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Original Contributions

THE PATHOPHYSIOLOGY OF TENSION PNEUMOTHORAX IN VENTILATED SWINE

Erik D. Barton, MD, MS, Paul Rhee, BS, Kevin C. Hutton, MD, and Peter Rosen, MD

Department of Emergency Medicine, University of California, San Diego Medical Center, San Diego, California
Reprint Address: Erik D. Barton, MD, MS, Brigham and Women's Hospital, Department of Emergency Medicine—L2, 75 Francis Street, Boston, MA 02115

□ **Abstract—It remains unclear as to whether the cardiovascular collapse observed in tension pneumothorax (TP) is strictly a mechanical pressure-related phenomenon or secondary to hypoxemia. This study describes the pathophysiologic changes associated with a surgically induced progressive TP in a ventilated swine model. With a balloon occlusion catheter surgically placed into the pleural space, progressive volumes of pneumothorax were created in six anesthetized pigs on positive-pressure ventilation. Air was introduced into the right hemithorax in 100-mL increments every 4–5 min, with measurements of heart rate (HR), mean arterial pressure (MAP), central venous pressure (CVP), mean intrapleural pressure (MIP), oxygen saturation (O_2%), arterial blood gas (ABG), and cardiac output (C.O.). With the induced progressive TP, results showed that O_2% measures decreased immediately and continued to decline throughout the experiment to levels below 50% prior to cardiovascular collapse. The MAP and HR remained relatively stable until approximately 57% total lung capacity progressive TP (600 mL) was reached. At this point, a significant decline in MAP and increase in HR was noted, indicating tension physiology. The C.O. showed a small but significant decrease after 200 mL of air was injected, with a progressive decline after this point. At >97% total lung capacity TP, lethal cardiovascular collapse occurred in all animals and was associated with an abrupt drop in C.O., HR, and MAP. There was a concurrent equalization of MIP with CVP at the point of collapse. Arterial blood gas measures correlated with O_2% trends during the trials. We conclude that the findings of this study support the alternative hypothesis that significant hypoxemia occurs early and precedes hypotension in ventilated animals with TP. Occlusive mechanical compression, suggested by equalization of MIP and CVP, is probably a late event.** © **1997 Elsevier Science Inc.**

□ **Keywords—tension pneumothorax; pneumothorax; chest trauma; positive-pressure ventilation; tension physiology; swine; hypotension; oxygen saturation**

INTRODUCTION

Tension pneumothorax (TP) is a serious pathophysiologic state that can develop in patients with thoracic trauma or during treatment with positive-pressure ventilation. The treatment of TP requires early recognition of clinical signs and symptoms and rapid decompression to avoid physiologic decompensation. It remains unclear, however, whether cardiovascular collapse is due to a direct compressive effect on central venous structures or secondary to the rapidly progressive hypoxemia with shunting caused by lung parenchymal collapse. The prevailing teaching that persists in most textbooks and training programs relies on the mechanical compression theory (1–3). This hypothesis, however, resulted from initial studies using a canine model (4). More recently, animal studies utilizing spontaneously respiring goats and monkeys (5) and sheep (6) support the hypothesis that central hypoxemia is the primary factor in the lethality of TP. These studies demonstrate that a variety of compensatory mechanisms help to maintain cardiac output while respiratory

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failure progresses. Very little work, however, has been published characterizing the effects of TP in ventilated patients in which compensatory respiratory mechanisms are absent.

This study describes the pathologic events of progressive TP in animals on positive-pressure ventilation. We have developed a swine model to assess the relationship between increasing intrapleural space air and positive-pressure ventilation on oxygenation and hemodynamics.

MATERIALS AND METHODS

Animals

Six domestic female swine weighing 20 kg (range = 17–21 kg, mean = 19.3 kg, SD = 0.82 kg) obtained from S & S Farms (Ranchida, CA) were used in this study. All animals were quarantined for a period of 3–6 d at the UCSD Medical Center Surgical Research Laboratory Vivarium and fasted for 12 h prior to each experiment. Animal use approval was obtained through UCSD Animal Subjects Committee.

Sedation and Anesthesia

Each animal was initially sedated using ketamine (20 mg/kg), xylazine (2 mg/kg), and atropine (0.5 mg/kg) by intramuscular injection. Ear vein access was then established for continuous normal saline infusion. Sodium thiopental at 25mg/kg was administered intravenously (IV), and the animal was placed in a supine position. Vocal cords were topically anesthetized with Benzocaine spray, and the animal was orally intubated using a 4.0 cuffed endotracheal tube. Heart rate (HR) by electrocardiography (ECG) and ear pulse oximetry (O_2%) were continuously monitored using a Propaq 106 Multifunctional Monitor (Prolocol, Benenton, OR). Tube position was confirmed by auscultation and end-tidal CO_2 ($ETCO_2$) monitoring (Sara Cap, Lenexa, KS). Total lung capacity (TLC) was estimated at 55 mL/kg (7). Each pig was connected to a large animal volume cycle ventilator (Harvard Apparatus, South Natick, MA) with a tidal volume of 15 cc/kg on room air (FiO_2 0.21) at a rate of 20 breaths per minute. Continuous anesthesia was maintained with alpha-chloralose (60 mg/kg IV bolus followed by 10 mg/kg/h infusion). Alpha-chloralose was selected because this agent has been shown to preserve cardiovascular reflexes in previous animal studies (8).

Surgical Procedures

A 5-French pediatric Swan-Ganz catheter with cardiac output (C.O.) capability was placed in the femoral vein and positioned by waveform analysis for measurements of central venous pressure (CVP) using a Grass Recorder. Cardiac output was measured by thermodilution saline infusion with a standard C.O. computer (American Edwards Laboratories, Irvine, CA). Femoral arterial cannulation was established for mean arterial blood pressure (MAP) and arterial blood gas (ABG) measurements. A superpubic urinary catheter was placed for urine output, and core temperature was monitored continuously by rectal probe. A 7-French balloon occlusion catheter (Boston Scientific Corp., Spencer, IN) was placed into the right pleural space by using a semi-open technique by blunt dissection at the fifth intercostal space anterior axillary line. The catheter was sutured in place to the chest wall, closing the surgical incision, and tunneled subcutaneously to penetrate the skin 5–6 cm distant from the chest wall site. The occlusion balloon was inflated and the catheter pulled taut against the chest wall to create an airtight system. This catheter was used to inject air by using a 50-mL measured syringe and to determine the mean intrapleural pressure (MIP) by using a Grass Recorder. In this manner, a reproducible, stable, and progressive pneumothorax could be established with complete evacuation of injected volumes. The right hemithorax was chosen to maximize the compressive effects of TP on central venous structures. The first two animals in the series had balloon occlusion catheters placed into the left intrapleural space to ensure there was no leakage of TP across the mediastinum.

Experimental Protocol

Baseline physiologic measures of HR, O_2%, $ETCO_2$, MAP, CVP, MIP, and C.O. were obtained after 15–30 min of stabilization prior to development of pneumothorax. Air was injected in 100-mL aliquots over a 10–15-s period with a measured syringe into the right intrapleural catheter at 4–5-min intervals with repeat physiologic measures taken at 1 and 3 min after injection. All C.O. measures were repeated 2–3 times for reproducibility. The experiment continued until cardiovascular collapse occurred or an injected volume of >120% of TLC (ca. 1300 mL) was obtained. Four animals had their TP evacuated with a syringe, and the volumes were measured. Two of the animals had ABG measurements done during the progressive TP by using a standard blood gas monitor (Corning, Medfield, MA) to assess correlation with O_2% measures. All pigs were

sacrificed using Beuthanasia-D Special Solution (1 cc/kg IV) at the end of the experiment.

Statistical Analysis

Results are shown as mean values with standard deviation (SD) shown in parentheses and illustrated by error bars on the figures. The C.O. for each animal was determined by using the mean of 2–3 reproduceable measurements at each stage of progressive TP. Values at successive stages of the experiment were compared with baseline by using paired *t*-tests and one-way analysis of variance (ANOVA). Statistical significance was determined as $p < 0.05$.

RESULTS

Table 1 summarizes the mean hemodynamic and O_2% data from six animals. All animals were able to tolerate pneumothorax volumes of up to 900 mL of air injected into the intrapleural space. The data from the 1000-mL stage (average 94% TLC) are from only three animals due to the rapid decompensation in hemodynamics demonstrated by the pigs at this degree of TP. In addition, O_2% measurements by ear oximetry at levels <50% were problematic and only obtainable in three of the animals during the 900-mL stage of TP. No measurements of O_2% were possible at <40%, or ≥1000-mL stages of TP, thus considered negligible at this point. Only one animal achieved 1300 mL of injected air, and data measurements were unreliable at this stage. Four of the six pigs had their total pneumothorax volumes aspirated at the end of the experiment and compared with injected amounts prior to cardiac arrest. There was a mean of 1070 ± 170 mL (range = 900–1300 mL) injected and of 1060 ± 230 mL (range = 860–1385 mL) aspirated in these animals ($p = 0.49$, NS), demonstrating 99% air recovery using this model. The first two animals tested had left intrapleural catheters placed, and 0% of injected volumes were aspirated from this side during the experiment in both cases.

Figure 1 shows the hemodynamic effects of progressive TP. The HR and MAP remained relatively stable until a 500-mL (47% TLC) pneumothorax was established. After this point, HR was increased variably in animals ($p = 0.036$) until cardiovascular collapse at >900 mL (>86% TLC) of pneumothorax. MAP decreased significantly at 600 mL of pneumothorax ($p = 0.028$) and progressively decreased further with air injection until collapse occurred. Central venous pressure increased immediately and significantly with progressive TP until pressures of 15 torr were reached at 900 mL TP ($p = 1.57 \times 10^{-7}$). Similarly, MIP was significantly elevated above baseline levels (0 torr) with injections of 100-mL aliquots and progressed linearly throughout the experiment to a maximum of 15 torr ($p = 8.01 \times 10^{-21}$). C.O. fell significantly from baseline after 200 mL of air was injected, with a decline of 18% ($p = 0.032$), and then decreased more slowly until higher levels of TP were reached. Rapid cardiovascular collapse, signified by a marked decline in C.O. from 1.6 to 0.3 L/min, occurred at 1000 mL of pneumothorax (94% TLC). The point of collapse occurred concurrently with a fall in HR and MAP.

When the relationship among C.O., CVP, and MIP was plotted, CVP was maintained significantly above MIP during progressive TP ($p = 2.37 \times 10^{-6}$) (Figure 2). These pressures trended together at higher stages of TP until a rapid collapse of C.O. was noted at the

Table 1. Mean (±SD) Values at Each Stage of Progressive TP For Cardiac Output (C.O.), Heart Rate (HR), Mean Arterial Pressure (MAP), Central Venous Pressure (CVP), Mean Right Intrapleural Pressure (MIP), and Oxygen Saturation (O_2%) in Six Farm Animals. (Underlined Values Denote Significant Change From Baseline at $p < 0.05$; † Denotes Data Obtained From 3 Animals Only)

	C.O.	HR	MAP	CVP	MIP	O_2%
Baseline	2.8 (0.6)	122 (13)	93 (7)	5 (3)	0 (1)	97 (1)
100 ml	2.7 (0.7)	121 (25)	100 (14)	6 (3)	2 (1)	96 (2)
200 ml	2.3 (0.4)	119 (24)	98 (14)	6 (3)	3 (1)	92 (2)
300 ml	2.2 (0.3)	124 (23)	99 (16)	8 (3)	5 (1)	86 (4)
400 ml	2.2 (0.4)	128 (16)	93 (20)	9 (2)	6 (1)	82 (4)
500 ml	2.0 (0.3)	130 (15)	85 (6)	10 (2)	8 (2)	75 (9)
600 ml	1.9 (0.4)	146 (23)	80 (8)	11 (2)	9 (2)	65 (12)
700 ml	1.8 (0.4)	143 (12)	75 (12)	13 (3)	11 (2)	55 (9)
800 ml	1.6 (0.3)	141 (24)	71 (10)	14 (3)	12 (3)	54 (3)
900 ml	1.4 (0.4)	150 (42)	66 (14)	15 (5)	14 (4)	42† (2)
1000 ml†	0.3 (0.3)	118 (61)	48 (18)	15 (6)	15 (5)	— (—)
ANOVA *p* (*<0.05)	*<0.0001	0.39	*<0.0001	*<0.0001	*<0.0001	*<0.0001

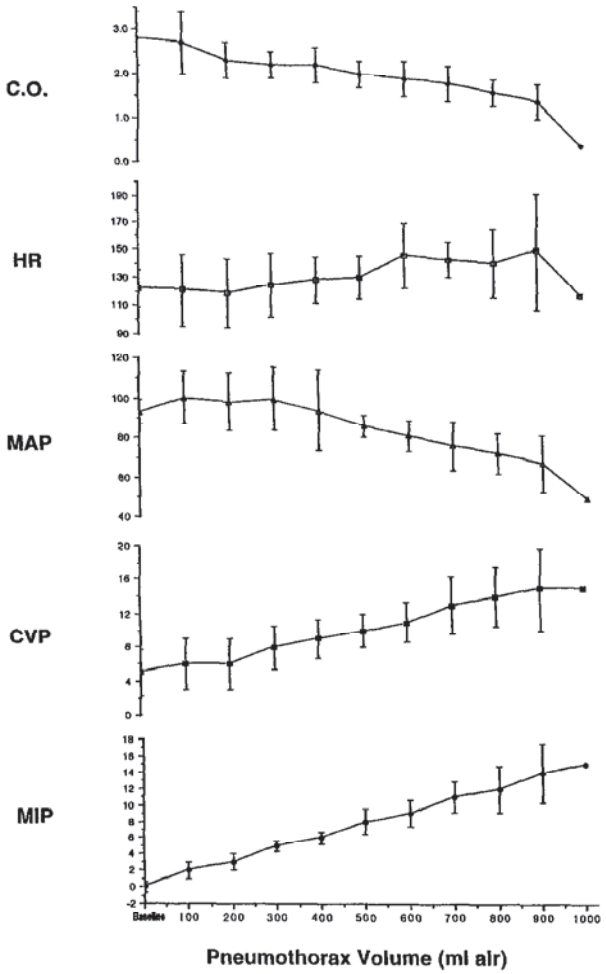

Figure 1. The hemodynamic effects of progressive TP on cardiac output (C.O.), heart rate (HR), mean arterial pressure (MAP), central venous pressure (CVP), and mean intraplueral pressure (MIP) in positive-pressure ventilated swine. Points with error bars indicate means ± SD; only three animals are included in the 1000-mL data point.

point of equalization of CVP and MIP at 15 torr (p = 0.74, NS).

Oxygen saturation showed an immediate and significant decline with increasing intrapleural space air ($p = 7.81 \times 10^{-23}$) (Figure 3). There was a significant drop from baseline with the first 100-mL bolus (p = 0.022) that progressed more dramatically as pneumothorax volume was increased. Oxygen saturation values of ≤50% were observed prior to complete cardiovascular collapse. Measurements below 40% were considered negligible and unobtainable at stages above 900 mL.

When C.O. was compared with MIP, a negative relationship was demonstrated (Figure 4). This relationship was illustrated by a slow decline in C.O. with increasing MIP, becoming significant at 3 torr (p = 0.032), and continuing over most of the range of increasing pressures. Cardiovacular collapse was demonstrated by a rapid decline in C.O. at MIP >14 torr.

RELATIONSHIP BETWEEN MIP, CVP AND C.O.

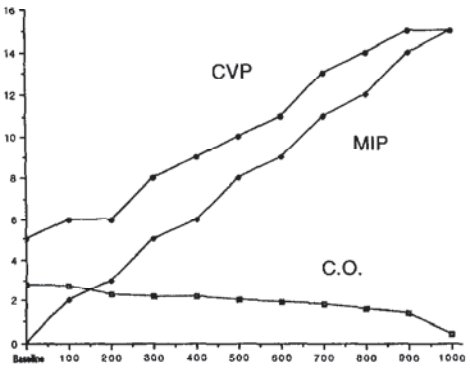

Figure 2. The relationship between MIP, CVP, and C.O. with progressive TP. Note the equalization of MIP and CVP at the point of collapse of C.O. (means ± SD).

Initially, $ETCO_2$ measures were taken on the first four animals tested. There was a trend toward CO_2 retention with progressive TP, especially with the higher pneumothorax volumes; however, these data were highly variable and changes were not significant (results not shown). Arterial blood gases were measured on the last two animals in the series (Figure 5). Arterial pH and PCO_2 remained relatively stable until >700 mL of pneumothorax but then pH fell more dramatically with a concomitant rise in CO_2. Arterial

OXYGEN SATURATION ON 0.21 FIO2

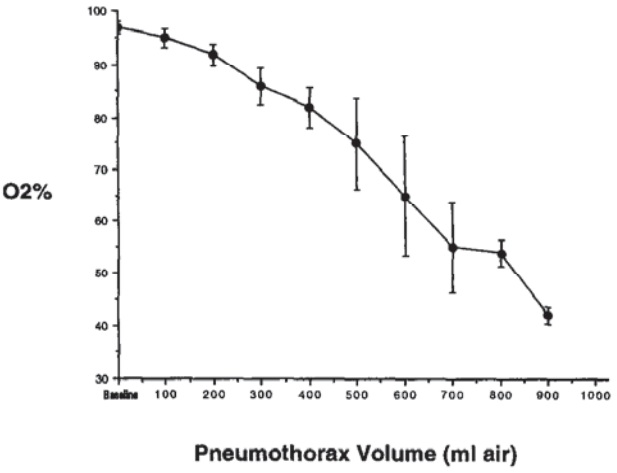

Figure 3. The effects of progressive TP on oxygen saturation (O_2%) in ventilated swine (means ± SD).

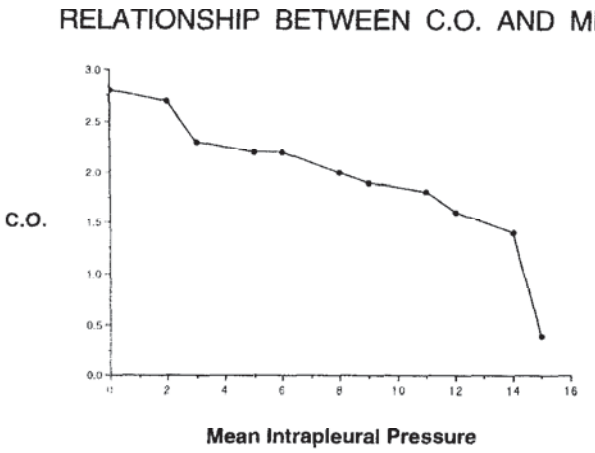

Figure 4. The relationship between C.O. and increasing MIP during progressive TP in ventilated swine.

PO_2 fell immediately with the development of TP and continued to decline throughout the experiment, corresponding to the O_2% data.

DISCUSSION

TP remains a life-threatening condition in the prehospital, emergency department, and critical care settings. Expedient and accurate recognition and treatment of this condition requires an understanding of the underlying pathophysiology involved. The recognition of "tension physiology," or hypotension and respiratory compromise in the presence of pneumothorax, has been promoted as the diagnostic indicator of TP (9). Experimentally, the definition of TP has been described as the state of intrapleural space pressures that are positive throughout the respiratory cycle in spontaneously breathing subjects, leading to cardiorespiratory compromise (5).

The proposed pathophysiology of TP is thought to be caused by mechanical compression or "kinking" of central venous and right heart structures, which results in cardiovascular collapse from occlusion of venous return to the heart, with a disastrous fall in C.O. Initial studies using dogs have supported this hypothesis (4). It is now known that the mediastinums in these animals, unlike in humans, are more mobile and are fenestrated to allow communication of air from one hemithorax to the other (4,10). Increased intrapleural pressures may be transmitted more directly to central structures in dogs to compromise cardiovascular function.

Subsequent studies using spontaneously breathing animals with mediastinums more similar to man have demonstrated a substantial hypoxemia with the development of TP in the absence of significant compromise in arterial pressures (5,6). The progressive hypoxemia observed is attributed primarily to increasing pulmonary vascular shunting associated with increasing parenchymal collapse. The following compensatory mechanisms may protect C.O. and MAP during TP:

1. incomplete transmission of increased intrapleural pressure to the mediastinum and contralateral pleural space;
2. increased respiratory effort and intrathoracic pressure fluctuations maintaining venous return; and
3. a baroreceptor-mediated reflex or catecholamine-induced tachycardia that compensates for a decrease in left ventricular stroke volume (6).

The present study addresses the effects of two extrinsic pressures: (a) increasing intrapleural pressure and (b) positive-pressure ventilation on hemodynamics and oxygenation. TP in patients on mechanical ventilation has been demonstrated to occur more abruptly and be associated with higher mortality (9). One series of 88 patients with 122 events of simple pneumothorax found that almost all patients who developed TP were on mechanical ventilation or had undergone cardiac resuscitation. In addition, the development of TP tended to occur in patients in whom the antecedent diagnosis of any pneumothorax had not been made

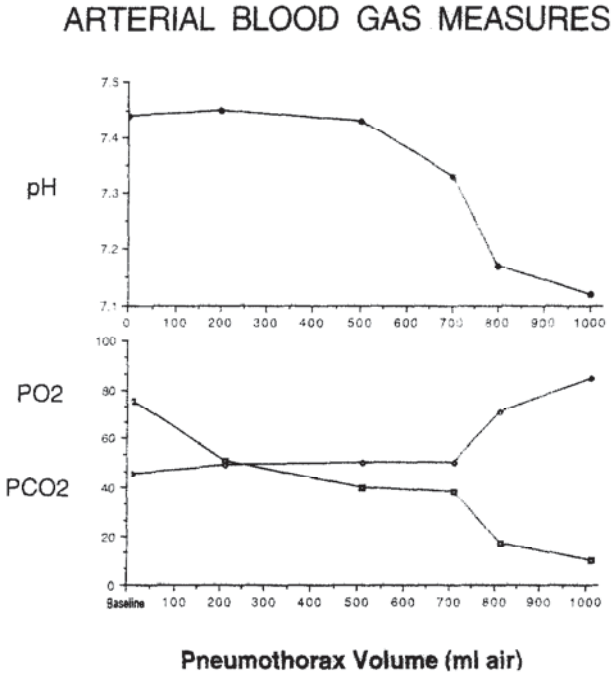

Figure 5. Averaged arterial blood gas measures of pH, PO_2, and PCO_2 during progressive TP in ventilated swine (n = 2).

(11). Previous studies in which hemodynamic measurements were taken during TP in human patients are, understandably, limited (12–16). One case report of three critically ill patients on positive-pressure ventilation demonstrated the initial presentation of TP as a decline in cardiac index (13). These investigators proposed that the absence of spontaneous respirations does not allow increased negative intrathoracic pressure fluctuations to act as a natural compensatory mechanism to preserve hemodynamics. Another case report also demonstrated concomitant decreases in cardiac output and mixed venous oxygen saturation as the dominant signs of TP in mechanically ventilated patients (14). Hemoglobin desaturation as measured by pulse oximetry was shown to be the earliest indication of TP in one ventilated patient (16).

We were able to induce a stable, progressive pneumothorax to tension levels by using a ventilated swine model. Farm pigs were chosen for the low cost and ease of handling, and because these animals have been demonstrated to be good models of human respiratory and cardiovascular systems (17). As seen with previous studies, these animals show an early and progressive hypoxemia associated with progressive TP. Both O_2 saturation and PO_2 fell immediately as air was introduced into the pleural space and continued to fall to levels well below 50% saturation before complete cardiovascular collapse (Figures 3 and 5). Conversely, MAP was maintained, and there was a significant but smaller decline in C.O. in the early stages of progressive TP that was not linear. Significant cardiovascular compromise, demonstrated by a drop in both C.O. and MAP and a significant increase in HR (Figure 1), did not occur until 600 mL of air had been injected (57% TLC). Complete collapse occurred at >900-mL volumes of pneumothorax when C.O. fell dramatically, concurrent with a terminal decline in HR and MAP and O_2% levels too low to measure. It was extremely difficult to record hemodynamic measures beyond this point due to the rapid demise of the animals.

It is interesting to note that CVP was maintained above MIP with progressive pneumothorax volumes until >900 mL (>85% TLC) of air was injected (Figure 2). These data demonstrate an augmentation of CVP with increasing MIP to preserve venous return. Complete collapse occurred when MIP equalized with CVP, which may indicate the critical point at which intrathoracic pressures overcome mediastinal forces to cause occlusion or "kinking" of the vena cava. This point, however, does not occur until a large pneumothorax, with volumes as large as 90–100% TLC, and severe hypoxemia are present. Rapid demise of the animals was imminent unless evacuation of the TP was immediately performed.

The combined effect of positive-pressure ventilation with TP would be expected to cause earlier mechanical compression and hemodynamic instability when compared with a spontaneously breathing subject. Increased compensatory negative thoracic space pressures during inspiration do not occur in this state, which may worsen extrinsic compromise of venous return. In this study, a progressive decline was demonstrated in C.O. as MIP increased until collapse was observed at >14 torr of pressure (Figure 4). Thus, our data suggest that some transmission of increased intrapleural pressure in ventilated animals inhibits venous return during the earlier stages of the experiment. This conclusion, however, is confounded by the rapidly progressive hypoxemia induced in these animals, which will also affect cardiac function.

LIMITATIONS AND FUTURE QUESTIONS

Although this study attempted to delineate the physiologic response to a progressive PT to tension, there are some obvious limitations with our model. First, the progression of increasing TP was determined based on the need for multiple physiologic measurements at each successive stage over a limited time period. We chose a 4–5-min interval between stages to obtain all the measurements with duplication if necessary. Obviously, the development of TP can occur with any number of temporal scenarios.

Second, there was a variety of physical and compensatory mechanisms that were not investigated in our model. Factors such as degree of thoracic injury, integrity of chest wall, patterns of respiration, and level of consciousness were not considered. Ventilatory settings were unchanged throughout the experiment, which undoubtedly increased the pressure effect of the progressive TP. It is impossible to reproduce all factors involved in the variety of conditions that can produce a TP, and we wanted to delineate the physiologic response to one scenario. We also assumed that natural cardiac compensatory mechanisms were intact with this model. In addition, lung functioning was normal prior to initiation of the experiment in these animals. Critically ill patients on ventilator support often exhibit alterations in sympathetic tone and cardiac response and may have lung diseases with compromised function or compliance. Such factors would affect the physiologic response to TP.

Another limitation may be the number of animals used. We chose six animals for a single TP protocol and for comparison of the baseline measurements with the experimental controls. All our animals demon-

strated a similar physiologic response, and significance was proved with paired *t*-tests and ANOVA.

We chose pulse oximetery in this experiment to follow changes in animal oxygenation, because this measure has been shown to accurately reflect changes in hemoglobin oxygenation (16). ABG measurements were cumbersome to coordinate and unavailable for the majority of our trials in the animal laboratory. The validity of oximetry is questionable at the lower levels measured, although our results were reproducible across trials.

One of the most important limitations was the decision to develop the TP model and measure the change from a baseline of ambient oxygen concentration (room air at sea level, 0.21% FIO_2). These conditons do not typically reflect patients being treated with ventilatory support in intensive care settings. When two animals were placed on 100% oxygen in secondary trials, there was a delay in the development of hypoxemia. However, the animals were highly unstable when attempts were made to perform additional TP trials, and results were unreliable (data not shown). Higher ventilatory oxygen tensions may delay the hypoxemic contributions to cardiovascular collapse. Further analysis under a variety of ventilatory settings would better define these relationships.

CONCLUSIONS

We describe the pathophysiologic changes associated with progressive TP in a positive-pressure room-air-ventilated swine model. From these data the following conclusions were drawn:

1. There is an early hypoxemia induced with progressive TP that falls to levels <50% O_2% before complete cardiovascular collapse is seen.
2. There is a smaller but significant decrement in C.O. throughout earlier stages of progressive PT that is not associated with a fall in MAP.
3. A significant decline in MAP is seen at higher volumes of TP (56% TLC).
4. Complete cardiovascular collapse does not occur until 1000 mL of progressive PT (94% TLC) is reached, and it is associated with equalization of MIP and CVP and severe hypoxemia.
5. Increasing MIP shows an early negative association with C.O. until a critical point is reached, at which time rapid collapse is observed.

Therefore, it remains unclear at what stage mechanical compressive forces become significant as progressive TP is heralded by significant hypoxemia in a ventilated model. Occlusive compression is suggested by equalization of MIP and CVP and may be a terminal event.

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