Complications of High Pleural Suction in Bronchopleural Fistulas

RONALD B. TILLES* AND HILLARY F. DON, M.D.†

There is little agreement on how to best manage a bronchopleural fistula. In any patient such an anatomic defect may be of serious consequence. Adequate ventilation, lung re-expansion, control of air leak, and maintenance of an optimal cardiac output are the essential therapeutic goals.[1,2] We describe below an interesting phenomenon that influenced the manner in which a patient was maintained on mechanical ventilation.

REPORT OF A CASE

An 80-year-old white woman was admitted for resection of a squamous-cell carcinoma. The patient underwent a right lower lobectomy and did well until the morning of the seventh postoperative day, when she developed a 40 per cent pneumothorax on the right side. An anterior chest tube was placed in the right second intercostal space, with relief of symptoms. A second chest tube was placed posteriorly in the right seventh intercostal space because a subpulmonic air collection developed on the ninth postoperative day (fig. 1). Mechanical ventilation (PVIIOB Veriflo) was instituted because of progressive CO_2 retention. Positive end-expiratory pressure (PEEP) was added to improve arterial oxygenation. The second tube continually drained as much as 50 per cent of each tidal volume, and only by using a very high suction pressure, approaching 70 cm H_2O, could the lung be maintained inflated. On the eleventh postoperative day it was found that the inspiratory phase of the ventilator was being cycled by negative airway pressure. This was thought at first to be the result of ventilatory effort by the patient. However, inspection did not reveal any inspiratory effort by the patient, and it was apparent that the negative pressure applied to the pleural space was being transmitted through the tracheobronchial tree. This was verified by clamping the chest tube, thereby stopping this inspiratory "effort." The patient did poorly despite a surgical attempt at closing the leak and died suddenly on the fourth day after the second thoracotomy.

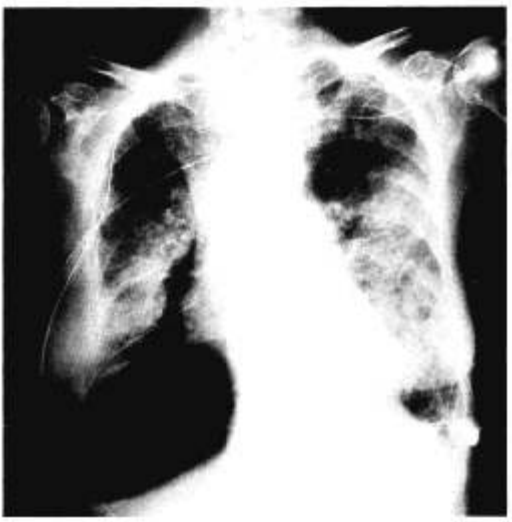

FIG. 1. Portable chest film taken on the ninth postoperative day, showing the subpulmonic air collection on the right.

DISCUSSION

As reported by several investigators,[3,4] bronchopleural fistulas are often best handled surgically as the first line of therapy. Surgery was considered inadvisable for our patient because of her poor condition. Nonsurgical treatment consisting of sufficient suction to maintain lung inflation was therefore instituted. The high suction had three possibly harmful effects: first, PEEP cannot be effectively applied in the face of an air leak as severe as that demonstrated in this case; second, the airways in fact are being subjected to negative pressure, which might promote atelectasis and pulmonary edema in normal lung; third, the negative pressure transmitted to the trachea causes the ventilator to cycle regularly when placed in the assist mode, possibly increasing the ventilatory rate.

* Senior Medical Student.

† Associate Professor, Department of Anesthesia.

Received from the Department of Anesthesia, University of California San Francisco Medical Center, San Francisco, California 94122. Accepted for publication April 2, 1975.

Address reprint requests to Dr. Don.

REFERENCES

1. Zimmerman JE, Colgan DL, Mills M: Management of bronchopleural fistula complicating therapy with positive end expiratory pressure (PEEP). Chest 64:526–529, 1973
2. Khurana JS, Sharma VN: Bronchopleural fistula management during anesthesia. Br J Anaesth 36:302–306, 1964
3. Barker WL: Management of persistent bronchopleural fistulas. J Thorac Cardiovasc Surg 62:393, 1971
4. Dorman JP, Campbell D, Grover FL, et al: Open thoracostomy drainage of postpneumonectomy empyema with bronchopleural fistula. J Thorac Cardiovasc Surg 66:979–81, 1973

An Unusual Cause of False Radial-artery Blood-pressure Readings during Cardiopulmonary Bypass

DEMIR SAKA, M.D.,* YEN TSE LIN, M.D.,† YASU OKA, M.D.‡

During cardiopulmonary bypass at a steady flow rate, vascular resistance is the basic factor regulating regional blood flow. Provided the flow rate is kept constant, the changes in the mean arterial pressure will directly reflect the corresponding changes in peripheral resistance. Therefore, in addition to maintaining an adequate pump flow, mean arterial pressure should be monitored as a useful guide in assessing vasomotor tone, and be kept within a reasonable range to ensure adequate tissue perfusion.[1] Recent literature suggests that at least for the cerebral circulation 50 to 100 torr mean arterial pressure is necessary.[2] Thus, accurate measurement of mean arterial pressure is mandatory during cardiopulmonary bypass, so that proper adjustments of flow rate, anesthetic depth, and the use of vasoactive agents can be made.

We present this case to illustrate an unusual cause of false low readings in mean radial arterial pressure and the potentially hazardous consequences of management based on such readings.

REPORT OF A CASE

A 63-year-old, moderately obese (83 kg) Caucasian man who had a history of angina was admitted for elective aortocoronary-bypass surgery. Cardiac catheterization and an angiogram confirmed triple-vessel disease. Preoperatively, the patient had a grade 2/6 systolic ejection murmur, an elevated blood pressure of 170/90 torr, and a moderately enlarged prostate. Other physical findings were within normal limits. The EKG indicated myocardial infarction of the inferior wall of indeterminate age and myocardial ischemia of the anterolateral wall. A chest x-ray showed slight left ventricular enlargement, mild tortuosity of the aorta, and a right paratracheal density, presumably secondary to the tortuous brachiocephalic vessels. There was no infiltration or consolidation in either lung field.

Prior to surgery, the left radial artery was cannulated for continuous blood-pressure monitoring and a central venous pressure catheter was placed percutaneously through the right internal jugular vein. Both lines were connected to strain-gauge transducers. Pressure readings were recorded continuously through a Hewlett-Packard polygraph monitor. Vital signs just before induction of anesthesia were: blood pressure, 180/90 torr; central venous pressure, 17 torr; pulse rate, 60/min.

Anesthesia was induced with morphine sulfate (total dose 45 mg), followed by diazepam, 10 mg, iv. The trachea was intubated following administration of 3 mg of *d*-tubocurarine, 100 mg succinylcholine, and 5 ml of 4 per cent lidocaine tracheal spray, in that sequence. Following intubation, blood pressure decreased from 180/90 to 150/80 torr. Operation commenced 50 minutes after induction of anesthesia and was associated with an increase of blood pressure to 180/80 torr, so that more morphine, diazepam, and *d*-tubocurarine were given to maintain an adequate depth of anesthesia and relaxation. The anesthetic course was uneventful, and vital signs were stable during sternal splitting and large-vessel cannulation. Partial, then total, cardiopulmonary bypass was instituted, with a stable mean arterial pressure of 80 torr.

Sixty minutes after the start of the total bypass and during the surgical dissection of the internal mammary artery, mean arterial pressure fell to 60 and then to 40 torr within 2 minutes. After ruling out the common causes of hypotension, infusion of phenylephrine hydrochloride (20 μg/ml) was

* Resident in Anesthesiology.
† Assistant Professor of Anesthesiology.
‡ Associate Professor of Anesthesiology.

Received from the Department of Anesthesiology, Albert Einstein College of Medicine, Bronx, New York 10461. Accepted for publication April 2, 1975.

Address reprint requests to Dr. Saka: Albert Einstein College of Medicine, 1300 Morris Park Avenue, Bronx, New York 10461.