The Use of High Positive End-Expiratory Pressure for Respiratory Failure in Abdominal Compartment Syndrome

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We report a case in which a non-trauma patient suffering hematemesis and undergoing massive volume resuscitation developed abdominal compartment syndrome (ACS). The abdominal distension severely compromised his pulmonary functioning: a chest radiograph showed low lung volumes and dense bilateral parenchymal opacities. His blood oxygen saturation reached as low as 32%. Because he was hemodynamically unstable and coagulopathic, decompressive surgery was not possible. We gradually raised the ventilator settings to reinflate the lungs (positive end-expiratory pressure [PEEP] was raised to 50 cm H$_2$O, peak inspiratory pressure to 100 cm H$_2$O, and plateau inspiratory pressure to 80 cm H$_2$O) and continued fluid resuscitation, and within an hour his blood oxygen saturation increased to 100%. In this case high PEEP was beneficial in a situation in which decompressive surgery was not feasible, but we do not suggest that high PEEP necessarily improves survival or that high PEEP is better than surgical decompression. On the contrary, high-pressure ventilation can be harmful in the setting of acute lung injury and acute respiratory distress syndrome, so we do not advocate high PEEP for all patients with hypoxemia and ACS, especially considering that many of the conditions associated with ACS can also precipitate acute lung injury and acute respiratory distress syndrome. As well, high-pressure ventilation can increase the risk of hypotension by impairing venous return. However, our case suggests that high PEEP may temporize in certain situations in which ACS causes life-threatening hypoxia but surgical decompression is not possible. *Key words: abdominal compartment syndrome, positive end-expiratory pressure.* [Respir Care 2004;49(3):286–290. © 2004 Daedalus Enterprises]

Introduction

Abdominal compartment syndrome (ACS) is broadly defined as organ dysfunction resulting from increases in intra-abdominal pressure.[1] ACS develops in the setting of acute and rapid elevations in intra-abdominal pressure and results in adverse effects on multiple organ systems.[2] Any insult that causes an acute increase in intra-abdominal volume can trigger ACS, including trauma to the abdomen as well as to distant sites, pancreatitis, hemorrhage, ruptured abdominal aortic aneu-

rysm, massive fluid resuscitation, and burns.[2–4] In the setting of massive fluid resuscitation, the pathophysiology is thought to involve resuscitation-induced bowel edema as well as ischemia and reperfusion injury.[1] Ultimately, the abdominal expansion exceeds the limited ability of this compartment to distend to accommodate it, which results in intra-abdominal hypertension.[4,5] This causes multiple physiologic derangements, including hemodynamic compromise and decreased cardiac output, renal impairment, and respiratory failure.[2,4,6] Even with early recognition and aggressive surgical management, including surgical decompression, ACS has a high mortality rate, particularly in non-trauma patients.[1]

ACS is known to cause respiratory compromise, which is generally treated with surgical decompression.[1] We report a case of ACS that developed in a patient following massive volume resuscitation for portal hypertension and variceal bleeding for which surgical decompression was not an option, because of severe coagulopathy and hemodynamic instability. Respiratory compromise in both ox-

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ygenation and ventilation was corrected using high levels of positive end-expiratory pressure (PEEP) to overcome extrinsic pulmonary limitations caused by ACS.

Case Summary

A 35-year-old man with a history of heavy ethanol use was admitted to the hospital with hematemesis. He had been well until 2 days prior to admission, when he had gradually increasing fatigue, malaise, and nausea. One day prior to admission he had mild, intermittent hematemesis. On the day of admission he suddenly developed large amounts of hematemesis as well as melenic stools. In the emergency department he was intubated for massive hematemesis and airway compromise. On admission, he was afebrile but tachycardic, with a heart rate of 144 beats/min. His blood pressure was 100/34 mm Hg. His initial empirical ventilator settings were intermittent mandatory ventilation rate 14 breaths/min, PEEP 5 cm H_2O, fraction of inspired oxygen (F_{IO_2}) 0.50, and tidal volume 600 mL, corresponding to 6.3 mL/kg. His physical examination was remarkable for tachycardia. His abdomen was soft and non-distended, without hepatomegaly. Neurologic exam was not obtained, because paralytic agents had been administered prior to endotracheal intubation. The remainder of his physical examination was unremarkable. His initial hematocrit was 10%, with an international normalized ratio of 3.4. His initial arterial blood gas values, obtained while on 100% oxygen via face mask, were pH 6.76, P_{aO_2} 465 mm Hg, P_{aCO_2} 35 mm Hg, and bicarbonate 5 mEq/L. The anion gap was 22 mmol/L.

In the emergency department 5 L of lactated Ringer's solution was rapidly infused, concurrent with 4 units of packed red blood cells and 6 units of fresh frozen plasma to attempt to correct his coagulopathy. Octreotide and pantoprazole drips were initiated. He was brought to the medical intensive care unit and underwent emergency endoscopy, which revealed 4+ esophageal varices, one of which was actively bleeding. Attempts were made at sclerosing with sodium morrhuate and banding, but without substantial impact on the bleeding. Resuscitative efforts continued during this time, with an additional 5 L of normal saline. In the course of resuscitation and endoscopy the patient developed increasing anasarca as well as gradually worsening hypoxemia that initially responded to increasing the F_{IO_2} to 1.0. Arterial blood gas values at that time were pH 6.87, P_{O_2} 91 mm Hg, P_{CO_2} 23 mm Hg, and bicarbonate 16 mEq/L. However, the hypoxemia progressed, with desaturations to 32% (measured via pulse oximetry) despite continued 100% oxygen. He remained tachycardic, with a heart rate of 113 beats/min, and was now hypertensive, with a blood pressure of 205/158 mm Hg. His physical examination was then notable for cyanosis and diffuse edema, with a firm, tympanitic abdomen. A chest radio-

graph revealed low lung volumes and dense bilateral parenchymal opacities (Fig. 1A). He continued to have massive hematemesis. Further attempts at endoscopic control of bleeding were aborted and attempts were made to place a Sengstaken-Blakemore tube, without success, because of severe esophageal and oropharyngeal edema. Surgical consultation was obtained for ACS and PEEP was increased to 20 cm H_2O, which increased blood oxygen saturation to 40%. Bilateral chest tubes were placed, with difficulty, and without further oxygenation improvement. He continued to have increasing abdominal distention and rigidity. Because of his unstable hemodynamic situation and coagulopathy, decompressive surgery was not possible. PEEP was gradually increased from 20–50 cm H_2O over 45 min, and there was a gradual increase in his blood oxygen saturation, from 40% to 91%. Over the same period his peak and plateau inspiratory pressures were increased to a maximum of 100 and 80 cm H_2O, respectively (see Table 1). Concurrent with endoscopy and attempts at improving ventilation and oxygenation, a further 5 L of lactated Ringer's
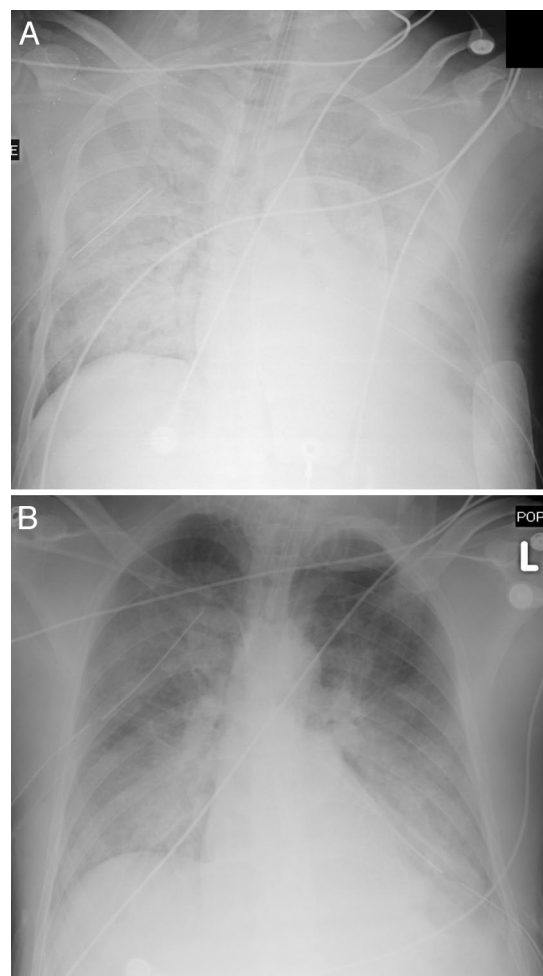

Figure 1 Chest radiographs (A) at admission and (B) on the day after admission.

Table 1. Improved Oxygenation and Ventilation With High Positive End-Expiratory Pressure in a Case of Abdominal Compartment Syndrome Causing Severe Hypoxemia

Time	F_{IO_2}	PEEP (cm H_2O)	pH	P_{aCO_2} (mm Hg)	P_{aO_2} (mm Hg)	HCO_3^- (mEq/L)	S_{PO_2}	Blood Pressure (mm Hg)	Peak Inspiratory Pressure (cm H_2O)	Plateau Inspiratory Pressure (cm H_2O)
16:00	50	5	6.94	29	425	6	100	160/84	55	NT
16:43	100	5	6.95	59	67	12	100	167/98	NT	NT
17:30	100	20	6.87	91	23	16	40	191/136	NT	NT
17:35	100	30	NT	NT	NT	NT	53	184/126	NT	NT
17:40	100	35	6.91	87	47	17	68	NT	NT	NT
17:45	100	40	6.98	73	55	16	81	175/121	NT	NT
17:50	100	45	NT	NT	NT	NT	83	NT	100	80
18:17	100	50	7.18	47	79	17	91	135/94	NT	NT
20:00	100	45	7.25	40	211	17	100	127/88	92	78
21:00	100	40	7.29	37	350	17	100	124/76	NT	NT
22:00	100	35	7.31	36	295	18	100	108/74	NT	NT
23:00	100	30	7.33	36	301	19	100	118/72	NT	NT
00:00	100	25	7.36	36	237	20	100	120/69	53	40
02:00	100	20	7.38	35	115	20	100	127/69	NT	NT
10:00	50	20	7.41	33	65	21	97	138/76	NT	NT
12:00	50	15	7.42	33	72	21	93	124/70	NT	NT
18:00	40	10	7.45	33	59	23	95	143/77	NT	NT
04:00	50	10	7.54	26	93	23	94	124/72	47	34
11:00	50	10	7.44	37	108	25	97	128/76	50	32
14:00	30	5	7.44	37	76	25	96	122/72	50	26

F_{IO_2} = fraction of inspired oxygen
PEEP = positive end-expiratory pressure
S_{PO_2} = blood oxygen saturation measured via pulse oximetry
NT = measurement not taken

solution, 4 units of packed red blood cells, and 6 units of fresh frozen plasma were infused while he was in the medical intensive care unit. One hour after increasing the PEEP to 50 cm H_2O his blood oxygen saturation increased to 100%. He remained tachycardic, with a heart rate of 120 beats/min, but normotensive at 136/91 mm Hg, with no further hematemesis. Arterial blood gas values showed marked oxygenation improvement: pH 7.25, P_{aCO_2} 40 mm Hg, P_{aO_2} 211 mm Hg, and bicarbonate 17 mEq/L. Despite the high PEEP level used to ventilate and oxygenate the patient, he remained hemodynamically stable, as reflected in serial blood pressure measurements (see Table 1). He remained diffusely edematous but with a less rigid abdomen. His oxygenation continued to improve, and over the next 24 hours PEEP was gradually decreased to 10 cm H_2O and F_{IO_2} was decreased to 0.4. His arterial blood gas values 1 day later were pH 7.45, P_{aCO_2} 33 mm Hg, P_{aO_2} 59 mm Hg, and bicarbonate 23 mEq/L while on PEEP 5 cm H_2O and F_{IO_2} 0.4. His hematocrit level was followed over the course of the day, and it remained stable at 43%.

A chest radiograph taken the day following admission showed decreased bilateral pulmonary parenchymal opacities (Fig. 1B).

Six days following admission the patient underwent transjugular intrahepatic portosystemic shunt placement, and he was extubated 5 days following that procedure.

Discussion

Increased intra-abdominal pressure results in respiratory compromise, primarily as a result of extraparenchymal restriction applied to the lungs. With increasing intra-abdominal pressure, upward displacement of the diaphragm compresses the lungs and worsens static and dynamic pulmonary compliance, decreasing total lung capacity.[4,5,7] In that situation higher airway pressures are needed to inflate the compressed alveoli and displace the same volume of inspired air.[7] Without the ability to do that, the net effect is increased ventilation of dead space and worsening hypoxemia and hypercarbia.[4,8,9]

The diagnosis of ACS is predominantly clinical: hypoxia, increasing airway pressures, tense abdominal distention, and progressive oliguria despite adequate cardiac output are sufficient findings to prompt abdominal decompression.[7] However, an adjunctive approach in-

volves measuring intra-abdominal pressure via a Foley catheter connected to a central venous pressure transducer or water manometer. Because the bladder acts as a passive diaphragm at volumes under 100 mL, it can transmit a wide range of intra-abdominal pressures without imparting further pressures through its own musculature.[2,5,7,8] Because the intra-abdominal pressure at which ACS occurs differs between patients, the diagnosis of ACS and the need for treatment is guided by the patient's clinical condition. Burch et al[7] proposed an ACS grading system that bases treatment on both intra-abdominal pressure and clinical findings. In that system, intra-abdominal pressure of < 15 mm Hg is considered grade I ACS, and grade I patients rarely need treatment. With grade II ACS (intra-abdominal pressure 16–25 mm Hg) the need for treatment should be guided by clinical findings. If there is no evidence of physiologic compromise (hypoxia, elevated airway pressures, oliguria), the patient can be managed with close monitoring alone. Most patients with grade III ACS (intra-abdominal pressure 26–35 mm Hg) will require surgical decompression, although physiologic changes may develop insidiously, leading to a delay in diagnosis and treatment. Almost all patients with grade IV ACS (intra-abdominal pressure > 35 mm Hg) are critically ill and require immediate treatment.[4,7,10]

The treatment of choice is decompressive celiotomy, whereby the abdomen is opened to relieve the ACS.[5] Although the timing of this intervention remains controversial most centers recommend abdominal decompression in the setting of definite organ failure, particularly with ventilatory insufficiency.[4,5]

An interesting aspect of our case was that ACS developed relatively quickly, in the midst of massive volume resuscitation, manifesting primarily as worsening hypoxemia and ventilatory failure. Because of the patient's cardiopulmonary instability and coagulopathy, decompressive celiotomy was not feasible. Instead, a very high level of PEEP was used to overcome the high pressure on the lungs from the distended abdomen and anasarca. With correction of the coagulopathy and stabilization of the bleeding, the patient's respiratory status rapidly improved, and he was extubated 12 days later.

One concern raised was the possibility of ventilator-induced lung injury as a result of using high-positive-pressure ventilation. However, the use of high PEEP in our case was to overcome the extrinsic pressure from the ACS and anasarca, so the high ventilatory pressure did not reflect the true transalveolar wall pressure. A useful analogy can be drawn from diving physiology. In an individual immersed to the neck in water but still breathing air, the external hydrostatic pressure on the thorax exceeds intrapulmonic (and intra-alveolar) pres-

sure by the weight of the column of water at any given point on the chest.[11,12] This decreases the compliance of the lungs and chest wall, which moderately decreases expiratory reserve volume, vital capacity, and functional residual capacity, which, in turn, increases work of breathing.[11,13,14] This hydrostatic imbalance can be countered with positive-pressure breathing, which normalizes these values and reduces work of breathing and dyspnea.[12,15,16]

Even with early intervention the case-fatality rate of ACS is high, particularly in non-trauma cases such as our patient.[1] That may be due to delayed recognition and treatment or, as with our patient, severe underlying illnesses that are not readily reversible. We do not suggest that high PEEP is necessarily going to improve survival or that high PEEP is better than surgical decompression. On the contrary, ventilation with high positive pressures may be harmful in the setting of acute lung injury and acute respiratory distress syndrome, as was demonstrated in the Acute Respiratory Distress Syndrome Network trial, in which higher tidal volumes were associated with higher mortality, presumably because of excessive alveolar stretch and/or injuriously high pressures to aerated lung.[17] Therefore we are not advocating high PEEP for all patients with hypoxemia and ACS, especially since many of the conditions that are implicated in the development of ACS may also precipitate acute lung injury and acute respiratory distress syndrome, and those conditions may co-exist.

In addition to the risk of ventilator-induced lung injury, high-pressure ventilation also increases the risk of hypotension by impairing venous return. However, our case suggests that such high PEEP may be a useful temporizing measure in unusual situations of ACS and life-threatening hypoxia under circumstances when surgical decompression is not an option.

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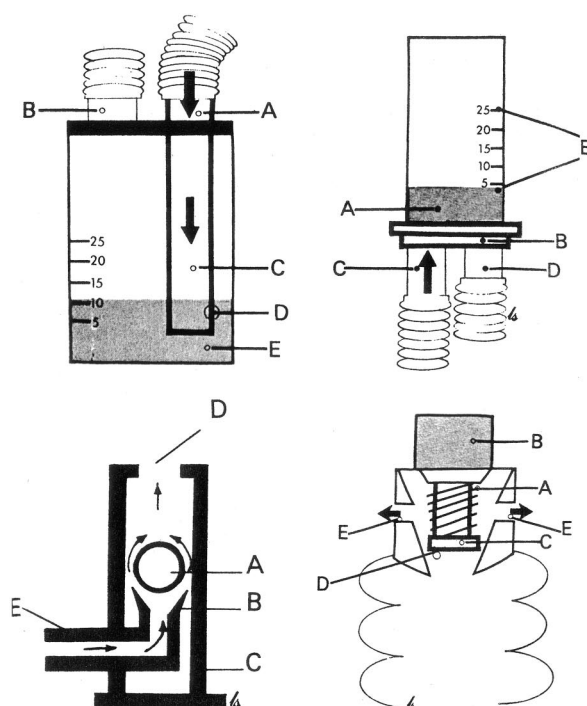
Devices for applying positive end-expiratory pressure (PEEP).
(Clockwise from upper left) : Underwater-seal PEEP system. Water-column
PEEP system. Weighed-ball PEEP valve. Spring-loaded PEEP valve.
From Kacmarek RM, Dimas S, Reynolds J, and Shapiro BA. "Techinal Aspects of
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