Heart Block after Methylmethacrylate Cementing

To the Editor—Learned et al. 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 digoxilis and quinidine in their particular patient.

The lack of preoperative serum level determinations of arrhythmics such as digoxilis 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 to IV systolic ejection murmur (ventricular outflow obstruction) and a resting sinus bradycardia of 45 beats/min should include not only serum arrhythmics levels but a thorough cardiac evaluation as well, none of which was mentioned in this report. The combination of digoxilis 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 digoxilis and quinidine reliably produces a two- to threefold increases in the plasma concentration of digoxilis. Quinidine, depending on its plasma concentration, appears to displace a percentage of digoxilis from plasma proteins, thus increasing the free plasma levels of the drug. Quinidine also decreases the renal clearance of digoxilis. 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 digoxilis 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 arrhythmics levels, thus creating a potentially troublesome situation for the many similar patients who present for total joint replacement.

...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 hypotension and second-degree block 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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References
1. Learned DW, Hanier CB: Lethal progression of heart block after prosthesis cementing with methylmethacrylate. ANESTHESIOLOGY 77: 1044–1046, 1992

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In Reply—Mirenda and Broyles are correct that, when quinidine is administered with digoxilis, the possibility of digoxilis toxicity should be of concern and looked for clinically or with serum digoxilis levels. They are also correct that sinus bradycardia and ectopic beats may be an early sign of digoxilis 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 manifest on the AV node in our patient. As 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. Had a high toxic level of digoxilis been measured in this case, this would support the contention of digoxilis toxicity as significantly contributing to the heart block. In light of the normal PR interval, we believe this is unlikely. The