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CASE REPORT

Decreased cardiac index as an indicator of tension pneumothorax in the ventilated patient

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Summary

We describe three critically ill patients receiving pressure-controlled ventilation who suffered acute hypotensive episodes associated with the development of tension pneumothoraces. In four documented episodes of tension pneumothorax a major decrease in cardiac index was the most consistently detected abnormality. The expected increases in central venous pressure and heart rate did not occur in three of the episodes in two of the patients, both of whom were receiving inotropic therapy. Any increases in airway pressure could not be assessed on pressure-controlled ventilation. The physiology of tension pneumothorax in the ventilated patient is described and the importance of decreased cardiac index as a haemodynamic indicator of tension pneumothorax is discussed.

Key words

Complications; tension pneumothorax.

Tension pneumothorax is a life threatening condition, particularly in critically ill patients undergoing artificial ventilation who are at increased risk of barotrauma. Although standard texts describe the features of tension pneumothorax as decreased cardiac output and systemic arterial pressure with an increase in central venous and airway pressures [1, 2], animal experimentation has clearly demonstrated that these changes are not significant during spontaneous respiration [3, 4]. However, very little work has been performed in ventilated animals [4] and, due to the clinical urgency of the situation, haemodynamic data in patients whose lungs are being ventilated are extremely rare. We have encountered three such patients in whom life threatening hypotensive episodes developed as a consequence of tension pneumothorax. In all these patients an acute decrease in the cardiac index was the principal abnormality leading to the diagnosis.

Case histories

Case 1

A 33-year-old woman, weighing 134 kg, presented initially with acute abdomen pain. Abdominal ultrasound demonstrated a left-sided tubo-ovarian abscess which had ruptured into the pouch of Douglas. A posterior colpotomy was performed and the collection was drained. Two days later she became pyrexial with a temperature of 38°C, tachypnoeic with a respiratory rate of 40 breath.min^{-1}, hypotensive with a blood pressure of 90/60 mmHg and developed a tachycardia of 140 beat.min^{-1}. A repeat laparotomy was performed and the left ovary and Fallopian tube removed. Two litres of pus were drained from the patient's abdomen and she was admitted to the intensive care unit for postoperative care. Within 48 h, she developed septic shock and severe adult respiratory distress syndrome with a lung injury score of > 2.5 [5]. She had a pulmonary artery catheter inserted and cardiac output measured using a thermodilution technique. Her cardiovascular system was eventually stabilised by administration of dobutamine 15 μg.kg^{-1}.min^{-1} to maintain a mean arterial pressure of greater than 80 mmHg. Her ventilatory requirements had progressively increased over the preceding 24 h so that she was requiring pressure-controlled ventilation with a pressure of 30 cmH_2O, 10 cmH_2O of positive end expiratory pressure (PEEP), an inspiratory/expiratory (I:E) ratio of 1:1, a respiratory rate of 28 breath.min^{-1} and an inspired fraction of oxygen (F_{IO_2}) of 0.9. Over a 5 min period, the patient became severely hypotensive, the mean arterial pressure fell to 33 mmHg and the pulse oximeter failed to give a satisfactory

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Table 1. Haemodynamic changes in patient 1. Baseline measurements performed 2 h prior to deterioration.

	CI $l.min^{-1}.m^{-2}$	MAP mmHg	HR $beat.min^{-1}$	CVP mmHg	PAWP mmHg	SVR $dyne.s.cm^{-5}$	Pa_{O_2} kPa
Baseline	7.3	97	137	21	14	452	10.9
Immediately prior to chest drain insertion	3.0	33	72	16	12	246	6.7
Following thoracocentesis	5.13	54	115	18	20	287	10.7

CI, cardiac index; MAP, mean arterial pressure; HR, heart rate; CVP, central venous pressure; PAWP, pulmonary artery wedge pressure; SVR, systemic vascular resistance; Pa_{O_2}, arterial oxygen concentration.

trace. Any possible clinical signs of a pneumothorax were obscured by the patient's size and her diffuse lung disease. Haemodynamic and arterial blood gas measurements are shown in Table 1. A decision was made to insert bilateral chest drains on the basis of the haemodynamic changes. Chest drain insertion on the left side resulted in a large gush of air and the patient's haemodynamic status rapidly improved (Table 1). The patient survived for a further 3 days but subsequently died from unresponsive haemodynamic failure secondary to sepsis.

Case 2

A 46-year-old man was undergoing treatment on the intensive care unit for *P. falciparum* malaria and severe adult respiratory distress syndrome [5]. By the 9th day of treatment his condition had considerably improved such that he was no longer requiring inotropic support. His ventilatory requirements had also decreased and consisted of pressure-controlled ventilation with a pressure of 25 cmH_2O, an I:E ratio of 1:1, PEEP of 8 cmH_2O, a respiratory rate of 18 $breath.min^{-1}$ and F_{IO_2} of 0.6. He had sustained an iatrogenic pneumothorax 3 days previously during insertion of a subclavian central line and had a left-sided chest drain *in situ*. An acute deterioration in the patient's condition was marked by a sudden decrease in the mean arterial pressure from 95 mmHg to 60 mmHg. Physical examination demonstrated a slight decrease in breath sounds on the left but was not significantly changed from before. Oxygen saturation measurements revealed only a modest decrease from 97% to 94%. Haemodynamic measurements were performed (Table 2) and demonstrated a marked decrease in cardiac index. This prompted re-examination of the patient which revealed a kinked and obstructed chest drain on the left. Relief of the obstruction produced a large gush of air and the patient's mean arterial pressure, cardiac index and oxygen saturation returned to their previous levels (Table 2). The patient subsequently recovered and was discharged from hospital.

Case 3

A 53-year-old male pedestrian was admitted following a high speed motor vehicle accident. He had a left-sided flail segment of the chest wall with posterior fractures of the first to seventh ribs. He had fractures of the first and second ribs on the right side and had a right-sided chest drain *in situ* for a tension pneumothorax. He had also suffered a severe degloving injury to his right calf and had a stable fracture of the transverse process of the seventh cervical vertebra. A laparotomy was performed which proved negative and an echocardiogram confirmed good left ventricular function with no evidence of aortic dissection. On admission to the intensive care unit he was treated with intermittent positive pressure ventilation (IPPV) and a pulmonary artery catheter was inserted. His haemodynamic parameters are given in Table 3. At this time he required an adrenaline infusion of 4.4 $\mu g.kg^{-1}.min^{-1}$ and a dobutamine infusion of 46 $\mu g.kg^{-1}.min^{-1}$ in order to maintain a mean arterial pressure > 80 mmHg. Over a 10 h period he remained hypoxaemic despite ventilation with pressure-controlled ventilation at a pressure of 36 cmH_2O, PEEP at 10 cmH_2O, an I:E ratio of 1:1 and F_{IO_2} of 1.0. After 10 h, he became markedly hypotensive with a mean arterial pressure of 46 mmHg. Haemodynamic data at this time (Table 3) demonstrated a marked decrease in cardiac index from 3.6 $l.min.^{-1}.m^{-2}$ to 2.1 $l.min^{-1}.m^{-2}$. Auscultation of his chest revealed no change from a previous examination performed 6 h earlier when poor air entry to both lung fields had been noted. Bilateral needle thoracocentesis was performed and a sudden gush of air from both cannulae confirmed the presence of bilateral tension pneumothoraces. Bilateral chest drains were inserted and the patient's arterial blood gas and haemodynamic parameters substantially improved.

Over a further 6 h period the patient again became hypotensive (mean arterial pressure of 57 mmHg) and had a further dramatic fall in cardiac index from 2.5 $l.min^{-1}.m^{-2}$ to 1.4 $l.min^{-1}.m^{-2}$. All the chest drains were

Table 2. Haemodynamic changes in patient 2. Baseline measurements 1.5 h prior to deterioration.

	CI $1\ min^{-1}m^{-2}$	MAP mmHg	HR $beat.\ min^{-1}$	CVP mmHg	PAWP mmHg	SVR $dyne.s.cm^{-5}$	MPAP mmHg	PVR $dyne.s.cm^{-5}$	Sa_{O_2} %
Baseline	4.8	96	133	13	14	737	37	204	97
Immediately prior to chest drain clearance	3.1	68	136	15	16	730	34	248	94
Following chest drain clearance	4.6	95	143	14	14	744	33	174	98

MPAP, mean pulmonary artery pressure; PVR, pulmonary vascular resistance; Sa_{O_2}, arterial oxygen saturation. Other abbreviations as for Table 1.

Table 3. Haemodynamic changes in patient 3. Baseline measurements 1 h prior to initial deterioration. Data prior to insertion of anterior chest drains obtained 6 h after insertion of previous bilateral chest drains.

	CI $l.min^{-1}.m^{-2}$	MAP mmHg	HR $beat.min^{-1}$	CVP mmHg	PAWP mmHg	SVR $dyne.s.cm^{-5}$	MPAP mmHg	PVR $dyne.s.cm^{-5}$	Pa_{O_2} kPa	Pa_{CO_2} kPa
Baseline	3.6	68	120	20	16	625	41	327	6.4	5.4
Immediately prior to chest drain insertion	2.1	46	103	22	22	548	45	525	6.2	5.7
Following bilateral chest drains	2.5	114	101	17	16	1802	37	390	13.4	4.3
Prior to insertion of anterior drains	1.4	57	140	22	14	1165	37	766	8.5	3.9
Following anterior chest drains	2.5	86	110	18	12	1264	30	334	14.0	3.3

Pa_{CO_2}: arterial concentration of carbon dioxide. Other abbreviations as for Tables 1 and 2.

freely swinging with respiration and no physical signs of a pneumothorax could be elicited. The patient's arterial blood gas values had remained relatively stable for 6 h. A 12 lead ECG showed left axis deviation but no signs of a pulmonary embolism. An antero-posterior chest X ray revealed no evidence of pneumothorax but in view of the low cardiac index a lateral chest X ray was performed which showed a large anterior pneumothorax. Bilateral anterior chest drains were inserted with an appreciable improvement in oxygenation and haemodynamic stability. However, the patient subsequently died 48 h later from unresponsive haemodynamic failure.

Discussion

Although pneumothorax constitutes one of the commonest life threatening emergencies in patients treated with IPPV, the haemodynamic effects remain poorly understood. The majority of available data are derived from animal experimentation and with one exception have described the physiological responses to pneumothorax seen during spontaneous breathing. Great care must be exercised in translating this animal data into human responses and, as our observations have shown, the majority of the physiological changes described in this animal work are absent in ventilated critically ill patients.

The belief that the mechanical effect of pneumothorax causes kinking and obstruction of mediastinal vessels was conclusively disproven by Rutherford *et al.* [3] who described the haemodynamic responses to induced pneumothoraces in spontaneously breathing Angora goats. These workers clearly demonstrated that the physiological features of developing pneumothorax during spontaneous ventilation are dominated by respiratory insufficiency. Compensatory hyperventilation maintains respiratory rate and minute volume although there is increasing hypoxaemia due to shunting of blood through the collapsed lung. As the presence of increasing amounts of intrapleural air decrease the tidal volume, this is compensated for by an increase in respiratory rate. With the onset of tension pneumothorax there is an inevitable decrease in minute volume resulting in increasing hypoxaemia and hypercapnea. Death in this study resulted from respiratory arrest, probably caused by hypoxaemia-induced depression of the central respiratory centre. Perhaps the most striking observation of these workers was the absence of significant haemodynamic effects even in the agonal stages of tension pneumothorax. Although there were significant increases in central venous pressure, right atrial pressure and pulmonary artery pressure, there was no significant variation in arterial blood pressure or cardiac output. This surprising observation reflected a further compensatory mechanism as increasing heart rate offset the effect of decreasing left ventricular stroke volume. These observations have been confirmed by Gustman *et al.* [4] who demonstrated that the maintenance of cardiac output, even in the presence of a large tension pneumothorax, results from three independent mechanisms. Firstly, the increase in intrapleural pressure is incompletely transmitted to the mediastinum as demonstrated by oesophageal manometry, secondly, a reflex baroreceptor-mediated tachycardia compensates for a decrease in ventricular stroke work index, and thirdly, an increase in negative intrathoracic inspiratory pressure increases venous return. These workers also demonstrated that the suppression of respiratory drive by hyperventilation abolished the increased swings in intrathoracic pressure resulting in decreased venous return and cardiac output. Studies on ventilated sheep clearly demonstrated that mechanical ventilation also results in loss of the negative intrathoracic pressure which forms the main compensatory mechanism in this model.

It is apparent from these animal models that even a patient whose lungs are adequately ventilated using IPPV is probably bereft of many of the natural compensatory mechanisms which are available during spontaneous respiration. Most importantly, the complete absence of negative intrathoracic pressures during the ventilatory cycle leads to decreased cardiac filling pressures and an inevitable decrease in cardiac output; the exact mechanisms for this decrease in cardiac filling pressures remains uncertain. Although the most commonly cited explanation is a decrease in venous return to the right atrium due to compression of the intrathoracic veins, the significance of this mechanism remains unproven. The available experimental data again relates to pneumothorax during spontaneous respiration in animals and demonstrates pressure increases of a similar magnitude throughout the right heart with no change in pressure gradients [3]. In the three patients we report here a significant increase in central venous pressure occurred in only one.

A second possible mechanism for the difference in the ventilated patient is that the effect of increasing intrapleural pressure decreases blood flow through the pulmonary circulation. West [6] defined three lung zones based on the relationship between alveolar pressure (PA), pulmonary arterial (Pa) and pulmonary venous pressures (Pv). In the dependent portions of the lung (Pa > Pv > PA, zone 3) pulmonary blood flow depends on the arterial venous pressure difference. The increase in PA in patients with tension pneumothorax can convert normal lung zone 3 to zone 2 (Pa > PA > Pv) where the pulmonary blood flow is dependent on the difference between arterial and alveolar pressures. Yu and Lee [7] have described evelation of the pulmonary artery diastolic pressure in a ventilated patient with a spontaneous tension pneumothorax and attributed the pressure change to this mechanism. The marked elevations in pulmonary vascular resistance recorded during the acute episodes in patients 2 and 3 would also support this theory. Further elevation of the intrapleural pressure and decrease in the pulmonary artery pressure could theoretically convert the affected lung to zone 1 (PA > Pa > Pv) where no blood flow is possible.

Whatever the exact mechanism of these haemodynamic changes, the decrease in cardiac output appears to be an inevitable concomitant of increasing tension pneumothorax in the ventilated patient and may provide the only indicator of the event.

Other commonly quoted indicators of tension pneumothorax include unilateral diminished breath sounds, and increased resonance to percussion on the affected side with displacement of the trachea and apex beat, increase in the central venous pressure, decreased oxygen saturation and increased airway pressures [1–3]. The signs on clinical examination can be absent even in patients with established tension pneumothorax [3, 7, 8] and mediastinal shift can be relatively minimal, particularly in adults where the mediastinal position is relatively fixed [3]. In the first case reported, new physical signs could not be elicited due to gross obesity whilst in the other cases the presence of known pneumothoraces and pulmonary injury made the physical signs unhelpful.

Increases in the central venous pressure can be minimal since the degree of mediastinal compression is far less than would be predicted from the increase in intrapleural pressure even in the spontaneously breathing patient [4]. In the critically ill patient the expected increase in central venous pressure may be absent due to other reasons such as sepsis, haemorrhage or the effects of inotropic therapy. In two of the four episodes reported here the central venous pressure remained constant or dropped. The expected increase occurred only in patient 2 who was previously haemodynamically stable without inotropic support and in the second episode in patient 3. In both these instances the elevation was small, illustrating the unreliability of central venous pressure measurements as an indicator of pneumothorax in the critically ill.

The vasomotor responses of increasing peripheral vascular resistance may also be impaired in the critically ill patient, particularly in the presence of severe sepsis and may be absent as we observed in patients 1 and 2 and in the first episode in patient 3 [3, 4, 9]. Severe illness and the use of inotropic therapy in these patients may also account for the loss of the expected chronotropic effect of tension pneumothorax which is believed to be mediated by an increase in sympathetic activity. The observation of increasing airway pressure, commonly the first and most suggestive indication of tension pneumothorax in ventilated patients, could not be appreciated in any of the present cases, all of whom were receiving pressure-controlled ventilation. The use of pressure-controlled ventilation in patients at risk of barotrauma will inevitably increase the number of patients developing tension pneumothorax in whom this valuable sign is not available.

Other haemodynamic variables are unlikely to provide any indication of the development of tension pneumothorax. Changes in the mean pulmonary artery pressure and pulmonary artery wedge pressure also appear to be nonspecific in this patient group and are unlikely to be sufficient to raise clinical suspicion. The decrease in mean arterial blood pressure, although invariable, is also nonspecific and may be partly compensated for in the acute phase by the reflex increase in systemic vascular resistance. The increase in diastolic pressures within the pulmonary artery described above can cause a distinctive although subtle change in the pulmonary artery pressure trace [8]. Other workers have also described a drop in the mixed venous oxygen concentration as an early indicator [7]. This presumably reflects the decreased oxygen delivery secondary to the decreased cardiac output and may provide a useful indicator where fibreoptic pulmonary catheters are available to give a constant readout of the mixed venous oxygen saturation.

In summary, the haemodynamic changes of tension pneumothorax in the critically ill patients whose lungs are being ventilated are complex and variable. Much of the commonly quoted data on the physiological response to pneumothorax are derived from experimental work on animal models during spontaneous respiration. The majority of these classical physiological responses are abolished by the use of artificial ventilation and others are confused by the abnormal physiological responses seen in critically ill patients. In ventilated patients most haemodynamic variables are unreliable and may be further complicated by the underlying clinical condition and treatment. Clinical indicators are also often unreliable in this patient group and increases in airway pressures cannot be appreciated in patients receiving pressure-controlled ventilation. Specific elevation of the pulmonary artery diastolic pressure and a decrease in mixed venous oxygen saturation should suggest a tension pneumothorax but these measurements may be unavailable or overlooked. The decrease in cardiac output is a striking and inevitable concomitant of tension pneumothorax in the ventilated patient and should be considered highly suggestive of this diagnosis. Although radiographic confirmation is highly desirable, the presence of an acute unexplained decrease in cardiac output in patients at high risk of barotrauma is sufficient to consider the possibility of a tension pneumothorax.

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