Lethal Progression of Heart Block after Prosthesis Cementing with Methylmethacrylate

DAVID W. LEARNED, M.D.,* CHARLES B. HANTLER, M.D.†

During insertion of a hip or knee joint prosthesis, methylmethacrylate (MMA) cement typically is used to ensure a strong bond between the bone and the prosthesis. However, hypotension has been reported[1] after use of MMA and may be related to its absorption into the systemic circulation with consequent peripheral vasodilatation.[2] Until Brown and Parmley[3] reported a second-degree atrioventricular block in a patient undergoing a prosthetic cementing, the only reported arrhythmia related to MMA use was associated with full cardiovascular collapse.[4] Brown and Parmley's patient exhibited the same second-degree heart block during two total hip replacement procedures separated by 1 month without a decrease in blood pressure.[3] On both occasions the arrhythmia was observed after the cementing of the acetabular component of a hip joint prosthesis.

The present report concerns a patient who developed a second-degree atrioventricular block after pressure application of MMA cement into the femoral medullary canal. Unfortunately, this block progressed quickly to a third-degree block for which pacing attempts failed.

CASE REPORT

A 67-yr-old, 55-kg woman with a 10-yr history of degenerative right hip disease was scheduled to have a total hip arthroplasty using MMA cement. She had been treated for hypothyroidism with sodium levothyroxine 0.1 mg daily for 30 yr. She had had hypertension for 10 yr, currently well controlled with a triamterene/hydrochlorothiazide compound. She had a history of intermittent atrial fibrillation, of indeterminate duration, treated with digoxin 0.25 mg daily and quinidine 300 mg twice per day in a timed-release form. She had a 10-yr history of hip discomfort, treated with tolmetin 400 mg four times per day and propoxyphene 32 mg once or twice per day.

The patient's lungs were clear on examination, and she had a slow heart rate with an occasional extra beat. A grade three/six systolic ejection murmur was present along the left sternal border. She was

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* Clinical Instructor, Department of Anesthesiology, University of Michigan Hospitals.

† Associate Professor, Department of Anesthesiology, University of Texas Health Science Center.

judged to be clinically euthyroid on admission. There was borderline cardiomegaly evident on the chest roentgenogram. Her electrocardiogram showed sinus bradycardia of 45 beats/min with a PR interval of 0.14 s and QRS duration of 0.08 s. Her hematology, enzyme, and electrolyte values were within normal limits. In particular, her serum potassium concentration was 4.8 mEq/l. Her digoxin concentration was not measured.

She received diphenhydramine 75 mg orally and triazolam 0.25 mg orally 90 min before surgery and received her morning dose of digoxin. She was monitored with a precordial and then an esophageal stethoscope, an automatic blood pressure cuff, a three-lead electrocardiogram with lead II displayed, and a calibrated oxygen analyzer. Anesthesia was induced with 50 μg fentanyl and thiamylal sodium 300 mg, and the trachea was intubated uneventfully after the intravenous administration of pancuronium 6 mg. She was placed in left lateral decubitus, and anesthesia was maintained with a mixture of nitrous oxide (3 l), oxygen (3 l), and 0.5–0.8% inspired concentration isoflurane using controlled ventilation.

For the first 2 h after induction, the patient's vital signs were remarkably stable. Her pulse rate ranged from 50 to 65 beats/min, and her systolic/diastolic pressure was easily maintained in the 120/80 mmHg range. Approximately 2 h after the beginning of surgery, freshly prepared MMA cement was injected under pressure into the prepared femoral medullary canal. The patient immediately developed a bradycardia. There was 10-mmHg decrease in systolic blood pressure preceding the bradycardia that followed cementing of the acetabulum. The electrocardioscope showed a rapidly developing second-degree atrioventricular block, which progressed within 1 min to a third-degree block with a unifocal idioventricular rhythm. The patient's blood pressure was 100/60 mmHg during the second-degree block, but her systolic pressure decreased to 40 mmHg with the complete heart block.

The patient was turned supine, and cardiopulmonary resuscitation was started. Two 0.6-mg doses of atropine administered before cardiopulmonary resuscitation were totally ineffective in changing the block, as were third and fourth doses administered after cardiopulmonary resuscitation had been initiated. While we inserted a pacing catheter (Edwards Pacing TD #93-208-7F), infusions of isoproterenol and epinephrine of up to 32 μg/min each were administered during the resuscitation attempt. The idioventricular rhythm, now with no P-wave, remained unifocal and increased to 80 beats/min, and the blood pressure increased to 110/60 mmHg.

When the infusion of isoproterenol was decreased and the infusion of epinephrine was slowed to approximately 16 μg/min, both heart rate and blood pressure decreased to a slow idioventricular rhythm with no palpable pressure. We were unable to obtain ventricular capture with maximal current while advancing and pulling a pacing catheter through the right ventricle. Another course of closed-chest cardiac massage with an increased dose of epinephrine failed to reverse the trend, and the patient was pronounced dead. External cardiac pacing was not available. Resuscitation was discontinued after approximately 45 min. The patient's husband refused permission for *post mortem* examination.

DISCUSSION

This is the second report of a patient in whom an atrioventricular block developed during the cementing of a

prosthesis with MMA. Several factors may have aggravated this patient's response to such a marked extent. Her femoral medullary canal was prepared elaborately with a plastic brush and Water Pik. This preparation may have maximized the number of channels available for absorption of the monomer MMA. The polymerizing cement was introduced into the area of absorption early after mixing, the objective being more complete penetration of the bone with the cement. This could have resulted in the availability of more monomer for absorption for a given quantity of cement introduced. A pressure injector was used. Adding pressure to the process could have further increased the amount of monomer and MMA absorbed. The patient probably had a therapeutic level of digoxin, but unfortunately, the patient's preoperative serum digoxin level had not been measured. Conceivably, the arrhythmia observed preoperatively and believed to be premature ventricular contractions could have been a sign of digitalis toxicity, but the patient's concurrent use of quinidine led us to believe that the premature ventricular contractions occurred on a more chronic basis. The hazard of treating arrhythmias with quinidine in the presence of atrioventricular block is known.[5] Perhaps the development of an atrioventricular block from another cause in the presence of quinidine is hazardous.

Heart block after MMA application, however, has not been a common finding clinically or in animal preparations. Willis et al.,[6] using isolated rat atrial tissue, have shown large (as great as 10–100-fold) decreases in chronotropic responses to isoproterenol after exposure to MMA. The decreased sensitivity to isoproterenol was greater during conditions of hypoxia. Svartling et al.[7] described a patient who developed severe ventricular extrasystoles during total knee replacement and who had the highest level of MMA in the group of patients studied; they suggested that MMA was the causative factor for the ventricular dysrhythmias.

Several authors have indicated that it is not release of MMA that induces the hypotension or hypoxemia after cement application. Rather, it may be mechanical plugging of the pulmonary arteries with or without release of vasoactive substances from the pulmonary artery that leads to the cardiorespiratory side effects of cementing the joint with MMA.[8,9,10] Both fat and air emboli have been implicated as the causative agents, and, in fact, a similar decrease in blood pressure and hemoglobin oxygen saturation have been demonstrated when bone wax is used in place of MMA.[10]

Other possible factors contributing to the second- and third-degree heart block occurring in the current case include toxicity from either digitalis or quinidine, myocardial infarction/ischemia, and hypoxemia with resultant atrioventricular nodal ischemia, and perhaps clinical or subclinical hypothyroidism. Although our patient had a systolic ejection murmur, there was no clinical evidence of significant aortic stenosis. There was no left ventricular hypertrophy on the electrocardiogram at the time of admission and no documentation of radiation of the murmur or delayed and diminished carotid pulses. The mechanism of syncope in patients with aortic stenosis remains unknown but might include bradyarrhythmias including advanced heart block.

Like the case described by Brown and Parmley,[3] our case documents the temporal association of the appearance of a second-degree atrioventricular block after the placement of MMA cement. Our case demonstrates also the legitimacy of concern should a second-degree block appear as a new finding in a patient. Rapid, aggressive therapeutic measures should be directed at maintaining either intrinsic, or, if needed, extrinsic cardiac pacing. The current case also raises some questions about the technique of MMA cementing in prosthetic repairs. Does pressurization of MMA into a well-prepared medullary canal significantly increase the risk of an atrioventricular block in the patient? Does the presence of digoxin and/or quinidine potentiate the risk? Should the surgeon consider an alternative system of prosthetic repair in a patient taking these drugs or taking other drugs capable of blocking atrioventricular conduction? If cement is necessary, should the anesthesiologist require substitution of adequate alternative forms of cardiac therapy before undertaking an anesthetic in a patient taking a drug or drugs capable of blocking atrioventricular conduction?

We believe that this case strengthens Brown and Parmley's[3] conjecture resulting from their observation of the development of atrioventricular block in a patient undergoing MMA cementing. They believed that a similar occurrence in a patient with significant cardiac disease might be of greater clinical significance. It may be that any patient who is taking any medication whose actions or side effects include inhibition of atrioventricular conduction is at increased risk for a serious or even fatal heart block when MMA cement is used. If an alternative surgical approach cannot be found for these patients, then perhaps a change in medication before surgery should be considered or a method of ventricular pacing (invasive or transcutaneous) be guaranteed before application of MMA. Presumably, a patient not taking his or her usual medication for the purpose of a safer prosthetic repair could begin taking them again immediately postoperatively.

REFERENCES

1. Schuh FT, Schuh SM, Viguera MG, Terry RN: Circulatory changes following implantation of methyl methacrylate bone cement. ANESTHESIOLOGY 39:455–457, 1973
2. Peebles DJ, Ellis RH, Stride SDK, Simpson BRJ: Cardiovascular effects of methyl methacrylate cement. Br Med J 1:349–351, 1972
3. Brown DL, Parmley CL: Second-degree atrioventricular block after methyl methacrylate. ANESTHESIOLOGY 56:391–392, 1982

4. Powell JN, McGrath PJ, Lahiri SK, Hill P: Cardiac arrest associated with bone cement. Br Med J 3:326, 1970
5. Bigger JT Jr, Hoffman BF: Antiarrhythmic drugs, The Pharmacological Basis of Therapeutics. Edited by Gilman AG, Goodman LS, Rall TW, Murad F. New York, Macmillan Publishing Company, 1985, p 758
6. Willis DW, Campbell MJ, Ravis WR, Hamrick ME, Born CK: Inhibition of isoproterenol positive atrial chronotropic effect by methylmethacrylate polymer and monomer. Res Commun Chem Pathol Pharmacol 53:117–120, 1986
7. Svartling N, Pfaffli P, Tarkkanen L: Blood levels and half-life of

methylmethacrylate after tourniquet release during knee arthroplasty. Arch Orthop Trauma Surg 105:36–39, 1986
8. Schlag G: Mechanisms of cardiopulmonary disturbances during total hip replacement. Acta Orthopaedica Belgica 54:6–11, 1988
9. Svartling N: Detection of embolized material in the right atrium during cementation in hip arthroplasty. Acta Anaesthesiol Scand 32:203–208, 1988
10. Orsini EC, Byrick RJ, Mullen BM, Kay JC, Waddell JP: Cardiopulmonary function and pulmonary microemboli during arthroplasty using cemented or non-cemented components. J Bone Joint Surg [Am] 69:822–832, 1987

Unconsciousness and Apnea Complicating Parascalene Brachial Plexus Block: Possible Subarachnoid Block

ANIS BARAKA, F.R.C.A.(HON.),* MYRNA HANNA, M.D.,† ROLA HAMMOUD, M.D.‡

The occurrence of subarachnoid block following brachial plexus block performed above the clavicle is a rare complication.[1] Only two cases of total spinal anesthesia following interscalene block have been reported in the literature.[2,3] The following describes a patient who developed signs and symptoms suggestive of inadvertent subarachnoid block as a complication of parascalene brachial plexus block. The complication was attributed to intraneural injection of the local anesthetic, with a subsequent centripetal spread to the subarachnoid space.

CASE REPORT

A 19-yr-old woman, weighing 52 kg, was scheduled for excision of a ganglion at the right wrist. The patient received no premedication. In the operating room, a right brachial plexus block was done using the parascalene approach.[4,5] The patient was placed in the dorsal recumbent position, with the head turned to the left side. A parascalene block was done using a 22-G needle attached by an extension tubing to a syringe filled with 15 ml 2% lidocaine. The site of puncture was the junction of the lower third and upper two-thirds of a line drawn from the midpoint of the clavicle to the C6 transverse process. The needle was inserted at 90° to the skin and was advanced in the anteroposterior plane until the patient felt paresthesia spreading to the

arm and hand. Aspiration did not show any blood or cerebrospinal fluid. Using the "immobile needle" technique of Winnie,[6] the needle was fixed in position and the local anesthetic injected. During the injection of the first 5 ml, marked resistance was encountered and agonizing pain was felt by the patient. Further injection was stopped, and the needle was withdrawn about 2 mm. When the resistance and pain decreased, an additional 10 ml lidocaine was injected.

Immediately following injection of lidocaine, the patient could not move the right arm but could move her left arm. Five minutes later, it was noted that the patient became unconscious, stopped breathing, and the arterial blood pressure decreased from 120/80 mmHg to 80/50 mmHg. The lungs were ventilated via a face mask using 100% oxygen. Also, 100 μg phenylephrine and 1 L lactated Ringer's solution was infused to increase the blood pressure. Laryngoscopy showed completely relaxed masseter muscles and immobile vocal cords. The trachea was readily intubated with no reflex bucking or coughing, and the lungs were ventilated with 100% oxygen. After 10 min, spontaneous breathing resumed. By the end of surgery, which lasted 45 min, the patient was awake, breathing adequately, and the trachea was extubated. Pinprick sensation was blunted bilaterally from C7 to T5. Complete recovery of pinprick sensations and motor activity was observed after about 1 h, with no neurologic sequelae.

DISCUSSION

Hypoventilation following supraclavicular brachial plexus block may result from phrenic nerve block or the occurrence of pneumothorax. However, the appearance of the objective signs of pneumothorax is almost always delayed for 2–6 h. Also, unilateral phrenic nerve block is usually inconsequential in the healthy patient with little or no respiratory disease.[1]

Our patient rapidly lost consciousness and developed apnea. The complication may be secondary to inadvertent intravascular injection of the local anesthetic or to an overdose. An overdose usually results in initial central nervous system stimulation manifesting as facial twitches

* Professor and Chairman.
† Chief Resident.
‡ Resident.