tance to venous return and prevent any net augmentation of preload.[6] Finally, if our observations were due to changes in afterload, similar changes in pressures would occur at both high and low levels of preload.

The pressure variations observed with the pneumatic compression stockings are similar to the systolic pressure variations observed with intermittent positive pressure ventilation. The phasic changes in venous return that occur with positive pressure breaths, with an increase in venous return during the expiratory phase, have long been known to cause an observable pulsus paradoxus. This effect is larger in hypovolemia and nearly disappears during euvolemia.[7] A similar effect was observed in our case, with the pressure variation most marked when the patient was venodilated.

The effect of changes in preload is useful during assessment of cardiovascular function. The MAST suit has been used to create a reversible change in preload.[8] Considering Gaffney *et al.*'s[4] data, the actual effect of the MAST suit may be a change in afterload. The systolic pressure variation seen with positive pressure ventilation has similarly been used to analyze a patient's volume status.[7,9,10] We demonstrate the hemodynamic effects of the pneumatic compression stockings at various levels of preload.

In conclusion, pneumatic compression stockings may cause variations in venous return and arterial pressures, which is an important fact to be aware of for those working in the ICU or operating room. The device appears to work predominantly by augmenting venous return. The observed hemodynamic effects of the pneumatic compression stockings provide another clinical illustration of the physiologic principles described in the Starling curve. Intermittent reversible augmentation of preload may also have value in assessing cardiovascular function.

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Sequential One-lung Ventilation for Bilateral Bullectomy

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The pathologic endpoint of alveolar destruction in emphysema is the formation of a bulla. Bullectomy is indicated when the chest roentgenogram shows localized bullae, dyspnea is intolerable despite maximal medical therapy, and that the bulla is compressing normal adjacent lung tissue.[1–3] Lung compression can be assessed by various combinations of a plain chest roentgenogram, a perfusion scan, angiography, and bronchography. The potential for the compressed lung to have normal function can be demonstrated by a relatively normal ventilation washout of intravenously administered ^{9}Xe.[1–3] Hypoxemia and hypercapnia are not contraindications to bullectomy, and, indeed, such patients may be the ones who benefit the most from surgery.[3,4]

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TABLE 1. Gas Exchange and Ventilatory Mechanics During Sequential One-lung Ventilation for Bilateral Bullectomy

Variable	Anesthesia Stage (In Sequence Left to Right)								
	Preoperative (Rm. Air)	End of Mask Induction, 10 Min	2LV, Chest Open, 10 Min	Left 1LV, 25 Min	Left 1LV, Right CPAP, 15 Min	2LV, 10 Min	Right 1LV, 20 Min	2LV, Chest Open, 10 Min	2LV, Chest Closed, 25 Min
Gas Exchange									
Blood Gases									
Arterial									
pH	7.36	7.27	7.37	7.33	7.37	7.38	7.35	7.36	7.36
P_{CO_2} mmHg	65	88	65	63	57	56	63	59	62
Pa_{O_2} mmHg	50	335	395	52	132	376	137	325	301
% SAT	84	99	99	85	99	99	98	99	99
BE	+8	+8	+10	+5	+6	+7	+8	+7	+8
Venous									
*p*H	7.31	7.23	7.33	7.33	7.32	7.33	7.32	7.33	7.32
P_{CO_2} mmHg	69	94	78	69	62	61	68	66	67
PA_{O_2} mmHg	36	65	56	37	41	64	57	56	55
% SAT	63	82	88	67	72	87	86	85	85
BE	+7	+7	+11	+10	+6	+7	+7	+8	+9
Q_sQ_t, %*	55	23	20	47	30	21	46	25	28
F_{ET} Halo	—	0.75	0.65	0.50	0.48	0.35	0.40	0.45	0.40
Ventilatory Mechanics									
Tidal Vol., ml	—	180	300	250	250	360	320	325	325
PIP, cm H_2O	—	15	30	34	34	29	28	22	24
Static compliance, ml/cm H_2O	—	12	10	7	7	12	11	15	14
RR, b/min	34	30–40	20	25	25	20	25	20	20

$Q_sQ_t = C_c'O_2 - C_aO_2/C_c'O_2 - C_v < O_2$; $C_c'O_2 = 713 - PA_{CO_2}$; C_aO_2 and C_vO_2 = 1.34 (Hb) (% Sat) + 0.0031 (PA_{O_2} or $P\bar{v}O_2$); BE = base excess; F_{ET} = fraction end-tidal; PIP = peak inspiratory pressures; RR = respiratory rate; 1LV = one-lung ventilation; 2LV = two-lung ventilation.

There are no anesthesia or surgical reports describing gas exchange and hemodynamics during the use of one-lung ventilation to facilitate the performance of either unilateral or bilateral bullectomy. In addition, in the few surgical reports that describe bilateral bullectomy *via* median sternotomy, there is a complete absence of anesthetic management information.[5–7] The purpose of this report is to describe the anesthetic management and the successful use of sequential one-lung ventilation for bilateral bullectomy *via* median sternotomy in a very ill patient with far advanced bilateral bullous emphysema.

REPORT OF A CASE

The patient was a 66-yr-old man who presented for bilateral bullectomy with severe emphysema and bilateral apical bullae. At age 20 yr, the patient began smoking one to two packs of cigarettes per day. At age 50 yr, he began experiencing dyspnea on exertion and chronic cough. By age 60 yr, his dyspnea had worsened and bronchodilator therapy was initiated. At age 61 yr, he had one hospitalization for dyspnea at rest, hypoxemia, and hypercapnia. At this time, his forced expiratory volume in 1 s was 23% of predicted, and the forced vital capacity was 39% of predicted. His chest roentgenogram showed flat diaphragms, and electrocardiogram showed possible right ventricular hypertrophy. Following this hospitalization, the patient stopped smoking and received nocturnal oxygen, prednisone, aminophylline, albuterol tablets, and inhaler therapy. Nevertheless, by age 62 yr, his dyspnea and degree of hypoxemia had increased further requiring continuous oxygen therapy by nasal prongs. His chest roentgenogram showed biapical lucencies with decreased vascularity and vascular crowding bilaterally at the bases.

At age 64 yr, he was reduced to a bed-to-chair existence due to progressive dyspnea. On physical examination, no breath sounds were heard at the apices, and breath sounds were distant with rales at the bases. The chest roentgenogram showed large bilateral apical bullae occupying 65% of the right hemithorax and 50% of the left hemithorax, and a bilateral crowding of vessels at the bases. Spirometry along with plethesmographic and helium dilution fuctional residual capacity measurements showed severe hyperinflation, increased airway resistance and expiratory obstruction, and a large noncommunicating compartment. Several room air arterial blood gases showed a Pa_{O_2} which ranged between 45–60 mmHg, Pa_{CO_2} between 50–72 mmHg, and a pH_a between 7.36–7.42. A perfusion scan showed almost no upper lobe perfusion bilaterally, good perfusion of the bases, the left base greater than the right base. A ventilation scan showed no ventilation to the upper lobes and equal washout from the lower lobes. The bullae were thought not to communicate with the bronchial tree. The electrocardiogram showed right ventricular hypertrophy, right axis deviation, and biatrial enlargement. The patient was scheduled for bilateral bullectomy *via* median sternotomy following a 2-week period of optimization of pulmonary function. The patient refused to allow a double-lumen tube to be inserted into his trachea while he was awake.

On arrival in the operating room, a 14-gauge peripheral venous catheter, a 20-gauge radial artery catheter, and a 7-French triple-lumen pulmonary artery catheter were placed percutaneously with the aid of local anesthesia (tables 1, 2, first column). Prior to the induction of anesthesia, the chest was prepared and draped for potential chest tube insertion if a pneumothorax developed during the induction of anesthesia. Additional noninvasive monitors were an electrocardiogram, pulse oximeter, end-tidal carbon dioxide and halothane tension, airway pressure, and tidal volume by spirometry. The induction of anesthesia (in the supine position) consisted of sequential administra-

TABLE 2. Hemodynamics During Sequential One-lung Ventilation for Bilateral Bullectomy

Variable	Anesthesia Stage (In Sequence Left to Right)								
	Preoperative (Rm. Air)	End of Mask Induction, 10 Min	2LV, Chest Open, 10 Min	Left 1LV, 25 Min	Left 1LV, Right CPAP, 15 Min	2LV, 10 Min	Right 1LV, 20 Min	2LV, Chest Open, 10 Min	2LV, Chest Closed, 25 Min
Pressure, mmHg									
P_{pas}/P_{pad}	58/30	25/14	18/10	35/22	25/15	18/10	33/26	23/16	23/16
P_{pam}	39	18	13	26	18	13	28	18	18
P_{paw}	7	8	8	10	10	11	10	8	8
P_{ra}	7	7	7	10	11	8	8	8	9
P_{sam}	85	79	88	80	88	94	97	97	90
Q_t, l/min	6.5	6.7	5.5	4.8	4.8	5.0	5.2	5.3	5.3
O_2 transport ml/min	960	1187	935	633	761	870	848	853	848
C (a-v) O_2, ml/100 ml	3.7	3.5	3.2	2.8	4.4	3.1	2.2	3.0	2.9
VO_2, ml/min	241	235	176	135	211	155	114	159	154
Resistance Dynes · s · cm^{-5}									
PVR	394	119	91	267	133	32	277	151	136
SVR	960	860	1178	1167	1283	1376	1400	1374	1224
Hb, gm %	13.0	12.0	11.8	11.7	11.7	12.1	12.1	11.3	11.3
HR, b/min	95	105	111	92	90	91	91	86	93

O_2 transport = $Q_t \times C_aO_2$; PVR = $(P_{pam} - P_{paw})$ (80)/Q_t; SVR = $(P_{sam}-P_{ra})$ (80)/Q_t; pas = pulmonary artery systolic; pad = pulmonary artery diastolic; pam = pulmonary artery mean; paw = pulmonary artery wedge; ra = right atrial; sam = systemic arterial mean; Q_t = cardiac output; VO_2 = oxygen consumption; PVR = pulmonary vascular resistance; SVR = systemic vascular resistance; Hb = hemoglobin concentration; HR = heart rate; 1LV = one-lung ventilation; 2LV = two-lung ventilation.

tion of fentanyl 150 ug iv in three divided doses, thiopental 150 mg iv, administration of halothane up to an inspired concentration of 2% in oxygen, pancuronium 4 mg iv, metocurine 12 mg iv, and lidocaine 100 mg iv. After the administration of the thiopental and during the period of deepening anesthesia and paralysis (10 min), ventilation was controlled *via* manual compression of the anesthesia circle system reservoir bag with a peak positive inspiratory pressure of 15 cm H_2O (tidal volumes were approximately 200 ml as measured by the in-line expiratory limb spirometer), and a respiratory rate between 30–40 breaths/min (tables 1, 2, second column). During the induction, arterial blood pressure and heart rate were kept relatively constant (range 140–115/90–70 mmHg and 110–90 beats/min, respectively) with ephedrine 10 mg iv in two divided doses. When the patient was thought to be adequately anesthetized and paralyzed, and was hemodynamically stable, the larynx and trachea were sprayed with 4% lidocaine, and a 39 French left-sided double-lumen tube (Bronchocath®) was inserted. The proper position of the double-lumen tube was confirmed by a fiberoptic bronchoscope; the left blue endobronchial cuff of the double-lumen tube was visualized to be just below the tracheal carina. Both lungs were then mechanically ventilated with a moderately higher peak inspiratory pressure and lower respiratory rate than used during manual compression of the reservoir bag (table 1). Anesthesia was maintained over the next 3 h with halothane in oxygen (see end-tidal values in table 1), fentanyl 400 μg iv in four divided doses in the first 2 h of anesthesia, morphine 18 mg iv in six divided doses in the last hour of anesthesia, and paralysis was maintained with one additional dose of pancuronium 1 mg iv, and metocurine 4 mg iv.

Surgery began with a median sternotomy. On opening the mediastinum, both lungs met in the midline and were markedly hyperinflated (tables 1, 2, third column). Since the bulla on the right side was thought to be larger than the one on the left side, and left lung function was better than right lung function, the lumen to the right lung was clamped, the left lung was ventilated with the same peak inspiratory pressure and respiratory rate that was used for both lungs (table 1), and the right parietal pleura was incised. The pleural incision delivered a large right upper lobe bulla which was resected and closed with multiple staplings (tables 1, 2, fourth column). Several smaller upper and middle lobe bullae were also resected and stapled. Following the right lung bullectomies, the remaining right lung was reexpanded with 40 cm H_2O, absence of air leaks confirmed, and two-lung ventilation commenced as before (tables 1, 2, sixth column). The major portion of the right middle lobe and all of the right lower lobe appeared to have a normal consistency and expansion. Since the patient was stable with regard to hemodynamics and gas exchange following the right bullectomy, the lumen to the left lung was clamped, the right lung ventilated with the same peak inspiratory pressure and respiratory rate as used before, and the left pleural space was incised and a left upper lobe bullectomy performed (tables 1, 2, seventh column). Several other smaller left lower lobe bullae were resected and stapled. Following the left lung bullectomies, the left lung was reexpanded with 40 cm H_2O, absence of air leaks confirmed, and two-lung ventilation commenced once again (tables 1, 2, eighth and ninth columns).

Table 1 shows the gas exchange and ventilatory mechanics that occurred during the various stages of the surgical period. Each individual lung, and both lungs together, were very poorly compliant, and only a very small tidal volume could be delivered using moderately high peak inspiratory pressures. Consequently, the patient was moderately to severely hypercapnic throughout the entire procedure. During the right lung bullectomies and ventilation of just the left lung, the arterial oxygen tension (Pa_{O_2}) steadily decreased to 52 mmHg (intermediate values [not reported] described an exponential decay). Five cm H_2O of CPAP to the nonventilated right lung (applied during the deflation phase of a tidal breath to the right lung) restored the Pa_{O_2} to 132 mmHg (tables 1, 2, fifth column). Satisfactory systemic oxygenation was much more easily obtained during one-lung ventilation of the post-bullectomy right lung.

Table 2 shows the hemodynamic changes that occurred during the various surgical stages. During periods of one-lung ventilation, pulmonary artery pressure and pulmonary vascular resistance were much increased compared to the immediately preceding or following two-

lung ventilation period. Systemic blood pressure, heart rate, and cardiac output were relatively constant during the entire procedure. The infusion of 2 l of lactated Ringer's solution for an estimated blood loss of 360 ml resulted in a 150 ml urine output for the 3-h procedure. The pulmonary artery wedge pressure and the right atrial pressure remained nearly constant throughout the procedure.

At the end of the surgical procedure, the double-lumen tube was changed to a single-lumen tube, the patient was turned into a lateral decubitus position, a T5-6 thoracic epidural catheter was inserted, and epidural fentanyl analgesia begun. The patient was ventilated for 3 postoperative days, and then weaned from the ventilator without incident. The patient was discharged 2 weeks postoperatively with improved arterial blood gases (Pa_{O_2} = 66 mmHg and Pa_{CO_2} = 48 mmHg) and spirometry (forced expiratory volume in 1 s = 28% of predicted and forced vital capacity = 56% of predicted). He experienced a significant improvement in dyspnea (exercise tolerance of walking 2–3 level blocks) at least for 1 yr following his bullectomy.

Discussion

This case report is unique in that it describes in detail the gas exchange and hemodynamic effects of using sequential one-lung ventilation to facilitate the performance of bilateral bullectomy. Before duscussing the one-lung ventilation experience *per se,* there are two important interrelated controversial issues concerning patients with large bullae that should be discussed. These two issues are method of induction of general anesthesia in a patient with a large thin-walled bulla, and the minute ventilation that should be sought in these chronically hypercapnic patients.

The great danger during the induction of anesthesia in a close-chested patient with a large thin-walled bulla is rupture of the bulla resulting in a tension pneumothorax.[8,9] A tension pneumothorax in a patient with preexisting marginal gas exchange would be life-threatening. There are four mechanisms by which a bulla may rupture during the induction of anesthesia. First, if the bulla is poorly communicating or noncommunicating with the bronchial tree, then nitrous oxide (if used) could diffuse from the blood into the bulla and cause inexorable expansion and, finally, rupture. Second, if the bulla is in good communication with the bronchial tree and is very compliant, then positive pressure ventilation may cause a large fraction of the tidal volume to enter and rupture the bulla. Third, if a ball valve effect traps inspired gas within the bulla, progressive expansion of the bulla will result in rupture. Fourth, if the wall thickness of the bulla is uneven and/or the pleural pressure surrounding the bulla is uneven, then a very positive pleural pressure due to a forceful exhalation and/or "bucking," may cause rupture of the bulla.[8]

Double-lumen tube endobronchial-tracheal intubation allows separation of the two lungs and administration of a controllable amount of positive pressure (including no positive pressure) to a lung that has a bulla. Although a double-lumen tube has been inserted under topical anesthesia while the patient was awake,[10] our patient rejected this alternative. The patient can be induced by spontaneously breathing a halogenated anesthetic until the depth of anesthesia permits double-lumen tube intubation without paralysis.[11] However, because this patient was hypercapnic and hypoxemic preoperatively, ventilatory depression that would accompany adequate anesthesia may cause life-threatening inadequate gas exchange. High frequency jet ventilation *via* a jet catheter attached to a single-lumen tube has been used (following intravenous induction and paralysis) in a patient with a large bulla undergoing coronary artery bypass grafting,[12] but this method precludes the use of one-lung ventilation.

During the induction of anesthesia, peak inspiratory pressures less than 15 cm H_2O were applied until the double-lumen tube was inserted. The risk of increased distension of the bulla was considered to be reduced (but not eliminated) by the failure to demonstrate bronchial communication on the preoperative ventilation scan. Since both hemithoraces were already prepared and draped, and the surgeons were standing by gowned and gloved, either or both chests could be entered and decompressed very quickly if any question of pneumothorax arose. Following double-lumen tube insertion, the compliance and end-tidal carbon dioxide tension of each lung could be monitored, and, therefore, the ability to detect and treat pneumothorax with rapidity and certainty was much increased. Consequently, peak inspiratory pressure was allowed to increase to 30 cm H_2O during both the subsequent two-lung ventilation and the sequential one-lung ventilation periods. There is one report in the literature describing a patient with a bulla who underwent a lumbar laminectomy in the prone position and was exposed to a limited two-lung ventilation peak inspiratory pressure of 20 cm H_2O for the duration of the case without incidence.[13] Nitrous oxide was avoided throughout the case, especially since the patient's bullae were thought to have poor communication with the bronchial tree.

The patient was chronically hypercapnic preoperatively. Consequently, the level of alveolar ventilation was designed to keep the end-tidal and arterial P_{CO_2} near the preoperative values and, thereby, keep the pH_a normal. Towards that end, the maximal pH_a decrease and increase during the operation was 0.07 and 0.03, respectively. If a normal level of alveolar ventilation had been achieved, the patient would have been severely alkalemic and at increased risk for rupture of a bulla due to an increased amount of airway positive pressure.

Sequential one-lung ventilation was used to facilitate sequential removal of bullae from the nonventilated contralateral lung. The right lung bulla was resected

first because it was the largest, and, on the basis of preoperative perfusion and ventilation scans, the left lung was thought to be able to support gas exchange better than the right lung. Each time one-lung ventilation was initiated, the tidal volume was decreased slightly and the respiratory rate was increased slightly. Each time one-lung ventilation was initiated, mean pulmonary artery pressure and pulmonary vascular resistance increased compared to the immediately preceding and following two-lung ventilation situations. The increase in pulmonary vascular resistance and mean pulmonary artery pressure is consistent with hypoxic pulmonary vasoconstriction in the nonventilated lung. Presumably, hypoxic pulmonary vasoconstriction in the nonventilated lung reduced shunt flow through the nonventilated lung and, thereby, minimized decreases in $P_{A_{O_2}}$.

After 25 min of ventilation of just the bullous left lung, the patient became moderately hypoxemic ($P_{A_{O_2}}$ = 52 mmHg). Five cm H_2O of oxygen CPAP applied to the right lung during the deflation phase of a tidal breath increased the $P_{A_{O_2}}$ to 132 mmHg. With only 5 cm H_2O CPAP, no redistribution of blood flow away from the nonventilated lung to the ventilated lung is expected.[14] The 5 cm H_2O CPAP to the right lung during just left lung ventilation decreased mean pulmonary artery pressure and pulmonary vascular resistance to a value intermediate between the last one-lung ventilation value and the before and after two-lung ventilation values, indicating that some hypoxic pulmonary vasoconstriction in the nonventilated right lung was reversed.

In summary, this case report demonstrates several important clinical anesthesia management points. First, gentle positive pressure ventilation may be used during the induction and maintenance of anesthesia, especially if the bullae are thought to have no, or poor, communication with a bronchus (as demonstrated by preoperative ventilation scanning). Both hemithoraces should be continuously auscultated and both hemithoraces prepared, and the surgeon gowned and gloved for immediate chest decompression, should a pneumothorax be suspected. Second, the patients may remain hypercapnic provided they have a normal pH_a and Pa_{O_2}. There is no need to strive for normocapnia, thereby exposing the patients to the dangers of increased airway pressure and alkalemia. Third, sequential one-lung ventilation may be used to facilitate the removal of bilateral bullae. The lung thought to have the worst disease should be operated on first, and the lung with the better function should support gas exchange first. When the roles of the lung are switched, the lung function and gas exchange capability of the post-bullectomy lung should be improved. If hypoxemia develops during one-lung ventilation, application of CPAP to the nonventilated lung during the deflation phase of a tidal breath should be expected to increase Pa_{O_2} as it routinely does in other thoracic surgery cases.[15] Finally, it may be especially important to keep the determinants of the mixed venous $P\bar{v}O_2$ and the $P\bar{v}O_2$ itself, near normal in these very ill patients, and, with that in mind, pulmonary artery pressure monitoring and oximetry is indicated.

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