LETTERS TO THE EDITOR

Triggering the IABP

To the Editor:

Intraaortic balloon counterpulsation (IABP) is used to decrease myocardial work while maintaining coronary arterial perfusion pressure, thus maximizing the myocardial oxygen supply/demand ratio. Timing of the intraaortic balloon deflation and inflation is critical to maximize physiologic benefits. Intraaortic balloon deflation and inflation is normally triggered by electrical activity recorded from a surface ECG, or systolic blood pressure peaks recorded from an arterial pressure transducer. A novel solution is reported for synchronizing the IABP in a patient in whom the standard triggers would not function.

A 78-year-old woman with a history of hypertension, hypercholesterolemia, severe peripheral vascular disease, and a previous myocardial infarction was transferred to this hospital following a second myocardial infarction complicated by unstable angina and congestive heart failure. Cardiac catheterization revealed a 60% stenosis of the left main coronary artery, 99% stenosis of the proximal left anterior descending coronary artery, diffuse disease of the left circumflex coronary artery, and 100% stenosis of the right coronary artery.

On the sixth hospital day she underwent an uneventful two-vessel coronary artery bypass procedure consisting of saphenous vein grafts from the aorta to the circumflex and left anterior descending coronary arteries. Bypass time was 1 hour 50 minutes and cross-clamp time was 1 hour 26 minutes.

Early on the morning of the second postoperative day she had an acute hypertensive episode followed by a sudden output of over 1 L of blood from her chest tube, and subsequent hypotension and asystole. After aggressive resuscitation efforts she was taken to the operating room under open cardiac massage and placed emergently on cardiopulmonary bypass (CPB). Examination of the heart revealed three right ventricular rents caused by cardiac massage. After these were repaired, a lateral left ventricular wall rupture was discovered in an area of the ventricle that appeared recently infarcted. This rupture was repaired with pledgets, sutures, and a pericardial patch.

Initial attempts to wean from CPB were unsuccessful. Placement of an IABP and institution of right heart bypass provided insufficient support. Therefore, partial CPB was instituted to allow the myocardium to rest and recover from the recent events. The IABP was run concurrently to maximize the supply/demand ratio in the damaged myocardium. Unfortunately, severe peripheral edema made the surface ECG voltage too low to trigger the IABP. In addition, the arterial pulse waveform was too small due to the partial CPB. Synchronous augmentation was still required so the V lead of the ECG was attached directly to the pacemaker terminal. This resulted in a large pacemaker spike, which easily triggered the IABP. After 6 hours of maintenance, the patient could not be weaned from CPB despite maximum mechanical and pharmacologic support. She died 12 hours after the initial hemorrhage.

The trigger signal required to permit proper timing of balloon inflation and deflation varies with the trigger mode. In this case, a system 90 (model P/N 0998-00-0058-x4, Datascope, Paramus, NJ) IABP was used. To trigger and time the IABP in the ECG mode, this model requires an R wave of 120 μV. In the pacemaker mode, it requires a 30 mV signal for a minimum of 0.1 ms duration, or 3 mV for a minimum of 2.0 msec duration, an AV interval of less than 250 ms, and an AV rate of less than 110 beats/min. In the pressure mode, it requires a 15 mmHg peak-to-peak pulse pressure.

A primary concern when connecting a monitor to a pacemaker lead is the risk of microshock. Fifty μA of current applied directly to the ventricle will produce fibrillation in about 0.1% of human hearts.[1] As little as 18 μA is sufficient in some situations.[2] The safety code of the International Electromechanical Commission sets 10 μA as the maximum permissible leakage current allowed through electrodes or catheters that contact the heart.[1,3] The "Merlin" Component Monitoring System (Hewlett Packard) was used. When properly functioning, this system has a patient leakage current of less than 10 μA at 110 V/60 Hz.

In spite of the poor outcome in this patient, it is hoped that this solution to the IABP triggering problem will be useful in a similar setting with a better outcome.

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"Heparin Allergy" and Cardiopulmonary Bypass

To the Editor:

Our Cardiac Anesthesia Service was recently involved in the care of a woman presenting for emergent myocardial revascularization. In addition to a number of more common medical problems, the patient had a well-documented history of clinical anaphylaxis and shock following prior exposure to heparin years earlier for the treatment of deep venous thrombosis. These reactions are extremely rare and are believed to be due to either the heparin compound itself or the preservative, chlorocresal, used in the formulation. Antibodies can be developed to each of the two types of heparin available, bovine or porcine. Antigenic testing and immunodiffusion studies may be used to define the exact allergen but these were impossible on this emergent basis.[1,2] The planned surgical procedure required full cardiopulmonary bypass, hence complete systemic anticoagulation. The newer low molecular weight heparin formulation was not available on such short notice, and, therefore, the administration of a full dose of regular heparin was required. Fortunately, the product currently in use in this institution does not contain chlorocresol as a preservative, thus eliminating this substance as a potential antigenic problem. Which type of heparin to use, bovine or porcine derived, remained an issue. After consultation with the Pharmacy Department, it was decided to use the pure beef extract because the patient's prior exposure would most likely have been to the porcine preparation. Using a derivative to which the individual has not been previously exposed may offer an advantage in such situations,[2] though this is controversial. The patient was premedicated with methylprednisolone, 125 mg, diphenhydramine, 50 mg, cimetidine, 300 mg, and an epinephrine infusion (5 μg/min) as prophylaxis prior to the administration of the bovine heparin. Then a 100 U test dose was given. No adverse response was evident and an anticoagulant dose (300 U/kg) was administered in incremental allotments. This was likewise uneventful and the rest of the procedure continued without incident.

The question as to whether this patient was truly allergic to porcine heparin or rather had experienced an anaphylactoid reaction to the preservative it was contained in could not be answered at the time she presented for emergency care. However, a very severe response had occurred upon previous exposure and efforts were made to avoid a recurrence. The patient was given a heparin formulation to which she had not been previously exposed (in this case bovine-derived heparin with a benzyl alcohol preservative), and premedication to ameliorate any reaction that might have still developed despite the antigen change. No problems were encountered. Which step was actually effective in preventing the adverse response is unknown, but a serious problem was avoided.

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Commentary to Letter by J.G. Bray

To the Editor:

Dr Bray's experience raises several issues. First, do any currently marketed formulations of heparin use chlorocresal as preservative? All those listed in the 1992 Physicians' Desk Reference (Montvale, NJ: Medical Economics Data) use benzyl alcohol. Second, would a low molecular weight heparin (LWMH) compound have been a superior choice? Not likely. Cross-reactivity is not assured. Because skin tests yield many false positives, a provocative trial similar to the one Dr Bray describes with bovine heparin would probably have occurred with LWMH. Unfortunately, the anticoagulant effect of