CASE REPORTS

Anesthesiology
1996; 85:675-7

Third-degree Heart Block Complicating Supraclavicular Brachial Plexus Block

Anil Vyas, M.D., Frank M. O'Connell, M.D.,† Charles A. Vacanti, M.D.‡*

REGIONAL anesthesia is frequently administered to elderly patients and those with known cardiovascular disease in the hope of minimizing the cardiovascular complications associated with general anesthesia. Drug interactions between local anesthetics used in regional techniques and calcium channel blockers have been described.[1,2] To date, however, potentially life-threatening dysrhythmias associated with brachial plexus blockade using the supraclavicular approach have not been reported. We now describe such a case.

Case Report

A 68-yr-old woman who weighed 62 kg was admitted for open reduction and internal fixation of a supracondylar fracture of the left humerus that she sustained secondary to a fall. The patient denied any loss of consciousness associated with the fall and had no history of syncope or near syncope. The patient had been receiving 360 mg sustained-release verapamil daily, orally, for hypertension. Her blood pressure was 132/72 mmHg, her heart rate was 50 beats per minute (bpm) and regular, her respiratory rate was 16 breaths/min, and she was afebrile. A preoperative electrocardiogram revealed incomplete right bundle branch block with a PR interval of 0.2 s, a QRS duration of 0.09 s, and a QT interval of 0.38 s, with a ventricular rate of 88 bpm (fig. 1). Laboratory investigations included a normal complete blood count and electrolytes, with a serum potassium of 3.9 meq/l.

On the day of surgery, the patient received her daily dose of sustained-release verapamil, approximately 5 h before repair of her humerus. On arrival to the operating room, an electrocardiogram, blood pressure cuff, and pulse oximeter were applied and monitored continuously. Preoperative blood pressure was 122/65 mmHg. Sinus bradycardia, with a heart rate of 55 bpm, was observed on the electrocardiogram. After intravenous administration of 1.25 mg midazolam and 50 μg fentanyl, a left supraclavicular brachial plexus block was performed. Using a peripheral nerve stimulator (Stimuplex 016 Model, STIM—200, B. Braun Medical, Bethlehem, PA), a 20-g needle was inserted into the interscalene groove one inch above the clavicle and advanced in a caudad direction at a 10-20° angle with the skin. Muscle twitches observed in succession during application of a one milliampere current using the nerve stimulator confirmed needle placement. A test dose of 2 ml of a 45 ml solution that contained 60 mg bupivacaine and 495 mg mepivacaine abolished the muscle twitches at 0.2 milliamperes. The remaining solution was slowly injected with intermittent aspiration after every 5 ml injected. This produced a dense sensory and motor blockade without any immediate complication. Horner's syndrome did not develop after the block was performed. Fifteen minutes after local anesthetic injection, the patient experienced first degree heart block, followed at 30 min by type one, second degree heart block. Her blood pressure and heart rate remained within normal limits throughout the operative procedure. The patient did not complain of lightheadedness, nausea, vomiting, or shortness of breath during this time. The surgical procedure, lasting 70 min, was performed distal to a tourniquet inflated to 275 mmHg. At the conclusion of the procedure, the tourniquet was deflated and the patient was transferred to the recovery room with a stable blood pressure and type one, second degree heart block.

Five hours after the initial administration of the supraclavicular block, while still in the recovery room, the patient experienced third degree heart block, with a ventricular rate between 50 and 70 bpm and a blood pressure ranging between 124/74 and 147/89 mmHg (fig. 2). The patient was transferred to the intensive care unit for cardiovascular monitoring. During the entire perioperative period, the patient's oxygen saturation remained stable at 96-98%. The patient received no narcotics or sedation before the onset of the third degree heart block. External pacemaker electrodes were placed on the patient as a precaution but were not used. The patient's rhythm returned to sinus with incomplete right bundle branch block approximately 10 h after the brachial plexus block (fig. 3). Serum isoenzyme concentrations for creatine phosphokinase of muscle band were 0%. An electrocardiogram taken 1 day later was within normal limits. The patient subsequently had an uneventful recovery and was discharged home without Holter monitoring or electrophysiologic testing.

* Anesthesiologist, Department of Anesthesiology, Marlboro Hospital, Marlboro, Massachusetts.

† Assistant Professor of Anesthesiology, Department of Anesthesiology, University of Massachusetts Medical Center, Worcester, Massachusetts.

‡ Professor and Chairman of Anesthesiology, Department of Anesthesiology, University of Massachusetts Medical Center, Worcester, Massachusetts.

Received from the Department of Anesthesiology, Marlboro Hospital, Marlboro, Massachusetts, and the Department of Anesthesiology, University of Massachusetts Medical Center, Worcester, Massachusetts. Submitted for publication February 12, 1996. Accepted for publication May 15, 1996.

Address reprint requests to Dr. Vyas: Department of Anesthesiology, Marlboro Hospital, 57 Union Street, Marlboro, Massachusetts 01752.

Key words: Pharmacology: calcium channel blockers. Heart: heart block. Anesthesia: regional. Supraclavicular Brachial Plexus Block, Verapamil: interactions (drug).

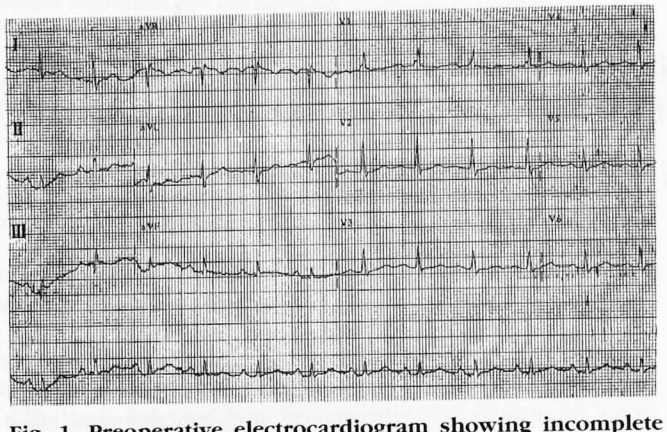

Fig. 1. Preoperative electrocardiogram showing incomplete right bundle branch block with sinus rhythm.

Discussion

We believe that the development of third degree heart block was due to an interaction between the sustained release form of oral verapamil and the two local anesthetics mepivacaine and bupivacaine. Several possible mechanisms may have been involved, as follows. Plasma concentrations of verapamil may have been increased because of the displacement of protein-bound verapamil by bupivacaine, and perhaps mepivacaine, as a result of vascular uptake after the block. Verapamil, normally more than 90% bound to plasma proteins, may be displaced by coadministration of local anesthetics such as lidocaine.[2,3] A toxic concentration of verapamil achieved with enteral administration is manifested as atrioventricular block before clinically significant cardiovascular depression in patients with normal ventricular function.[4] Coadministration of either lidocaine or bupivacaine in a conscious, chronically instrumented canine model receiving verapamil infusion was shown to produce second degree atrioventricular block.[1]

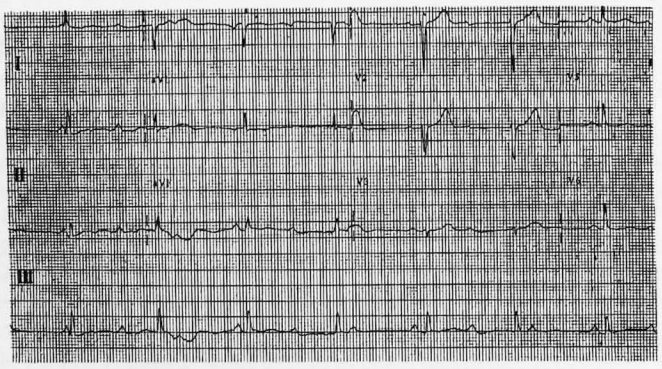

Fig. 2. Immediate postoperative electrocardiogram showing complete atrioventricular dissociation due to third degree heart block.

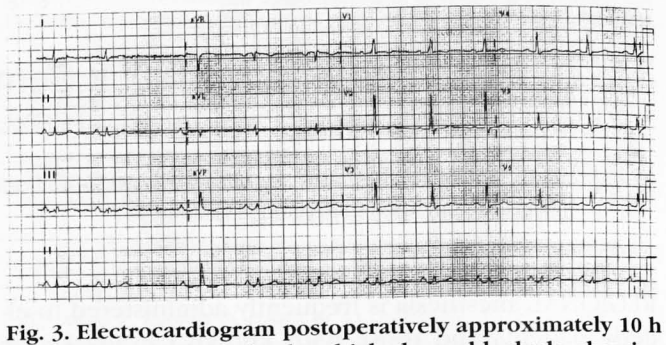

Fig. 3. Electrocardiogram postoperatively approximately 10 h after the supraclavicular brachial plexus blockade showing return of sinus rhythm with incomplete right bundle branch block.

Another potential factor is that chronic use of oral verapamil may lead to a high plasma concentration of norverapamil, a metabolite of verapamil, which has been shown to increase free verapamil plasma concentrations by as much as 30%.[2] This situation may have made the patient more vulnerable to the effects of displacement of protein-bound verapamil by local anesthetic agents. In addition, chronic oral administration of sustained-release verapamil has a time to peak concentration in healthy volunteers of approximately 8 h, and the serum concentration achieved in fasting subjects is approximately twice that seen in nonfasting patients given the same dose orally.§ The pharmacokinetics mentioned earlier would approximate the onset of third degree heart block seen in our patient.

It is also possible that this patient experienced an unintended stellate ganglion block, which has an incidence of approximately 83% when using a volume greater than 40 ml in a supraclavicular approach to brachial plexus blockade.[5] Stellate ganglion blockade has been associated with loss of cardioaccelerator activity, which may result in bradyarrhythmias and hypotension.[6] Variations in endogenous sympathetic tone may alter the dose-response relation such that patients with an increased catecholamine concentration may be less sensitive to the dromotropic effects of verapamil.[7] In an experimental model, the depressant effects of verapamil on the atrioventricular node were

§ Physicians' Desk Reference. Montvale, NJ, Medical Economics Data Production, 1994.

antagonized by beta adrenergic agonists.[8] Conversely, a decrease in sympathetic tone may render a patient more sensitive to the dromotropic effects of verapamil.

Finally, the effect of the tourniquet on tissue binding of local anesthetics may have influenced the development of third degree atrioventricular block in this patient. In general, the more hydrophobic agents (bupivacaine) are bound to a greater extent to serum proteins than are their more hydrophilic counterparts (lidocaine).[9] The proportion of local anesthetic in the protonated, cationic, unbound form is influenced by both its pKa and the pH of the tissue milieu to which it is exposed. Therefore, one could postulate a greater amount of unbound fraction of mepivacaine and bupivacaine being released from the site of injection once the tourniquet was released and acidotic blood from the upper extremity returned to the central circulation.

Because the patient's QRS complexes remained narrow during the episode of heart block, and previous investigators demonstrated a widening of the QRS complex with intravenous bupivacaine infusion in a dog model, it is unlikely for verapamil to have displaced bupivacaine as an explanation for the observed third-degree heart block.[10] In this same model, coadministration of both verapamil and bupivacaine did not produce an increase in serum levels of bupivacaine from those observed by the infusion of bupivacaine alone.

Treatment for patients with signs of severe toxicity due to verapamil include decontamination of the gut with activated charcoal and intravenous administration of calcium salts, glucagon, atropine, or epinephrine in patients refractory to treatment with calcium.[11]

In summary, we report a case of transient third degree heart block that occurred after a supraclavicular approach to brachial plexus anesthesia in a patient receiving a sustained-release enteral form of verapamil. We emphasize that interactions between local anesthetic agents administered to patients taking calcium entry blockers should be considered when performing regional anesthetic technique.

References

1. Edouard AR, Berdeaux A, Ahmad R, Samii K: Cardiovascular interactions of local anesthetics and calcium entry blockers in conscious dogs. Reg Anesth 1991; 16:95–100

2. Yong CL, Kunka RL, Bates TR: Factors affecting the plasma protein binding of verapamil and norverapamil in man. Res Commun Chem Pathol Pharmacol 1980; 30:329–39

3. Schomerus M, Spiegelhalder B, Stieren B, Eichelbaum M: Physiological disposition of verapamil in man. Cardiovasc Res 1976; 10:605–12

4. Mangiardi LM, Hariman RJ, McAllister RG Jr, Bhargava V, Surawicz B, Shabetai R: Electrophysiologic and hemodynamic effects of verapamil. Correlation with plasma drug concentrations. Circulation 1978; 57:366–72

5. Moore DC, Bridenbaugh LD, Eather KF: Blocks of the upper extremity. Supraclavicular approach vs axillary approach. Arch Surg 1965; 90:68–72

6. Lofstrom J, Cousins MJ: Sympathetic neural blockade of upper and lower extremity, Neural Blockade in Clinical Anesthesia and Management of Pain. 2nd edition. Edited by Cousins MJ, Bridenbaugh PO. Philadelphia, JB Lippincott, 1988, pp 461–500

7. Dominic J, McAllister RG Jr, Kuo CS, Reddy CP, Surawicz B: Verapamil plasma levels and ventricular rate response in patients with atrial fibrillation and flutter. Clin Pharmacol Ther 1979; 26:710–4

8. Zipes DP, Fischer JC: Effects of agents which inhibit the slow channel on sinus node automaticity and atrioventricular conduction in the dog. Circ Res 1974; 34:184–92

9. Strichartz GR, Covino BG: Local anesthetics, Anesthesia. 3rd edition. Edited by Miller RD. New York, Churchill Livingston, 1990, pp 437–70

10. Timour Q, Freysz M, Couzon P, Loufoua J, Bertrix L, Gerentes I, Faucon G: Possible role of drug interactions in bupivacaine-induced problems related to intraventricular conduction disorders. Reg Anesth 1990; 15:180–5

11. Smilkstein M: Common cardiac medicines, Emergency Medicine Concepts and Clinical Practice. 3rd edition. Edited by Rosen P, Barkin R. St. Louis, MO, Mosby Year Book, 1992, pp 2541–51