "toxicity" that was asymptomatic to symptomatic (*i.e.*, high-degree block) and, worst of all, converted a myocardium that was once responsive to catecholamines and transvenous pacing to one that was not. The burden of proof fell upon the authors to rule out such toxicity prior to assuming the former scenario.

Clearly, we should not dismiss the hazards of MMA cementing as prior reports of hypotension² and second-degree block³ indicate. However, the case presented by Learned *et al.* impresses upon us the necessity of proper preoperative evaluation and preparation more so than the fear of another disastrous complication of MMA.

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Mirenda and Broyles are also no doubt aware, there is significant overlap in clinical toxicity and nontoxicity when serum levels alone are used. Not only is the timing of drawing blood for serum analysis critical, but there is tremendous patient variability in therapeutic and toxic response to this compound.^{2,3} Had a high toxic level of digitalis been measured in this case, this would support the contention of digitalis toxicity as significantly contributing to the heart block. In light of the normal PR interval, we believe this is unlikely. The

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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.

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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—