

Prone Positioning to Treat Acute Severe Pulmonary Edema in the Post-Cardiac Surgical Patient: A Case Report

Pavan Atluri, MD,¹ Patrick J. Neligan, MD,² Michael A. Acker, MD,¹
Dean L. Bensall, BA, RRT,¹ Jiri Horak, MD²

¹Division of Cardiothoracic Surgery, Department of Surgery, and ²Division of Cardiac Anesthesia, Department of Anesthesia, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania, USA

ABSTRACT

Noncardiogenic pulmonary edema can be fatal without adequate resuscitation. We report, for the first time, the use of prone positioning in the immediate post-cardiac surgical period to treat a patient with profound hypoxemia secondary to massive (noncardiogenic) pulmonary edema. Prone positioning corrects ventilation-perfusion mismatch and allows gravity-dependent drainage of capillary leak-mediated endobronchial pulmonary fluid.

CASE REPORT

We report the case of prone positioning to treat severe hypoxemia secondary to severe acute pulmonary edema immediately after cardiac surgery to improve ventilation-perfusion mismatch and drain extravasated fluid.

A 60-year-old man with idiopathic dilated cardiomyopathy underwent aortic valve replacement as well as mitral and tricuspid valve repair for worsening heart failure, New York Heart Association class III. The preoperative echocardiogram demonstrated a moderately dilated left ventricle (ejection fraction, 25%), mild left atrial dilation, 3-4+ mitral regurgitation, 3+ aortic insufficiency, and 3+ tricuspid insufficiency. Preoperative cardiac catheterization demonstrated normal coronary arteries without disease. The patient had hypothyroidism, chronic renal insufficiency, breast cancer, atrial fibrillation, and a dual-chamber pacemaker for Wolf Parkinson White syndrome. Medications included levoxyl, tamoxifen, metoprolol, enalapril, bumetanide, and warfarin. Preoperative hemoglobin was 12.3 g/dL, with a creatinine of 1.8 and an International Normalized Ratio of 1.6.

The patient underwent an uncomplicated aortic valve replacement with a 25-mm Carpentier-Edwards pericardial

valve (Edwards Lifesciences, Irvine, CA, USA), mitral valve repair with a 28-mm physio ring, tricuspid valve repair with a 32-mm Carpentier-Edwards annuloplasty ring (Edwards Lifesciences), and placement of a left ventricular lateral epicardial ventricular lead. Cardiopulmonary bypass (CPB) time was 133 minutes (aortic cross-clamp time, 109 minutes). Heparin-coated CPB lines were utilized during the case. He was weaned from CPB without difficulty on epinephrine (6 µg/min) and milrinone (0.25 mg/min). Heparin was reversed with protamine without immediate hemodynamic consequences. Post-CPB transesophageal echocardiography (TEE) revealed stable ventricular function with normal function of the aortic, mitral, and tricuspid valves.

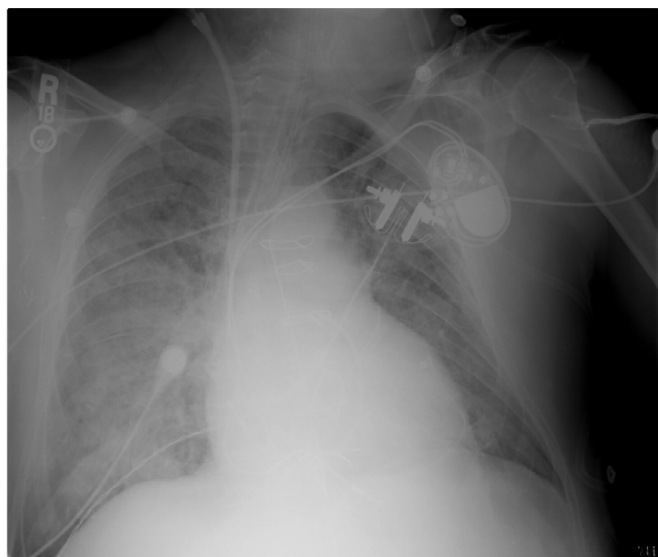
Following chest closure, the patient developed severe pulmonary hypertension (pulmonary artery pressure, 80/50 mmHg), systemic hypotension (blood pressure, 76/39 mmHg; cardiac output, 5.2 L/min; cardiac index, 2), and hypoxemia. Frothy serosanguineous fluid filled the endotracheal tube. As seen in the Figure, the chest roentgenogram at this time revealed extensive bilateral infiltrates. A repeat TEE confirmed severe pulmonary edema with unchanged cardiac function. The patient was treated with norepinephrine (5 µg/min), vasopressin (0.8 units/min), and epinephrine (6 µg/min) targeting a mean arterial pressure of 60 mmHg.

This patient's acute lung injury appeared most likely to be due to an anaphylactoid reaction to an antibiotic (vancomycin or gentamycin), material (ie, latex), or protamine. A total of 100 mg of IV methylprednisolone was administered. Over the next hour, the patient's pulmonary status deteriorated. Massive quantities of edema fluid were suctioned from the endotracheal tube. Lung compliance and arterial oxygen tension dropped dramatically (Table), requiring institution of neuromuscular blockade, and inverse-ratio pressure-controlled ventilation.

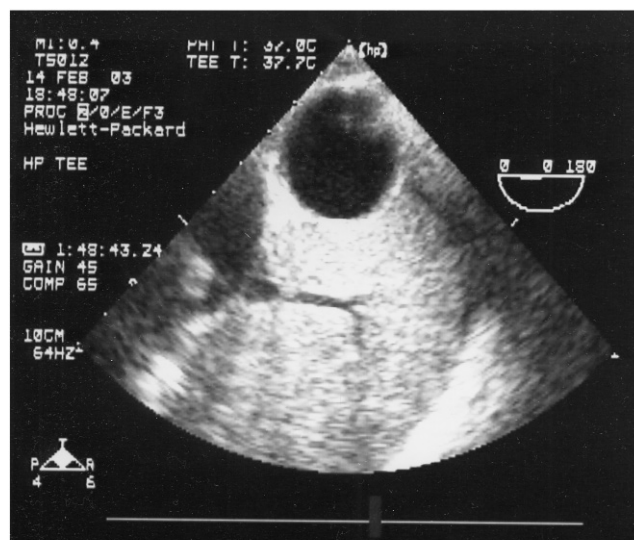
The patient was ventilated manually with an ambu-bag and disconnected from the endotracheal tube every 5 minutes to drain secretions from the ventilator circuit. There was a dramatic improvement in his oxygen saturation but a significant alveolar-arterial oxygen gradient persisted. After 2 hours, the patient was turned prone to facilitate postural drainage of secretions. He was mechanically ventilated and intermittently disconnected for drainage. There was an immediate improvement in lung mechanics. Over the course of 4 hours, approximately 3

Received February 15, 2006; received in revised form May 10, 2006; accepted May 24, 2006.

Address correspondence and reprint requests to: Jiri Horak, MD, Assistant Professor, Department of Anesthesia, 680 Dulles Hospital of University of Pennsylvania, 3400 Spruce Street, Philadelphia, PA 19104, USA; 215-349-8771; fax: 215-349-8133 (e-mail: horakj@uphs.upenn.edu).



A



B

A, Portable chest roentgenogram during the initial episode of severe hypoxemia demonstrating severe pulmonary edema. B, Transesophageal echocardiography image of lung parenchyma representing the fluid-filled lung with outlined bronchial tree.

liters of pulmonary secretions were removed. The pulmonary edema fluid gradually tapered off over several hours.

The patient was weaned from mechanical ventilation over the next 5 days and was discharged home on postoperative day 15 without further complications.

DISCUSSION

This patient had noncardiogenic pulmonary edema, loss of lung compliance, and hypoxemia, a syndrome referred to as acute respiratory distress syndrome (ARDS). A dramatic increase in capillary permeability, most likely as a consequence of an anaphylactoid reaction to protamine [Kimmel 1998], developed. Hypoxemia results from perfusion mismatch and right-to-left shunt through collapsed lung segments in the dorsal and juxta-diaphragmatic lung regions [Puybasset 1998]. There is a dramatic loss of functional residual capacity. Liquid bridges or plugs span the lumen of alveolar ducts, preventing flow of gas [Martynowicz 2001]. This form of lung injury, secondary ARDS, responds to recruit-

ment maneuvers, high levels of positive end expiratory pressure, and prone positioning [Gattinoni 1998; Gainnier 2003].

Prone positioning has been used for 30 years in the management of patients with acute hypoxic respiratory failure [Nakos 2000; Fletcher 2003]. Improvements in both oxygenation and ventilation principally occur due to improved dorsal lung ventilation, resulting from reduced compressive effects of consolidated lung, redistribution of abdominal contents to the cephalad and sternal portion of the lung, reduction in the volume of lung under the heart, and movement of liquid plugs from respiratory bronchioles and alveoli into central airways [Albert 2000]. Pleural pressure becomes more evenly distributed, optimizing ventilation-perfusion distributions.

Dramatic improvements in oxygenation have been described in patients with hydrostatic pulmonary edema [Nakos 2000]. It is likely that fluid removal and drainage plays an important role. Movement of liquid plugs and debris from small to larger airways, according to Laplace's law, allows airways to open at lower distending pressures. This appears to be

Oxygenation Status in the Postoperative Period

Time	FiO ₂ , %	Positive End Expiratory Pressure, cmH ₂ O	Alveolar-Arterial Oxygen Gradient, mmHg	Lung Compliance, mL/cmH ₂ O
Postoperative	100	10	379	31
Supine, 1 h postoperatively	100	10	601	31
Immediate prone, 3.5 h postoperatively	100	15	579	45
Prone, 5.5 h postoperatively	100	12.5	572	46
Postoperative day 1	70	12.5	403	48
Postoperative day 2	40	7.5	153	48

the mechanism for the dramatic improvement in oxygenation and total respiratory system compliance in the current case.

REFERENCES

- Albert RK, Hubmayr RD. 2000. The prone position eliminates compression of the lungs by the heart. *Am J Respir Crit Care Med* 161:1660-5.
- Fletcher SJ, Atkinson JD. 2003. Use of prone ventilation in neurogenic pulmonary oedema. *Br J Anaesth* 90:238-40.
- Gainnier M, Michelet P, Thirion X, Arnal JM, Sainty JM, Papazian L. 2003. Prone position and positive end-expiratory pressure in acute respiratory distress syndrome. *Crit Care Med* 31:2719-26.
- Gattinoni L, Pelosi P, Suter PM, Pedoto A, Vercesi P, Lissoni A. 1998. Acute respiratory distress syndrome caused by pulmonary and extrapulmonary disease. Different syndromes? *Am J Respir Crit Care Med* 158:3-11.
- Kimmel SE, Sekeres MA, Berlin JA, Goldberg LR, Strom BL. 1998. Adverse events after protamine administration in patients undergoing cardiopulmonary bypass: risks and predictors of under-reporting. *J Clin Epidemiol* 51:1-10.
- Martynowicz MA, Walters BJ, Hubmayr RD. 2001. Mechanisms of recruitment in oleic acid-injured lungs. *J Appl Physiol* 90:1744-53.
- Nakos G, Tsangaris I, Kostanti E, et al. 2000. Effect of the prone position on patients with hydrostatic pulmonary edema compared with patients with acute respiratory distress syndrome and pulmonary fibrosis. *Am J Respir Crit Care Med* 161:360-8.
- Puybasset L, Cluzel P, Chao N, Slutsky AS, Coriat P, Rouby JJ. 1998. A computed tomography scan assessment of regional lung volume in acute lung injury. The CT Scan ARDS Study Group. *Am J Respir Crit Care Med* 158:1644-55.