Hypotension Secondary to Air Trapping Treated with Expiratory Flow Retard

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Expiratory flow retard has been employed to simulate "pursed lip" breathing in an attempt to prevent premature airway collapse and air trapping in patients with obstructive pulmonary disease.¹ However, there are no clinical or laboratory studies to substantiate its efficacy in relieving acute air trapping. Schmidt et al.² found that expiratory retard increased vital capacity in patients with stable, chronic emphysema, but not with asthma patients in remission. This increase only occurred when the expiratory airflow rate was reduced. Neither group showed any signs of acute air trapping. Abbadou et al.³ showed that expiratory retard increased efficiency of breathing by decreasing respiratory rate, tidal volume, and thus minute ventilation without altering arterial blood gases in patients with chronic obstructive airway diseases. The characteristics of lung emptying were not studied. Also, these patients showed no signs of acute air trapping. The case reported below is one in which severe pulmonary air trapping developed intraoperatively leading to a cardiac tamponade effect during closure of the sternum after cardiac surgery, which was effectively relieved by expiratory retard.

REPORT OF A CASE

A 55-year-old, 63-kg woman with a one-month history of progressive angina was found to have three-vessel coronary artery disease during angiography, and was scheduled for aorto-coronary bypass graft surgery.

Relevant past medical history included a 50 pack-year history of smoking, positive history of bronchitis, and/or asthma with episodes of shortness of breath for which she took oral theophylline frequently. However, she had not taken any for at least 48 hours prior to surgery. Her chest roentgenogram was interpreted as negative. Analysis of arterial blood gases revealed $pH = 7.43$, $P_{CO_2} = 46$ mmHg, $P_{O_2} = 74$ mmHg, and $P_{O_2} = 2.01$. Screening pulmonary function tests revealed FVC of 600 ml and FEV₁ of 250 ml, which were believed to be due to submaximal effort by the patient.

Diazepam, 10 mg; morphine sulfate, 10 mg, and scopolamine, 0.2 mg were given intravenously. She was well-sedated upon arrival in the operating room. Prior to induction of anesthesia, intravenous, arterial, and thermography measurements of pulmonary artery catheters were inserted without difficulty. The pulmonary arterial pressure (PAP) was 19/10 mmHg with a pulmonary capillary wedge pressure (PCWP) of 10 mmHg, arterial pressure (AP) of 110/70 mmHg, and cardiac output (CO) of 4.5 l/min⁻¹. Induction and maintenance of anesthesia were achieved with a total of 5 ml Innovar, 1.25 mg fentanyl, 10 mg diazepam, and 12 mg pancuronium iv. Ventilation was controlled with a rate of 6 to 8 breaths ⋅ min⁻¹ throughout the case. Prior to cardiopulmonary bypass, $pH = 7.38$, $P_{CO_2} = 45$ mmHg, and $P_{O_2} = 264$ mmHg at $F_{IO_2} = 1.0$. Surgery and anesthesia were uneventful. Weaning from bypass was accomplished without difficulty. Post-bypass hemodynamics were maintained relatively stable on low-dose nitroprusside (SNP) (≤0.5 µg/kg/min) and dobutamine (∼1–3 µg/kg/min); $pH = 7.41$, $P_{CO_2} = 37.3$ mmHg, $P_{O_2} = 107$ mmHg at $F_{IO_2} = 1.0$; AP was 100/60 to 130/70 mmHg, PAP was 22/14 mmHg, PCWP was 10 to 12 mmHg, and CO was 4.6 l/min⁻¹. A relatively normal intravascular blood and fluid volume status, and normal cardiac performance were indicated by the normal PCWP, CO, and AP. Hemodynamic stability was extremely well-maintained until attempts were made to close the sternum. Upon apposition of the sternal halves, despite an increase in dobutamine infusion to 10 µg/kg/min and discontinuation of SNP, the AP precipitously fell to 60/40 mmHg with a concomitant rise in PAP from 22/14 to 35/22 mmHg and PCWP from 10 to 14 mmHg. The CVP was not monitored because the CVP part of the PA catheter was being used for dobutamine infusion. The chest was re-opened immediately for further evaluation and possible repair. Once the chest was re-opened the AP, PAP, and PCWP promptly returned to their previous levels without further treatment (fig. 1).

At this time, the lungs were noted to be hyperinflated and herniating out of the widely open right and left pleural cavities across the mediastinum. They would not deflate during the expiratory phase despite a slow rate of six breaths/min via an Ohio® Fluidic Ventilator. Faint expiratory wheezes were heard. Arterial blood gases at this time revealed $pH = 7.35$, $P_{CO_2} = 43$ mmHg, and $P_{O_2} = 346$ mmHg at $F_{IO_2} = 1.0$. Aminophylline, 250 mg iv, was administered with little effect. On each of several subsequent attempts to close the chest, we observed that the lungs literally herniated out of the wound, and hypotension recurred. This confirmed the impression that a tamponade effect of the hyperinflated lungs on the heart was the cause. Although a PEEP attachment was not used on the Ohio ventilator, we considered the possibility of "inadvertent PEEP," which has been noted in patients with chronic lung disease who are treated with conventional mechanical ventilation and in experimental animals treated with high-frequency ventilation (HFV). Since the ventilator rate was only 6/min and the condition was not relieved by intermittent manual ventilation, this possibility was deemed unlikely to be playing a major role.

Expiratory retard (minimal expiratory flow rate) was applied in an attempt to facilitate exhalation and to relieve the air trapping.

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Initial attempts with the Ohio fluidic anesthesia ventilator were unsuccessful, and a greater degree of expiratory retard was deemed necessary. Therefore, an IMV Bird ventilator at the same rate and with the expiratory retard attachment to the exhalation port set at the most restricted setting was employed. Adequate deflation of the lungs occurred rapidly, and the chest subsequently was closed with maintenance of hemodynamic stability. At this time, arterial blood-gas analysis revealed $\text{pH} = 7.23$, $\text{PaCO}_2 = 49$ mmHg, and $\text{PaO}_2 = 379$ mmHg at $\text{FiO}_2$ 1.0. AP was 120/70 mmHg. PAP returned to 25/10 mmHg and PCWP to 4 mmHg. One unit of blood was transfused to augment preload.

Immediately postoperatively in the surgical intensive care unit, arterial blood-gas analysis while the patient was treated with the same ventilator at identical settings and retard showed $\text{pH} = 7.31$, $\text{PaCO}_2 = 42$ mmHg, and $\text{PaO}_2 = 112$ mmHg at $\text{FiO}_2$ 0.4. Several attempts to remove expiratory retard resulted in hemodynamic instability, and support with IMV Bird Ventilator, expiratory retard, and aminophylline infusion was required for 38 hours before weaning and extubation of the trachea was successful. She continued to improve and was discharged from the hospital on the eighth postoperative day.

**DISCUSSION**

Hypotension accompanying cardiac tamponade is caused by extracardiac mechanical limitation of ventricular filling which decreases venous return and therefore cardiac output. Similarly, the precipitous hypotension during closure of the chest in this patient was caused by mechanical limitation of ventricular filling as well as the high intrathoracic pressure transmitted by the hyperinflated lungs decreasing venous return to both ventricles.

The patient had a long history of chronic bronchitis and/or asthma. Following cardiopulmonary bypass, she developed severe small airway obstruction and air-trapping that could be attributed to bronchospasm, bronchiolar mucosal edema, thick, innsipated mucus plugging of small airways, and collapsed small airways caused by lack of elastic support in the lung secondary to coalescence of alveoli and destruction of alveolar septae. Expiratory retard employed in our patient appeared to dramatically decrease air-trapping and allow closure of the sternotomy without cardiovascular instability.

The exact timing and causes of the severe pulmonary air trapping in this case are not known. Post-bypass, because of normal blood gases and stable hemodynamics, no specific attention was paid to the overinflated lungs until after the episode of precipitous hypotension during closure of the chest. Stable arterial pressure and heart rate during the application of sternal wires prior to closure of the chest seemed to indicate an adequate anesthetic level. Reflex bronchospasm from light anesthesia probably cannot be implicated. The fact that pancuronium, approximately 2 mg/kg, had been given makes it unlikely that inadequate muscle relaxation could have caused chest wall rigidity and protrusion of the lungs. Furthermore, the degree of air trapping that caused the lungs to literally herniate out of the wound clearly indicated that air trapping, rather than chest wall rigidity, was the problem. No additional anesthetics or muscle relaxants were given during the period immediately prior to or after instituting expiratory retard via the IMV Bird Ventilator. This further demonstrated that anesthetic depth and degree of muscle relaxation were not significant contributing factors to the causes of air trapping, nor to the efficacy of the expiratory retard.

The retard capability of various ventilators have not been well-quantified and compared. A far greater range of retard can be administered with the Bird expiratory retard valve than with the Ohio Fluidic Ventilator. It was the inadequate degree of retard obtainable with the Ohio Fluidic Ventilator rather than any other ventilator characteristics that caused the switch to IMV Bird Ventilator.

How expiratory retard affects respiratory mechanics in anesthetized, paralyzed, control-ventilated, open-chested, patients is not presently known. Respiratory mechanics, e.g., minute ventilation, peak dynamic or static inspiratory airway pressures, etc., prior to or after the use of retard were not measured or recorded. It was considered by the surgeons and us that the patient was in a dire state and that treatment, rather than measurement of pulmonary function, was essential. However, arterial blood gases during the period of lung overinflation and after imposition of retard were measured, and $\text{PaO}_2$ and $\text{PaCO}_2$ were not changed greatly. Regardless of how retard affected the respiratory mechanics, its efficacy in relieving acute air trapping was clearly documented in this case. With the chest open, the direct observation was made that profound pulmonary air trapping had occurred. The obvious and dramatic relief of this air trapping, with the use of expiratory retard via IMV Bird Ventilator, also was observed directly. Other possible factors such as inspiratory characteristics of the ventilator, delayed effects of aminophylline, depth of anesthesia, degree of muscle relaxation, etc., which might partly con-
tribute to the relief of air trapping did not seem to play a significant role in this case.

Presently there is no satisfactory explanation as to how expiratory retard effectively enhances exhalation in patients with severe small airway obstruction. Studies on flow limitation during forced expiration have shown that maximum expiratory flow rate is limited by lung recoil regardless of the expiratory effort. This has been explained on the basis of the equal pressure point (EPP) concept. During normal forced expiration, pleural pressure becomes positive and is transmitted to the alveoli and intrapleural airways. Alveolar pressure is the sum of elastic recoil pressure of the lung and pleural pressure. An airway intraluminal pressure gradient is generated. Pressures are equal to alveolar pressure in the respiratory bronchioles and taper to atmospheric pressure at the mouth. At some point along the airway the intraluminal pressure equals pleural pressure, i.e., the equal pressure point. Intraluminal pressure downstream from the EPP is less than pleural pressure; thus, the airways have a tendency to collapse. Smaller, less rigid airways collapse most easily, larger airways with cartilaginous support are more resilient. In normal individuals, the EPP is presumably high (i.e., at or close to the larynx) and airway closure does not occur on expiration. In patients with diseases producing loss of elastic recoil, bronchial mucosal edema, or constriction, the EPP may move peripherally to the collapsible small airways. The application of expiratory retard moves the EPP toward the larynx and thereby prevents airway closure. In the present case, however, air trapping occurred while the chest was open, with the patient paralyzed and mechanically ventilated. Pleural pressure equaled atmospheric pressure, and expiration was passive. Total intraluminal pressure during expiration is always greater than pleural pressure when airway pressure at the mouth equals atmospheric pressure. The conventional EPP concept, therefore, in this case becomes inapplicable. However, similar principles may be applied. Lateral pressure exerted on the wall of a narrowed area of an airway may be subatmospheric by the Bernoulli effect, even when total intraluminal pressure at that site is greater than atmospheric. The EPP can be said to be the site at which lateral pressure exerted on the wall equals pleural pressure (atmospheric pressure). Similarly in this case, application of expiratory retard moved the EPP toward the larynx, thereby relieving air trapping.

Historically, Alvan L. Barach first employed expiratory airway pressure maneuvers to enhance exhalation in patients with chronic obstructive pulmonary disease (COPD). In 1934, he described a patient with COPD who exhaled forcibly against partially closed lips. "When the patient tried to follow the suggestion to open his mouth and 'breathe easily', he seemed to suffocate; when his mouth was held open, no air came out until suddenly he pursed his lips and exhaled the pent up air with ap-

parent great relief." This impressive experience stimulated Barach to develop the continuous positive pressure breathing (CPPB) technique to treat COPD patients, by which the premature airway closure appeared to be prevented. In 1939, he showed roentgenographically that the width of the branches of the bronchial tree consistently increased when CPPB was applied in patients with asthmatic attacks.

CPPB, as developed by Barach, is identical to CPAP (continuous positive airway pressure) later introduced by Gregory et al., in spontaneously breathing patients. Positive expiratory airway pressure used in conjunction with mechanical ventilation is termed PEEP. Both CPAP and PEEP differ from expiratory retard in that during expiratory retard the positive airway pressure gradually returns to atmospheric level at the end of expiration.

Whether CPAP or PEEP would promote exhalation in patients with severe airway obstruction as effectively as expiratory retard is presently not known. Some authorities believe that CPAP or PEEP is contraindicated in COPD patients because positive expiratory airway pressure either in the form of CPAP or PEEP is known to increase the functional residual capacity (FRC), and that patients with COPD already have marked increases in FRC. Thus, application of CPAP or PEEP might theoretically worsen the air-trapping. This belief has yet to be proven. It was in fact CPAP that Barach employed in treating COPD patients in which he indicated that the expiratory pressure tended to prevent collapse of the bronchial airway and the inspiratory pressure provided a slight assistance to expansion of the lungs. Recently, Martin et al. demonstrated that in patients with acute experimentally induced asthma attacks, CPAP reduces airway resistance and increases expiratory flow in addition to its major effect of assistance in inspiration.

In the present case, impedance to exhalation was so great that emptying of the lungs to FRC would have required a protracted period of time. Inspiration was unimpeded and easily accomplished. Thus, "stacking" or accumulation of end expiratory lung volume was occurring. The problem at hand was to provide a maneuver to allow deflation of the over-distended lungs and deliver a reasonable minute volume. This was accomplished with expiratory flow retard. CPAP or PEEP may increase FRC in patients without air trapping. Whether or not CPAP or PEEP will relieve air trapping as might expiratory flow retard by maintaining a patent bronchial tree needs further study.

REFERENCES

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Air Embolism in Upright Neurosurgical Patients: Detection and Localization by Two-dimensional Transesophageal Echocardiography

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The clinical use of M-mode contrast echocardiography, a technique in which saline containing microbubbles of gas injected iv is utilized as an echocardiographic contrast medium, was first described by Seward et al.1 in 1975. With new technology, transesophageal echocardiography (TEE) utilizing M-mode transducers have been designed to detect air embolism in neurosurgical patients.2 With the development of 2-dimensional (2-D) sector echo transducers, a large tomographic field of view could be obtained which permits better appreciation of anatomic relationships. The heart is visualized in a “bread-slice tomographic plane” rather than the “ice-pick” view of the M-mode echo.

METHODS

This work describes the use of a prototype, 3.5-MHz transesophageal echocardiographic (TEE) probe (Diasonics) interfaced with a 3400 Diasonics phased array sector echo instrument. With informed consent and after the induction of general anesthesia, the probe was inserted in 15 patients undergoing suboccipital craniotomy in the sitting position. Precordial Doppler and right atrial catheter were used in all patients. The echo image was monitored on a video screen and recorded on 3/4-inch video tape for real time and slow motion independent review by an experienced cardiologist (JBS). An image at just below the level of the aortic valve which displayed right and left atrial cavities was utilized (fig. 1). The forced injection of saline (echo contrast medium) was used to determine Doppler position and chamber visualization on echo. Saline injection was repeated hourly during surgery. This study was designed not to replace conventional monitoring but to test the comparative sensitivity of 2-D TEE in the neurosurgical environment. Thus, once in position, the TEE was turned off and precordial Doppler was used to monitor for the presence of an air embolism. During episodes of detectable air embolism, the echo probe was activated to confirm and localize air to right and/or left heart chambers. A single bubble of air can be detected by 2-D echocardiography.1,3

RESULTS

In nine of 15 cases, the precordial Doppler detected air. Every TEE easily visualized and localized the air. In two cases where precordial detection was questionable, TEE confidently verified air. The most important finding

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