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Bronchospasm Following Interscalene Brachial Plexus Block

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Complications from an interscalene brachial plexus nerve block¹ range from Horner's syndrome, hoarseness,² carotid bruit,³ to more serious complications, such as subarachnoid, epidural, or phrenic nerve blocks.⁴⁻⁶ Permanent neurologic damage and cardiac arrest also have been reported.^{7,8} We observed two cases of bronchospasm that occurred in nonasthmatic patients immediately following the use of this technique.

REPORT OF TWO CASES

Patient 1: A 43-year-old, 62-kg man with a tentative diagnosis of sarcoidosis was scheduled for a diagnostic right axillary lymph node biopsy. History revealed a progressive shortness of breath during mild exertion, but no history of bronchial asthma was evident. On physical examination he had generalized lymphadenopathy. Lungs were clear to auscultation. Chest roentgenogram revealed bilateral mid and lower zonal infiltrates and extensive pleural disease. The pulmonary function studies indicated moderate restrictive and obstructive airway disease.

After receiving diazepam, 5 mg iv, an interscalene brachial plexus block was performed. He was placed in a 20-degree head-up position. After identification of the interscalene groove with a #25 gauge 1.5-inch needle, a paresthesia (midarm area) was elicited with the first attempt. Twenty milliliters 1.5% lidocaine and 15 ml 0.5% bupivacaine

were injected. Within the next 10 min, analgesia of the operative site resulted. His vital signs remained stable, and after the operative site was draped, surgery began. At this time the patient experienced difficulty in breathing and was using the accessory muscles of respiration. Surgery was stopped. A right Horner's syndrome was evident, and the extent of the nerve block was limited to the right side. On auscultation of the chest bilateral expiratory wheezing was heard. The chest roentgenogram was negative for pneumothorax, and the diaphragm was not elevated. Arterial BP and HR were stable. Aminophylline 300 mg in 100 ml 5% dextrose was given iv with diminution of the expiratory wheezing. The diagnosis at that time remained elusive and, since the drapes covered the chest and airway, the trachea was intubated following the administration of thiopental 150 mg and succinylcholine 60 mg iv. Anesthesia subsequently was maintained with halothane and oxygen. pH_a was 7.36, Pa_{O_2} 212 mm/Hg, Pa_{CO_2} 44 mm/Hg. Forty minutes after completion of surgery, the patient was awake and breathing without evidence of respiratory distress; his lungs were clear. pH_a was 7.46, Pa_{O_2} 67 mm/Hg, Pa_{CO_2} 37 mm/Hg, with an FI_{O_2} of 0.24. Recovery from sensory and motor paralysis occurred gradually over the next 2 h with no sequelae.

Patient 2: A 52-year-old nonasthmatic male, with an unremarkable medical history, presented for repair of a severed tendon of his right hand. Anesthesia for the tendon repair was achieved with an interscalene brachial plexus block performed as noted above for the first patient. Within 10 min of the onset of anesthesia the patient complained of difficulty in breathing, although respiratory distress was not noted clinically; a right-sided Horner's syndrome was evident, and the analgesia was limited to the right arm. On auscultation, however, mild expiratory wheezing was heard. The operation was postponed. The chest roentgenogram was negative for pneumothorax or elevation of the diaphragm and the bronchospasm resolved without treatment.

DISCUSSION

Several factors may cause respiratory distress following an interscalene nerve block. Examples are the occurrence of unilateral and bilateral phrenic nerve paralysis, pneumothorax, or bronchial spasm. The local anesthetics when injected into the sheath of the brachial plexus not

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only spread to the ipsilateral phrenic nerve⁹ but also spread across the midline to the contralateral nerve,¹⁰⁻¹² paralyzing the diaphragm and causing respiratory distress. Severe respiratory distress or even respiratory arrest may occur if the local anesthetic inadvertently is injected into the cervical subarachnoid or epidural space.^{4-6,8} In our patients the anesthetic block was limited to the intended arm, the chest roentgenogram was normal, and the arterial blood pressure remained stable. Therefore, the above causes for the respiratory distress can be excluded. A pneumothorax produced by a #25 needle was unlikely; there were no findings demonstrable in the chest roentgenogram.

Bronchospasm has been described in an asthmatic patient following an interscalene brachial plexus block.¹³ Our two patients have denied previous overt bronchospastic disorders, and therefore these case histories underscore the possibility of bronchospasm occurring even in nonasthmatic patients.

Emotional and psychologic factors may cause bronchospasm via the efferent pathway of vagus.¹⁴ Our patients, however, were not visibly upset and appeared calm and emotionally stable. The operative site was tested and found to be analgesic, and therefore pain was not the cause of the bronchospasm. The bronchospasm was not related to an anaphylactic reaction, because there was no history of allergy, erythema, macularpapular rash, edema, or hemodynamic instability. Furthermore, the amide local anesthetics used are most unlikely to cause allergic reactions. Recurrent laryngeal nerve paralysis following the interscalene block² can lead to upper airway obstruction and wheezing. Our patients did not have inspiratory wheezing, stridor, or hoarseness (the cardinal signs of upper airway obstruction) and responded to aminophylline therapy.

The sympathetic nerve supply to the bronchi arises from the T1-T4 segments of the sympathetic chain, while the dominant parasympathetic supply is derived from the vagus nerve.¹⁵ The integrity of normal airway tone is the balance between the sympathetic (beta) bronchodilators and the parasympathetic and the alpha sympathetic constrictors. The balance may be tipped in the direction of either bronchodilatation (*e.g.*, by atropine) or bronchoconstriction by pharmacologic agents (*e.g.*, acetylcholine, neostigmine, histamine, serotonin) and by sympathetic blockade. The embryologic origin of the lung is from the foregut, and the innervation of the airway is similar to the gastrointestinal tract.¹⁶ Sympathetic blockade associated with the spinal or an epidural anesthesia produces constriction of the bowel due to the unopposed parasympathetic vagal action.¹⁷ Therefore, blocking the sympathetic nerve supply to the bronchi (T1-T4) should cause bronchoconstriction due to the unopposed parasympathetic vagal action. The local an-

esthetics, when injected in the interscalene groove, have been shown to spread to the opposite side if the midline septa are deficient and also may spread upward and downward as far as T4 segments.¹²

Why bronchospasm has not been observed frequently when T1-T4 sympathetic segments are blocked with a high or a total spinal is not clear. However, these patients are frequently either premedicated with atropine or perhaps treated with atropine or ephedrine for the bradycardia or hypotension that may occur with high or total spinal. Both atropine and ephedrine are also bronchodilators, and, therefore, bronchospasm may not occur in these pretreated patients. Interestingly, neither our patient or those who developed bronchospasm following a spinal,¹⁸ epidural,¹⁹ or an interscalene brachial plexus block,¹³ received atropine premedication. The volume of local anesthetic used for stellate ganglion block may not be sufficient to block all the T1-T4 sympathetic segments and cause bronchospasm.

Perhaps in our patients who did not receive atropine, the large volume of the local anesthetic combined with the use of the reverse Trendelenberg position could have caused a blockade of the sympathetic bronchodilator fibers with a resulting acute transient bronchospastic episode.

When patients with compromised respiratory function present for surgery of the arm, the tendency (as we did) may be to select a regional anesthetic technique. These case histories underscore the possibility of diminishing an already compromised respiratory function by bronchospasm. This seems a rare but conceivable complication of this technique. Furthermore, since the treatment of respiratory distress that follows interscalene brachial plexus block differs, depending upon its etiology, it is important to identify the reason for the respiratory distress by auscultation of the chest, observation of the vital signs, assessment of the extent of the neural block, and evaluation of the chest roentgenogram.

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Management of the Patient with Protamine Hypersensitivity for Cardiac Surgery

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Patients may present for cardiac surgery with a history of protamine hypersensitivity occurring during cardiac catheterization or previous cardiac surgery.¹⁻¹⁰ Over a recent 24-month period, four patients of the 1,452 requiring open-heart surgery at our hospital presented with a history of protamine hypersensitivity (incidence 0.28%). This report describes the first two cases and discusses the evaluation of these patients and management options during cardiac surgery.

REPORT OF TWO CASES

Patient 1: A 42-year-old, 58-kg woman with unstable angina pectoris and an uncomplicated myocardial infarction 7 months previously was admitted for cardiac catheterization. She had no history of asthma, food or drug allergies, or other atopic disorders. Premedication for cardiac catheterization was im meperidine, scopolamine, diphenhydramine, and cefazolin. She received heparin, 5,000 units, iv, and procaine local anesthesia at the time of catheterization. Coronary arteriography, performed using radioopaque contrast media (Hypaque-76®, Winthrop) without incident, demonstrated two-vessel coronary artery disease, and normal left ventricular function. At the

conclusion of the study, the administration of protamine sulfate, 50 mg, iv, was immediately followed by hypotension (systolic blood pressure 50 mmHg), dyspnea, urticaria, and pruritis. Resuscitation was accomplished within minutes with oxygen, epinephrine, iv fluids, diphenhydramine, and hydrocortisone.

The patient was readmitted 6 weeks later for double aortocoronary bypass surgery. Preoperative hematologic and coagulation values were normal. Intradermal testing to 100 µg of intradermal protamine sulfate was negative. The plan for management was avoidance of protamine administration and omission of heparin neutralization.

Prior to the institution of cardiopulmonary bypass (CPB), heparin was titrated to bring the activated clotting time (ACT) over 400 s. Twenty-five thousand units (4,300 units/kg) of heparin were administered in divided doses, and an ACT of 670 s achieved. No further heparin was administered, and after 94 min of hypothermic CPB the ACT was 386 s. The patient was weaned from CPB, and surgical hemostasis was accomplished using electrocautery and thrombin-soaked Gelfoam® (Upjohn Co). During chest closure, the oxygenator contents were washed and concentrated in a Dideco model BT 795 Autotrans® cell washer and 480 ml of autologous erythrocytes reinfused. In addition, 400 ml of lactated Ringer's solution, 500 ml of homologous packed erythrocytes, and 355 ml of fresh-frozen plasma were administered in the operating room. At the conclusion of the operative procedure the ACT was 366 s.

In the intensive care unit, the prothrombin time was 17.2 s, partial thromboplastin time greater than 150 s, platelet count 124,000/mm³, and hemoglobin 9.2 g/dl. Mediastinal tube drainage was approximately 650 ml/h during the first 6 h. The drainage was collected, washed, and concentrated for reinfusion. Additional blood volume replacement was accomplished using homologous packed erythrocytes, fresh-frozen plasma, platelet concentrates, and colloid solutions. Calcium chloride was administered intermittently (total dose, 3 g) to maintain a normal blood ionized Ca⁺⁺ level. Mediastinal drainage slowed to 150 ml/hr after the initial 6 postoperative hours and to 25 ml/h 13 h after surgery. At that time, chest tube drainage was 4,985 ml, and replacement consisted of 2,000 ml autologous erythrocytes, 1,750 ml homologous packed erythrocytes, 1,500 ml fresh-frozen plasma, 450 ml platelet concentrates, and 250 ml 5%

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