Removal of Broken Fragment of Biopsy Forceps with Magnetic Extractor

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Transbronchial lung biopsy performed on a 51-year-old woman for the evaluation of nodular densities with cavities in the bilateral apical areas was complicated by the breakage of the biopsy forceps. The broken fragment of the forceps was successfully retrieved by use of the magnetic extractor through a fiberoptic bronchoscope under fluoroscopic guidance.

(Chest 1989; 95:700-01)

Flexible fiberoptic bronchoscopy with transbronchial lung biopsy is widely performed for the diagnosis of a variety of pulmonary diseases.1 As a rare complication of the biopsy procedure, the breakage of the biopsy forceps has been reported.2,3 We describe herein the first case, to our knowledge, in which the broken piece of the biopsy forceps was successfully retrieved by use of the magnetic extractor through the fiberoptic bronchoscope.

CASE REPORT

A 51-year-old woman with a 30 pack-year history of smoking was admitted for the evaluation and treatment of nodular densities with cavities in the bilateral apical areas of the lungs. Sputum smear was negative for acid-fast bacilli. A tuberculin skin test was positive. Antituberculosis chemotherapy (isoniazid, 0.4 g; rifampin, 0.45 g; and ethambutol, 1.0 g) had been empirically begun in the outpatient clinic four weeks before admission. Since the sputum specimen did not grow acid-fast bacilli, transbronchial lung biopsy was performed by use of a fiberoptic bronchoscope (Olympus BF-B3R). Three biopsy specimens were obtained from the right apical segment. However, the biopsy result was nongenotypic, and she underwent the second fiberoptic bronchoscopy one week later. At this time, the alligator biopsy forceps (Olympus FB-15C) was used. After five biopsy specimens were smoothly obtained from the left apical posterior segment, the breakage of the biopsy forceps was noticed, and under fluoroscopy the broken piece of the forceps was found to be lodged in the peripheral, small bronchus (Fig 1). Multiple attempts to retrieve this broken piece by use of another biopsy forceps and a curet (Olympus CC-3C) were unsuccessful. The patient was placed under observation. One week later, the magnetic extractor (Olympus 1E-SP) (Fig 2) was available. During the period of observation, the patient suffered from no episode of fever, cough, or hemoptysis.

After topical anesthesia with 2 percent lidocaine was given, the bronchoscope (Olympus BF-B3R) was passed transorally. The bronchus of the left apical posterior segment was free of erythema, discharge, and bleeding. The magnetic extractor was passed into the involved bronchus through the inner channel of the bronchoscope. Under fluoroscopic guidance, the magnet-tipped extractor was advanced further and was successfully attached to the fragment of the forceps, which was removed without difficulty. The biopsy specimens obtained during the second procedure were compatible with tuberculosis, and the patient was discharged with further

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Metal fatigue and an intrinsic metal defect were suggested as the cause of the breakage of the forceps. The real cause of the breakage remains to be determined. The breakage of the grasping forceps and the wire cytology brush was also reported. We recommend that the magnetic extractor be kept on hand at any institution where bronchoscopic examination is performed. It is also useful to retrieve other metallic foreign bodies.

**References**


**Management of Life-Threatening Bradycardia in Spinal Cord Injury**

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A 19-year-old man with SCI at C5 suffered recurrent life-threatening bradycardia and asystole. We detail his course, which included continual movement in a motion bed and propantheline-bromide (Pro-Banthine) therapy, over 3½ months. Possible causes of bradycardia and autonomic dysfunction in this setting are discussed.

* Chest 1989;95:701-02*

Bradycardia and asystolic cardiac arrest are potentially preventable complications in SCI. Those with lesions above T6 exhibit exaggerated autonomic responses after innocuous stimuli, including bradycardia and cardiac arrest. Because of the scarcity of reports in the American literature, we detail the course of a 19-year-old man with SCI at C5, who suffered recurrent life-threatening bradycardia and asystole. We discuss the hypotheses for the pathogenesis of the bradycardial rhythms and autonomic dysfunction and describe their management.

**Case Report**

A 19-year-old man was admitted to the ICU in coma with multiple traumas following a suicide attempt. He sustained a compression fracture of C6 and dislocation of C5, and had facetted thorax, abdomen, and extremities, absent deep tendon and bulbo cavernous reflexes, and a negative Babinski reflex. His Glasgow coma scale response was 6.† His chest x-ray film was normal. He was intubated to receive respiratory support. Analysis of his arterial blood gases on 35 percent FIO2, respiratory rate 14, and tidal volume 800 ml, were: pH, 7.41; PaCO2, 36 mm Hg; PaO2, 154 mm Hg; and bicarbonate, 16 mEq/L. His vital signs indicated blood pressure of 100/60 mm Hg, a regular pulse of 75 to 80 beats/min, and a respiratory rate of 18 beats/min. While he had spontaneous breathing, weaning from mechanical ventilation was unsuccessful owing to ineffectual inspiratory efforts with paradoxic chest wall movements.

On day 17 he had +1 reflexes in the extremities, and tracheostomy and posterior cervical fusion were done. A new right upper lobe infiltrate was noted; his WBC count was 14.2 x 10³ cu mm. He began receiving therapy with cefotan and gentamicin and had gradual clearing. His heart rate declined to between 65 and 70 beats/min, with bradycardia occurring during suctioning and when moving the patient.

Occasionally, the heart rate would fall unpredictably to 10 beats/min. On one such occasion, he became hypotensive with decreased mental responsiveness. He began receiving atropine, 0.4 mg subcutaneously every 6 h, which controlled the bradycardia. A motion bed was introduced that moved the patient side to side, slowly and continuously, 15-16 times per hour. During this time, he was weaned from mechanical ventilation and atropine therapy. He was transferred to floor care with a normal mental status. The tracheostomy was allowed to close, and he was returned to an ordinary bed.

Two months later, while on standard floor care, he was noted to be bradycardic after a seizure. He was returned to the ICU, where two additional episodes of bradycardia occurred during suctioning and without apparent external stimuli. Hyponxia was not documented before these episodes. He was reintubated and given atropine but continued to have intermittent severe bradycardia. Atropine dosage was increased to 0.6 mg every 4 h, but it failed to inhibit these episodes. Isoproterenol 1:200 (Isuprel) drip at 0.25 µg/min was substituted and then increased to 0.5 µg/min, with poor control of the bradycardia. Cardiac pacemaker implantation was considered but deferred because of pneumonia and two large infected decubitus ulcers over the buttocks and scapula. Two weeks later, Isuprel was still needed, causing excessive perspiration, which limited skin care. Furthermore, bradycardia control was not achieved. The motion bed therapy was resumed, and propantheline (Pro-Banthine), 30 mg every 4 h, added. This successfully controlled the bradycardia without Isuprel.

Therapy with Pro-Banthine induced gastric atony, which was incompletely alleviated with metoclopramide. Gastrostomy placement with sustained feedings helped. The decubitus ulcers necessitated a diverting colostomy and skin grafting to heal. A gradual, monitored decrease of the Pro-Banthine dosage from 30 mg to 15 mg every 4 h, then to every 6 h, was cautiously done. As the patient's status improved and spasticity developed, the motion bed therapy was discontinued. Pro-Banthine dosage was decreased further to 15 mg every 6 h, then to 15 mg every 12 h, then to 15 mg nightly, and finally was successfully discontinued after a total course of 3½ months. The patient had no further bradycardial rhythmics over the next two weeks, when he was transferred to a rehabilitation center.

**Discussion**

There are no reports to our knowledge in the American literature detailing the management of cardiac arrhythmias in SCI. The frequency of death from this complication is unknown. While the spinal shock syndrome includes reflex depression and vasomotor instability, the most threatening aspect of this dysfunction is bradycardia, occasionally progressing to heart block.

Kewalramani reported that the earliest onset of autonomic...