

Cardiopulmonary Effects of an Anterior Mediastinal Mass in Dogs Anesthetized with Halothane

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The authors evaluated the cardiac effects of an anterior mediastinal mass to better understand the acute cardiovascular collapse that has been associated with anesthesia and positive-pressure ventilation. An 800-ml-capacity intravenous bag was placed within the anterior mediastinum of 12 dogs to simulate a mediastinal mass. After mediastinal mass inflation, the authors measured cardiac index (CI) during periods of spontaneous ventilation (SV), SV with added continuous positive airway pressure (CPAP), intermittent positive-pressure ventilation (IPPV), and continuous positive-pressure ventilation (CPPV). Similar mediastinal mass volumes resulted in similar decreases in CI during SV (169 ± 51 to 105 ± 10 ml · kg⁻¹ · min⁻¹); CPAP (175 ± 48 to 122 ± 34 ml · kg⁻¹ · min⁻¹); IPPV (151 ± 15 to 93 ± 24 ml · kg⁻¹ · min⁻¹); and CPPV (183 ± 56 to 117 ± 46 ml · kg⁻¹ · min⁻¹). The authors also found, by linear regression, that the relationship between CI and mass volume was similar during both SV and IPPV. In six dogs, transesophageal echocardiography (TEE) was used to measure ventricular short axis dimensions. The authors found that mass inflation caused left ventricular end-diastolic dimension to decrease significantly by 6 ± 2 mm and 4 ± 1 mm during SV or IPPV, respectively, and right ventricle dimensions to increase by 2 ± 1 mm and 3 ± 1 mm during SV or IPPV, respectively. The changes in chamber dimensions were similar with either SV or IPPV. These results suggest that the decrease in CI associated with a mediastinal mass results from an increase in right ventricular afterload, causing right ventricular enlargement. Subsequently, there is impingement on the left ventricle volume because of interventricular interdependence. (Key words: Anesthesia; thoracic. Complications: cardiac. Heart: compression. Lung: pulmonary artery compression.)

ANESTHESIA for biopsy or excision of an anterior mediastinal mass has been associated with major airway and cardiac complications.¹ Operative mishaps have been described as resulting from extrinsic compression of the trachea after the patient becomes supine or with induction of and emergence from anesthesia.²⁻⁵ There have also been cases of cardiovascular collapse on induction of anesthesia without evidence of tracheal obstruction or impaired ventilation. These incidents have been restricted to isolated case reports and have included speculation that pulmonary artery occlusion⁶ or encasement by the ex-

trinsic mass⁷ may have resulted in right ventricular outflow obstruction. Three case reports⁸⁻¹⁰ have included deaths during anesthesia, after which cardiac or pulmonary artery compression was demonstrated on necropsy. Hypoxemia and hypotension during anesthesia were reported during spontaneous ventilation^{9,10} or assisted ventilation with or without muscle relaxation.^{7,8}

There have been recommendations that an attempt to shrink the mass by treatment with steroids or irradiation should be considered before surgical exploration if right ventricular outflow obstruction is suspected.⁸ Other recommendations have included the initiation of extracorporeal oxygenation before the induction of anesthesia.⁶ Because these recommendations were often based on experience with only a few patients, we wanted to more rigorously explore the anesthetic problems associated with a large mediastinal mass.

We developed an animal model of a mediastinal mass to more accurately define its effects on cardiac performance. We speculated that compression of the pulmonary artery by an extrinsic mass could cause right ventricular distension; impair cardiac performance; and, through the phenomenon of ventricular interdependence, limit left ventricular volume. The phenomenon of ventricular interdependence has been used previously¹¹ to explain some of the cardiac effects of positive end-expiratory pressure (PEEP). We speculated that right ventricular failure could be solely responsible for the hypotension and hypoxemia occurring in symptomatic patients with an anterior mediastinal mass even in the absence of tracheal obstruction. We also wanted to evaluate the interactions of ventilation mode with the mediastinal mass. We speculated that spontaneous ventilation might affect right ventricular performance differently than does mechanical ventilation. Similarly, continuous positive airway pressure (CPAP) during spontaneous ventilation or PEEP during mechanical ventilation could alter the vascular compression by the mediastinal mass. Therefore, using this model, we also evaluated whether CPAP or PEEP has a role in the management of the cardiovascular and gas exchange compromise.

Materials and Methods

ANIMAL PREPARATION

Twelve mongrel dogs (20–30 kg) were anesthetized with sodium thiopental (20 mg/kg) and pentobarbital (15

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mg/kg). The study protocol followed guidelines established by the University Animal Care Committee. The trachea was intubated with a 9-mm tube positioned above the carina. The animals' lungs were ventilated with a Bennett MA2 ventilator with 100% oxygen, tidal volume (V_T) of 20 ml/kg and frequency of 18 breaths per min.

During surgical preparation, anesthesia was maintained with 1% halothane and 65 mg pentobarbital every 30 min. A catheter, inserted through the femoral vein, was positioned in the right atrium. The catheter was advanced into the right ventricle with the use of continuous pressure monitoring and then pulled back into the atrium, thus ensuring proper position. The right atrial catheter was used to measure right atrial pressure (RAP) and administer fluids and drugs. A catheter was inserted through the femoral artery into the ascending aorta and was used to measure systemic arterial pressure (SAP) and for withdrawal of blood for arterial blood gas analysis. A pulmonary artery thermodilution catheter was inserted through the right external jugular vein and positioned in the pulmonary artery under continuous pressure measurement. This was used to measure pulmonary artery pressure (PAP) and pulmonary capillary wedge pressure (PAOP) and for withdrawal of blood for mixed venous blood gas analysis. This catheter was also used to inject cold saline (0° C) for thermodilution measurement of cardiac output (CO) by a cardiac output computer (Edwards Model 9520A). All three catheters were connected to pressure transducers (Statham) with the use of low-compliance tubing. After catheter insertions, heparin (5,000 U) was administered and 1,000 U was administered hourly.

Airway pressure was measured at the level of the carina (Pac) and at the airway opening (Pao). Pao was measured at a port on the distal end of the endotracheal tube. Pac was measured by inserting a low-compliance catheter (1 mm diameter) through the endotracheal tube and advancing the catheter until resistance was met. The catheter was then withdrawn by 1 cm. The position of the catheter was verified to be below the level of the carina at the time of thoracotomy (see below). Both Pao and Pac were measured with the use of an air phase transducer (Validyne®). The endotracheal tube was connected to a breathing circuit with unidirectional valves in both the inspiratory and expiratory limbs of the circuit. Fresh gas flow (100% oxygen) was set at 8 l/min, and a reservoir bag (6 l) and Ohio vaporizer were attached to the inspiratory circuit. When required by the experimental protocol, a mechanical ventilator (MA2) was attached at the site of the reservoir bag. The expiratory circuit was connected to a water spirometer (Tissot spirometer), and the minute ventilation (\dot{V}_E) was measured as a timed (1 min) volume collected in the water spirometer. During spontaneous ventilation, the fresh gas flow was stopped and the expired

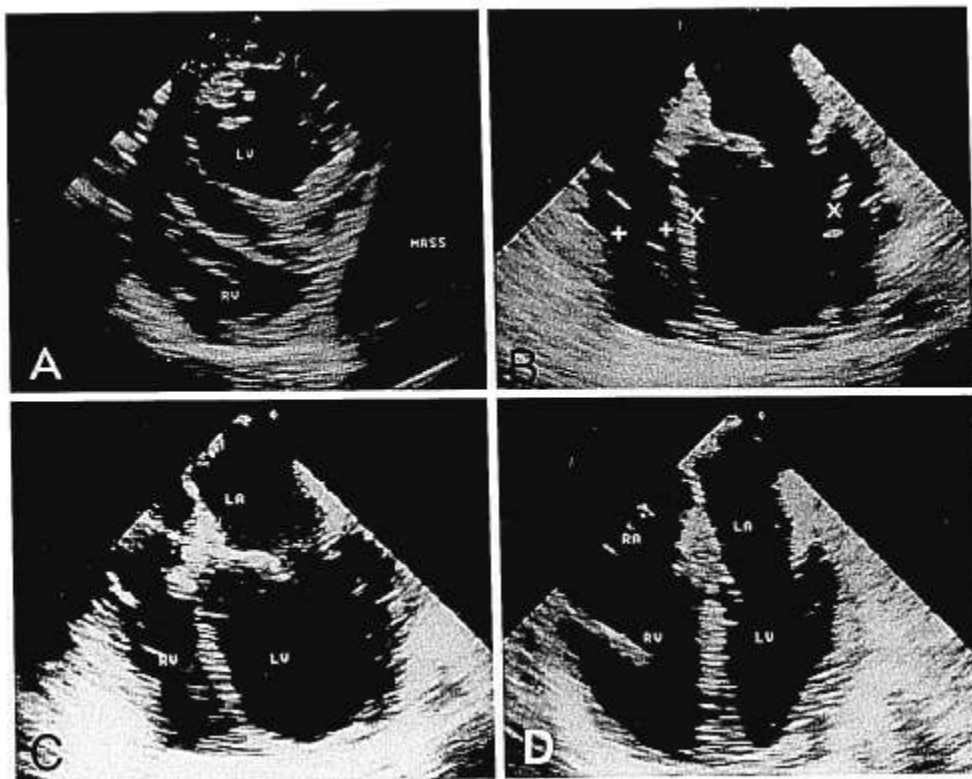
\dot{V}_E was measured with the animal breathing from the reservoir bag.

The pleural cavity was entered through the left sixth intercostal space. A catheter, introduced through the left atrial appendage, was used to measure left atrial pressure (LAP). After the catheter was securely sutured in place, the pericardium was closed. An empty intravenous bag (800 ml capacity) was carefully positioned in the anterior mediastinum, and this served as our mediastinal mass. A large-bore tube connected to the bag allowed inflation or deflation of the bag with water. Both the tube connected to the mediastinal bag and a 14-G chest tube positioned in the left pleural cavity were exteriorized through the ninth intercostal space. A mediastinal mass was simulated by addition of 50-ml aliquots of water to the mediastinal bag through the previously exteriorized tubing. The aliquots of water were infused until CO decreased by 25% below baseline values. The water could be aspirated through the tubing to empty the bag and thereby relieve the acute effects of the mass. The ribs were appositioned and the thoracotomy carefully closed in two separate layers that were air tight.

The animals were then repositioned and remained supine for the entire experiment. Pao was increased to a value of 20 cmH₂O to reinflate the lung, and the chest tube was connected to continuous suction (25 cmH₂O). A latex esophageal balloon was inserted in six dogs, with the distance from mouth to left atrium having been previously measured. Proper positioning to measure esophageal pressure (Pes) was verified by noting the presence of cardiac oscillations on Pes deflections. All airway and vascular pressure waveforms were recorded with an eight-channel oscillograph (Hewlett Packard).

In the remaining six dogs, TEE was performed with the use of a biplane 5-MHz, 32-element phase array transducer interfaced with an Aloka 870® imaging system. The probe was positioned to obtain a standard four-chamber view. All studies were recorded on a Panasonic 6300® video cassette recorder and subsequently analyzed both in slow motion and stop frame. Endocardial measurements were made of maximal diastolic right and left ventricular short axis dimensions at the level of the mitral chordae or within 1 cm of the mitral and tricuspid annulus (fig. 1B). When the position of the heart was altered by inflation of the mediastinal mass or mechanical ventilation, the position of the transesophageal transducer was adjusted so that maximal chamber dimension was still obtained. The position of the mediastinal mass was also verified during the experimental protocol by TEE. Figure 1A shows the inflated anterior mediastinal mass adjacent to the right and left ventricles. The esophageal probe is located at the apex of the triangular echocardiographic field. Chamber dimensions, at end-expiration, were mea-

FIG. 1. A composite of transesophageal recordings obtained during these experiments. A shows the relative spatial position of the heart and mediastinal mass. B shows a typical measurement of short axis diameter in the left and right ventricles. Distances between + marks (right ventricle) and X marks (left ventricle) were measured. C and D depict the changes in short axis diameters noted during mass deflation (C) and mass inflation (D).



sured in triplicate, and each measurement was calculated from the means obtained during three cardiac cycles.

CALCULATIONS

Intrapulmonary shunt (Q_s/Q_t) was calculated from measured values of arterial and mixed venous blood gases with the following equation: $Q_s/Q_t = (Cc'O_2 - CaO_2) / (Cc'O_2 - C\bar{V}O_2)$, where $Cc'O_2$ = end capillary content; $C\bar{V}O_2$ = mixed venous content; and CaO_2 = arterial content. Alveolar P_{O_2} was calculated with the use of the alveolar gas equation, whereas arterial and mixed venous values of P_{O_2} (Pa_{O_2} and $P\bar{V}O_2$, respectively) were measured directly. Blood gases were analyzed for P_{O_2} , P_{CO_2} , and pH at $37^\circ C$ (Corning 162-2) with the use of appropriately calibrated electrodes and then corrected for core body temperature after hemoglobin saturation was calculated from a standard canine nomogram.¹² Oxygen contents were calculated from derived oxyhemoglobin saturations and hemoglobin concentrations. Hemoglobin, in turn, was estimated as one third of the measured hematocrit. Although not exact, this estimate of hemoglobin result does not affect the calculation of changes in oxygen content.

EXPERIMENTAL PROTOCOL

Part 1

After surgical preparation, the animals were allowed to breathe spontaneously. Inspired halothane concentra-

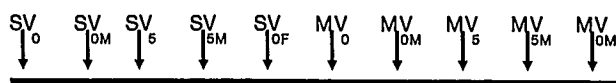
tion was adjusted to allow for the lowest concentration that ablated the blink reflex but still maintained regular spontaneous breathing. We attempted to maintain this concentration of halothane near constant for the duration of the experiment by not varying the initial halothane vaporizer setting.

In the first six dogs (group 1), the effects of the mediastinal mass on cardiac performance were assessed with an esophageal balloon in place to calculate transmural cardiac pressures. Transmural pressures were calculated by subtracting end-expiratory P_{es} from end-expiratory intracavitary pressure. Each animal received ten consecutive ventilatory periods: five periods of spontaneous ventilation and five periods of mechanical ventilation. Measurements during each period included SAP, PAP, PAOP, RAP, LAP, P_{ao} , P_{ac} , P_{es} , heart rate (HR), respiratory rate (RR), and \dot{V}_E . Blood samples were also obtained for measurement of hematocrit and arterial and mixed venous blood gases. Core body temperature and CO were measured by the thermistor tip of the pulmonary artery catheter. A sample of expired gas was obtained and analyzed for halothane concentration with the use of an SARA (Allegheny International Medical Technology, St. Louis, MO) mass spectrometer.

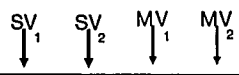
Measurements were first obtained during spontaneous ventilation (fig. 2). The mediastinal bag was empty, and the level of CPAP was set to 0 cmH₂O (period SV₀). This was then followed by a period of spontaneous ventilation

Part 1

Group 1 (n=6)



Group 2 (n=6)



Part 2 (n=12)

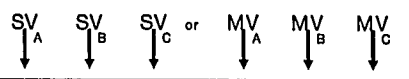


FIG. 2. The experimental protocol. Details on individual periods during spontaneous ventilation (SV) or mechanical ventilation (MV) can be found within the text.

with the bag inflated with water (period SV_{OM}); 5 cmH₂O CPAP was then added and measurements were obtained during spontaneous ventilation with the mediastinal bag empty (period SV_5) and then during spontaneous ventilation with the mediastinal bag filled (period SV_{5M}). A final set of measurements was obtained after discontinuation of CPAP with the animals spontaneously breathing (period SV_{OF}). Pancuronium (0.1 mg/kg) was then administered and mechanical ventilation instituted. V_T and RR during mechanical ventilation were adjusted to maintain Pa_{CO_2} similar to that observed during spontaneous ventilation. An identical protocol was followed for mechanical ventilation as for spontaneous ventilation, and all animals received an additional five periods of mechanical ventilation. In this manner, during mechanical ventilation the animals received a period of mechanical ventilation with the mass empty (period MV_O) and the mass filled (period MV_{OM}). PEEP (5 cmH₂O) was then added, and the animals received two additional periods with the bag empty and then filled (periods MV_5 and MV_{5M} , respectively). The animals received a final period of mechanical ventilation with the bag empty (period MV_{OF}). Between periods the animals received three large breaths to help prevent atelectasis.

In the remaining six dogs (group 2), the effects of an anterior mediastinal mass were assessed with measurements that included cardiac chamber size assessed by TEE. As in group 1 experiments, spontaneous ventilation always preceded mechanical ventilation. In group 2, however, before baseline measurements, 300 ml of blood was withdrawn and 5,000 units heparin added, and the blood was saved for subsequent reinfusion. With the animals breathing spontaneously, all measurements were obtained

as in group 1 with the exception of Pes , because the presence of TEE precluded measurement of Pes . With the use of TEE, left and right ventricular diameters were measured. All measurements were first obtained with the mediastinal bag empty (period SV_1). The mediastinal bag was then inflated to the end point of reduction of CO by 25% over baseline. The previously stored blood was transfused to restore CO to baseline values, and all measurements were repeated (period SV_2). Pancuronium (0.1 mg/kg) was then administered and mechanical ventilation instituted. Again, V_T and RR during mechanical ventilation were adjusted to maintain Pa_{CO_2} similar to that observed during spontaneous ventilation. The protocol during mechanical ventilation was identical to that during spontaneous ventilation. Measurements were obtained with the bag empty (period MV_1) and filled (period MV_2).

Part 2

At the conclusion of the mechanical ventilation periods, neostigmine (0.05 mg/kg) and glycopyrrolate (1 mg) were administered to reverse the effects of pancuronium. All animals from both groups 1 and 2 ($n = 12$) were then randomized to receive either a period of spontaneous ventilation (period SV_A) or a period of mechanical ventilation (period MV_A). Those receiving mechanical ventilation also received muscle relaxant (pancuronium 0.1 mg/kg). All measurements were obtained, and then the mediastinal bag was first inflated until CO decreased by 25% over baseline (periods SV_B or MV_B). After measurements, the bag was further inflated until CO decreased by at least 50% over baseline (periods SV_C or MV_C). After measurements, the animals were killed by injection of 20 ml supersaturated KCl solution. The total elapsed time of these experiments—including surgical preparation and parts 1 and 2—ranged between 3 and 4 h. The position of the mediastinal bag was then verified on opening of the mediastinal cavity.

STATISTICS

Values of blood gases, hemodynamics, and ventilatory parameters were compared between ventilatory periods with a one-way analysis of variance (ANOVA). Where the F statistic showed a significant difference, t tests were used to determine which periods were different. Sidak's multiplicative inequality was used to correct for the number of comparisons made between groups.¹³ As the number of comparisons increases, the requisite t value to show significance increases. Therefore, we prospectively limited our comparisons to the following: period SV_O versus SV_{OM} , period SV_5 versus SV_{5M} , period SV_O versus SV_{OF} , period SV_O versus SV_5 , period SV_{OM} versus SV_{5M} , period MV_O versus MV_{OM} , period MV_5 versus MV_{5M} , period MV_O

TABLE 1A. Hemodynamic and Ventilatory Variables during Spontaneous Ventilation

	SV _O	SV _{OM}	SV _S	SV _{SM}	SV _{OF}
PaCO ₂ (mmHg)	46 ± 4	46 ± 5	48 ± 5	44 ± 5	48 ± 5
PaO ₂ (mmHg)	247 ± 91	206 ± 81	232 ± 66	241 ± 111	230 ± 79
pH	7.2 ± 0.04	7.2 ± 0.03	7.21 ± 0.03	7.24 ± 0.05	7.19 ± 0.04
PvO ₂ * (mmHg)	64 ± 4	54 ± 7	64 ± 10	54 ± 6	61 ± 7
SAP* (cmH ₂ O)	175 ± 21	124 ± 24†	165 ± 13	123 ± 17	159 ± 27
PAP* (cmH ₂ O)	19 ± 3	23 ± 3†	26 ± 5‡	31 ± 3†‡	21 ± 4
PAOP (cmH ₂ O)	7 ± 3	8 ± 4	10 ± 5	12 ± 6	7 ± 3
PLAP* (cmH ₂ O)	5 ± 2	6 ± 4	9 ± 4‡	11 ± 5‡	6 ± 3
PRAP* (cmH ₂ O)	3 ± 2	6 ± 5	8 ± 4‡	10 ± 5‡	4 ± 3
CI*	169 ± 51	105 ± 10†	175 ± 48	122 ± 34†	164 ± 32

Data are expressed as mean ± SD.

PaCO₂ = partial pressure of arterial carbon dioxide; PaO₂ = partial pressure of arterial oxygen; pH_a = arterial pH; PvO₂ = partial pressure of venous oxygen; SAP = mean systemic arterial pressure; PAP = mean pulmonary arterial pressure; PAOP = transmural pulmonary artery occlusion pressure; PLAP = transmural left arterial pressure; PRAP = transmural right arterial pressure; CI = cardiac index (ml · kg⁻¹ · min⁻¹). Note: not all potential comparisons between groups were made.

* Significant difference by ANOVA.

† Significant difference in comparing periods of mass deflation with mass inflation: SV_O versus SV_{OM}; SV_S versus SV_{SM}; MV_O versus MV_{OM}; or MV_S versus MV_{SM} (P < 0.05).

‡ Significant difference comparing zero end-expired pressure to positive end-expired pressure ventilation, i.e., SV_O versus SV_S; SV_{OM} versus SV_{SM}; MV_O versus MV_S; or MV_{OM} versus MV_{SM} (P < 0.05).

versus MV_{OF}, period MV_O versus MV_S, and period MV_{OM} versus MV_{SM}. A P value less than 0.05 was considered to show a significant difference. Values shown are means ± standard deviations. The relationship between the volume of mediastinal mass and CO was analyzed by linear regression, and comparisons between periods were made with a restricted maximum likelihood analysis described by Feldman.¹⁴

Results

PART 1

In group 1, the volume of the mass was similar (P > 0.05) between periods SV_{OM}, SV_{SM}, MV_{OM}, and MV_{SM}

(566 ± 85 ml, 566 ± 85 ml, 500 ± 51 ml, and 500 ± 102 ml, respectively). Hematocrit (range, 38 ± 8 to 41 ± 5%); temperature (range, 36.9 ± 5 to 37.3 ± 3° C); HR (range, 136 ± 16 to 167 ± 23 beats per min); and Q_S/Q_T (range, 21 ± 4 to 30 ± 3%) were similar for spontaneous and mechanical ventilation periods. Blood gases, with the exception of PV_{O₂}, were also similar between periods (tables 1A and 1B). PV_{O₂} was significantly less during periods of mass inflation when compared with matched periods of mass deflation (P < 0.05) during all periods except on comparing periods MV_S and MV_{SM} (P > 0.05).

Tables 1A and 1B also show mean values of selected hemodynamic measurements obtained during spontaneous and mechanical ventilation periods, respectively.

TABLE 1B. Hemodynamic and Ventilatory Variables during Mechanical Ventilation

	MV _O	MV _{OM}	MV _S	MV _{SM}	MV _{OF}
PaCO ₂ (mmHg)	46 ± 7	45 ± 5	47 ± 6	42 ± 8	47 ± 7
PaO ₂ (mmHg)	304 ± 106	224 ± 116	247 ± 64	222 ± 28	248 ± 87
pH _a	7.25 ± 0.06	7.23 ± 0.07	7.23 ± 0.04	7.23 ± 0.06	7.22 ± 0.05
PvO ₂ * (mmHg)	61 ± 8	48 ± 11†	59 ± 9	52 ± 6	62 ± 7
SAP* (cmH ₂ O)	169 ± 23	116 ± 23†	167 ± 12	131 ± 22†	170 ± 27
PAP (cmH ₂ O)	20 ± 1	24 ± 4	25 ± 3	28 ± 3	21 ± 3
PAOP (cmH ₂ O)	9 ± 3	9 ± 5	9 ± 4	12 ± 6	8 ± 3
PLAP (cmH ₂ O)	5 ± 3	6 ± 5	8 ± 4	10 ± 5	5 ± 2
PRAP (cmH ₂ O)	4 ± 2	4 ± 5	6 ± 3	8 ± 5	4 ± 2
CI*	151 ± 15	93 ± 24†	183 ± 56	117 ± 46†	152 ± 18

Data are expressed as mean ± SD.

PaCO₂ = partial pressure of arterial carbon dioxide; PaO₂ = partial pressure of arterial oxygen; pH_a = arterial pH; PvO₂ = partial pressure of venous oxygen; SAP = mean systemic arterial pressure; PAP = mean pulmonary arterial pressure; PAOP = transmural pulmonary artery occlusion pressure; PLAP = transmural left arterial pressure; PRAP = transmural right arterial pressure; CI = cardiac index (ml · kg⁻¹ · min⁻¹). Note: not all potential comparisons between groups were made.

* Significant difference by ANOVA.

† Significant difference in comparing periods of mass deflation with mass inflation: SV_O versus SV_{OM}; SV_S versus SV_{SM}; MV_O versus MV_{OM}; or MV_S versus MV_{SM} (P < 0.05).

‡ Significant difference comparing zero end-expired pressure to positive end-expired pressure ventilation, i.e., SV_O versus SV_S; SV_{OM} versus SV_{SM}; MV_O versus MV_S; or MV_{OM} versus MV_{SM} (P < 0.05).

TABLE 2A. Selected Ventilatory Values for Spontaneous Ventilation

	SV _O	SV _{OM}	SV ₅	SV _{5M}	SV _{or}
P _{IN} (cmH ₂ O)	-2 ± 1	-1 ± 0.4	1 ± 2	0 ± 3	-2 ± 1
P _{EX} * (cmH ₂ O)	4 ± 2	3 ± 1	8 ± 3‡	10 ± 3‡	4 ± 1
PEEP (cmH ₂ O)	0 ± 0	0 ± 0	5.5 ± 0.2‡	5.5 ± 0.2‡	0 ± 0
P _{ES} * (cmH ₂ O)	0.3 ± 0.5	6 ± 2†	0.5 ± 1	6 ± 3†	0.5 ± 0.3
RR* (beats per min)	23 ± 8	26 ± 8	16 ± 6	25 ± 10†	28 ± 10
V _E (l/min)	2.7 ± 0.8	2.7 ± 0.9	2.8 ± 1.0	3.1 ± 0.8	2.5 ± 0.6
Hal* (%)	0.21 ± 0.08	0.23 ± 0.08	0.10 ± 0.03‡	0.10 ± 0.04‡	0.22 ± 0.10

Data are expressed as mean ± SD.

P_{IN} = peak inspired pressure at mouth; P_{EX} = peak expired pressure at mouth; PEEP = end-expired pressure at mouth; P_{ES} = end-expired esophageal pressure; RR = respiratory rate; V_E = minute ventilation; Hal = concentration of expired halothane.

* Significant difference by ANOVA.

† Significant difference comparing SV_O versus SV_{OM}; SV₅ versus SV_{5M}; MV_O versus MV_{OM}; or MV₅ versus MV_{5M} (*P* < 0.05).

‡ Significant difference comparing SV_O versus SV₅; SV_{OM} versus SV_{5M}; MV_O versus MV₅; or MV_{OM} versus MV_{5M}.

SAP was significantly less during periods of mass inflation when compared with matched periods of mass deflation during both spontaneous and mechanical ventilation. Transmural PRAP, PLAP, PAP, and PAOP were similar during all mechanical ventilation periods. During spontaneous ventilation, transmural LAP, RAP, and PAP increased during CPAP periods compared with their matched periods without CPAP. Mean pulmonary artery pressure also increased significantly during periods of mass inflation compared with matched periods of mass deflation (periods SV_{OM} vs. SV_O and periods SV_{5M} vs. SV₅). PCWP was similar during all periods of spontaneous ventilation. Cardiac index (CI) decreased significantly during periods of mass inflation compared with matched periods of deflation with both spontaneous and mechanical ventilation. The addition of 5 cmH₂O positive airway pressure (PEEP or CPAP) did not further depress CI, whether the mass was inflated or deflated.

Tables 2A and 2B show the values of airway and esophageal pressures during spontaneous and mechanical ventilation, respectively, in group 1. There were no differences in pressures measured either at the airway opening (Pao) or at the carina, and, therefore, only values for

Pao are shown. By definition, the addition of CPAP and PEEP increased end-expiratory pressure to at least 5 cmH₂O. During mechanical ventilation, the addition of PEEP also caused significant increases in peak airway pressure. Mass inflation increased P_{es} by approximately 6 cmH₂O during both mechanical ventilation or spontaneous ventilation (*P* < 0.05). V_E was similar during all periods of spontaneous or mechanical ventilation. RR during period SV₅ was significantly less than during other periods. Expired halothane concentration was similar in all periods of mechanical ventilation, but during spontaneous ventilation expired halothane was significantly decreased during periods SV₅ and SV_{5M}.

Table 3 illustrates selected ventilatory and hemodynamic parameters for spontaneous and mechanical ventilation for group 2 experiments. As well, Q_s/Q_t (range, 26 ± 4 to 32 ± 8%); temperature (37.4 ± 4° C); hematocrit (range, 27 ± 2 to 28 ± 4 %); and HR (range, 142 ± 10 to 162 ± 22 beats per min) were similar between periods of spontaneous or mechanical ventilation. During period SV_O, PaO₂ and P_VO₂ were decreased and V_E was increased compared with values for period SV_{OM} (*P* < 0.05). PAP, PRAP, PLAP, and PAOP were increased

TABLE 2B. Selected Ventilatory Values for Mechanical Ventilation

	MV _O	MV _{OM}	MV ₅	MV _{5M}	MV _{or}
P _{IN} * (cmH ₂ O)	8 ± 1	11 ± 2	13 ± 3‡	13 ± 2‡	8 ± 1
P _{EX} * (cmH ₂ O)	0 ± 0	0 ± 0	6 ± 1‡	6 ± 1‡	0 ± 0
PEEP (cmH ₂ O)	0 ± 0	0 ± 0	5.8 ± 0.2‡	6.2 ± 0.4‡	0 ± 0
P _{ES} * (cmH ₂ O)	0.2 ± 0.2	6 ± 1†	2 ± 0.2	6 ± 1†	0.2 ± 0.2
RR (beats per min)	13 ± 2	13 ± 2	14 ± 2	16 ± 3	14 ± 2
V _E (l/min)	3.0 ± 0.5	3.0 ± 0.4	2.6 ± 0.5	2.2 ± 0.4	3.2 ± 0.5
Hal (%)	0.16 ± 0.04	0.17 ± 0.06	0.13 ± 0.04	0.12 ± 0.04	0.16 ± 0.06

Data are expressed as mean ± SD.

P_{IN} = peak inspired pressure at mouth; P_{EX} = peak expired pressure at mouth; PEEP = end-expired pressure at mouth; P_{ES} = end-expired esophageal pressure; RR = respiratory rate; V_E = minute ventilation; Hal = concentration of expired halothane.

* Significant difference by ANOVA.

† Significant difference comparing SV_O versus SV_{OM}; SV₅ versus SV_{5M}; MV_O versus MV_{OM}; or MV₅ versus MV_{5M} (*P* < 0.05).

‡ Significant difference comparing SV_O versus SV₅; SV_{OM} versus SV_{5M}; MV_O versus MV₅; or MV_{OM} versus MV_{5M}.

TABLE 3. Ventilatory and Hemodynamic Values for Group 2 Studies

	SV ₁	SV ₂	MV ₁	MV ₂
PaCO ₂ (mmHg)	47 ± 4	45 ± 6	44 ± 5	48 ± 5
PaO ₂ (mmHg)	244 ± 54	148 ± 44*	212 ± 62	246 ± 92
pHa	7.22 ± 0.04	7.22 ± 0.04	7.25 ± 0.04	7.24 ± 0.04
PvO ₂ (mmHg)	53 ± 5	42 ± 7*	56 ± 10	53 ± 8
Hal (%)	0.20 ± 0.06	0.19 ± 0.08	0.13 ± 0.04	0.17 ± 0.06
SAP (cmH ₂ O)	133 ± 12	133 ± 24	139 ± 30	129 ± 35
PAP (cmH ₂ O)	20 ± 3	32 ± 5*	20 ± 3	26 ± 3*
PLAP (cmH ₂ O)	8 ± 2	19 ± 3*	8 ± 3	17 ± 4*
PAOP (cmH ₂ O)	9 ± 3	22 ± 3*	19 ± 3	18 ± 4*
PRAP (cmH ₂ O)	7 ± 3	17 ± 3*	6 ± 3	14 ± 3*
RR (beats per min)	33 ± 14	44 ± 25	14 ± 1	14 ± 1
V _E (l/min)	2.5 ± 0.7	4.0 ± 1.2*	2.8 ± 0.6	2.9 ± 0.7
CI	155 ± 34	147 ± 22	146 ± 36	130 ± 26
LV (mm)	27 ± 6	22 ± 5*	26 ± 7	21 ± 7*
ΔLV (mm)		-6 ± 2		-4 ± 1
RV (mm)	13 ± 2	15 ± 3*	11 ± 2	15 ± 4*
ΔRV (mm)		2 ± 1		3 ± 1

Data are expressed as mean ± SD.

PaCO₂ = partial pressure of arterial carbon dioxide; PaO₂ = partial pressure of arterial oxygen; pHa = arterial pH; PvO₂ = partial pressure of venous oxygen; Hal = concentration of expired halothane; SAP = mean systemic arterial pressure; PAP = mean pulmonary arterial pressure; PAOP = pulmonary artery occlusion pressure; PLAP = left

arterial pressure; PRAP = right arterial pressure; RR = respiratory rate; V_E = expired minute ventilation; CI = cardiac index (ml · kg⁻¹ · min⁻¹); LV = left ventricle diameter; RV = right ventricle diameter; ΔLV = change in left ventricle diameter; ΔRV = change in right ventricle diameter.

* P < 0.05 comparing SV₁ versus SV₂ or MV₁ versus MV₂.

during periods SV_{OM} and MV_{OM} compared with periods SV_O and MV_O, respectively (P < 0.05). Pes could not be measured because of the presence of the esophageal echocardiographic probe and, therefore, transmural pressures were not calculated. Left ventricular chamber dimensions significantly decreased with mass inflation in both spontaneous and mechanical ventilation. In contrast, right ventricular dimensions significantly increased with mass inflation during spontaneous and mechanical ventilation (fig. 1C and D). The volume of fluid added to the

mediastinal bag (366 ± 90 ml) was similar between spontaneous and mechanical ventilation periods. Before each experiment, the position of the mass was verified to lie in the anterior mediastinum by TEE (fig. 1A).

PART 2

Table 4 illustrates selected ventilatory and hemodynamic variables during all three periods of spontaneous and mechanical ventilation. As well, values for Q_sQ_t

TABLE 4. Ventilatory and Hemodynamic Values (Part 2 Studies)

	SV _A	SV _B	SV _C	MV _A	MV _B	MV _C
PaCO ₂ (mmHg)	42 ± 4	45 ± 6	41 ± 8	41 ± 7	46 ± 4	49 ± 6
PaO ₂ (mmHg)	242 ± 97	131 ± 52	112 ± 73	310 ± 68	239* ± 50	195 ± 102
pHa	7.23 ± 0.05	7.21 ± 0.04	7.23 ± 0.05	7.26 ± 0.05	7.22 ± 0.02	7.26 ± 0.05
PvO ₂ (mmHg)	61 ± 6	47 ± 7	40 ± 8	56 ± 12	49 ± 14	41 ± 13
Q _s Q _t (%)	33 ± 12	37 ± 11	36 ± 15	24 ± 10	24 ± 11	24 ± 9
Hal (%)	0.20 ± 0.09	0.20 ± 0.10	0.21 ± 0.11	0.18 ± 0.08	0.18 ± 0.06	0.18 ± 0.04
SAP (cmH ₂ O)	166 ± 27	130 ± 17	109 ± 23	165 ± 36	102 ± 38	91 ± 37
PAP (cmH ₂ O)	21 ± 3	25 ± 3	28 ± 2	21 ± 4	22 ± 4	25 ± 5
PAOP (cmH ₂ O)	11 ± 4	14 ± 7	13 ± 5	11 ± 4	10 ± 3	11 ± 5
PLAP (cmH ₂ O)	8 ± 5	12 ± 6	12 ± 5	8 ± 4	7 ± 5	9 ± 6
PRAP (cmH ₂ O)	7 ± 4	8 ± 5	9 ± 6	7 ± 5	6 ± 4	7 ± 7

Data are expressed as mean ± SD.

PaCO₂ = partial pressure of arterial carbon dioxide; PaO₂ = partial pressure of arterial oxygen; pHa = arterial pH; PvO₂ = partial pressure of venous oxygen; Q_sQ_t = shunt fraction; Hal = concentration of expired halothane; SAP = mean arterial pressure; PAP = mean pulmonary arterial pressure; PAOP = transmural pulmonary artery oc-

clusion pressure; PLAP = transmural left arterial pressure; PRAP = right arterial transmural pressure.

* Significant difference comparing mechanical ventilation versus spontaneous ventilation (i.e., SV_A vs. MV_A; SV_B vs. MV_B; and SV_C vs. MV_C).

(range, 24 ± 11 to $37 \pm 12\%$); temperature (range, 36 ± 1 to $37 \pm 0.6^\circ \text{C}$); hematocrit (range, 28 ± 8 to $38 \pm 8\%$); and HR (145 ± 15 to 165 ± 29 beats per min) were similar between spontaneous and mechanical ventilation. Values of PaO_2 were significantly greater during mechanical ventilation compared with spontaneous ventilation (SV_B vs. MV_B). All other variables were similar between spontaneous and mechanical ventilation.

The relationships between CO and mediastinal mass volume for each individual dog during spontaneous ventilation ($n = 6$) and mechanical ventilation ($n = 6$) as analyzed by linear regression¹⁴ were similar ($P > 0.05$). Figure 3 shows values of CO (normalized as a percentage value of initial CO) plotted against mediastinal mass during spontaneous and mechanical ventilation. This pooling of all spontaneous and mechanical ventilation values for CI and mediastinal mass produced two regressions that were also similar ($P > 0.05$). Finally, we combined the results of the six animals that had measurements of Pes, and we analyzed the relationship between Pes and the volume of the mediastinal mass by linear regression. We found the relationship to be described by the equation $\text{Pes} = 0.012 \times (\text{volume}) - 0.11$ ($r = 0.963$, $n = 18$).

Discussion

RATIONALE

It is recognized that an anterior mediastinal mass can cause extrinsic compression of the trachea or major bronchus, leading to airway obstruction and respiratory fail-

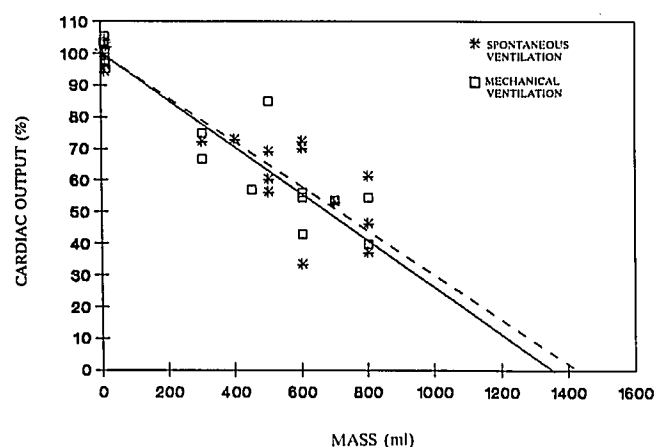


FIG. 3. The combined group data of percent cardiac output at varying volumes of mass inflation or deflation during spontaneous or mechanical ventilation. The y axis is the relative percent cardiac output calculated using initial cardiac output with mass deflation as 100% in each experiment. The x axis is the volume (milliliters) infused into the mediastinal mass. Regression lines during spontaneous ventilation (continuous line) and mechanical ventilation (dashed line) were not statistically different.

ure.^{1,2,5,9,15,16} The absence of tracheal compression, however, does not guarantee the absence of complications and a safe intraoperative course. Severe cardiorespiratory compromise, even resulting in death,⁸⁻¹⁰ has been reported despite the presence of adequate air entry by auscultation or the presence of a patent airway on direct visualization. This has led to speculation that an anterior mediastinal mass may cause compression of major thoracic structures other than the trachea, including compression of the right ventricular outflow tract.¹ Our experiments are the first of which we are aware that systematically describe the effects of an anterior mediastinal mass on ventricular function.

MODEL

This canine model of an anterior mediastinal mass allows for reversible inflation or deflation with fluid of a bag filling the entire anterior mediastinum. Proper positioning of the mass was verified at necropsy and, when possible, by TEE. In preliminary experiments, we evaluated this model using animals with differing anterior mediastinal anatomy and chest wall compliance. We found that a mediastinal mass volume of 400 ± 100 ml resulted in similar decreases in CO (near 25%) in sheep, pigs, or dogs. We speculated that, with the relatively shallow anterior mediastinum found in humans, the mediastinal mass would tend to compress the right ventricular outflow tract even more than in the animal species we studied. Thus, we were confident that we could extrapolate hemodynamic results of the canine model to species with differing thoracic compliances and configurations, including humans. This extrapolation was strengthened when we observed similar echocardiographic changes in a human clinical report,¹⁷ as was noted in our studies (see below).

We did not attempt to develop concurrent tracheal obstruction in this model. Using a fiberoptic bronchoscope, we could not detect any evidence of tracheal compression after mass inflation. We also observed that mouth pressures and distal airway pressures were almost identical after mass inflation during both spontaneous and mechanical ventilation. If tracheal compression occurred with mass inflation, we would have expected a higher flow-related pressure loss across the obstructing segment. We did not detect this, and thus we are confident that our observations are not related to a mechanical tracheal compression.

During all the mechanical ventilation periods, we administered pancuronium to the animals to produce muscle paralysis. Therefore, in part 1 experiments, group 1 studies (spontaneous ventilation) always preceded group 2 studies (mechanical ventilation). In this way we avoided any possibility that there may have been residual muscle

paralysis that was not completely reversed by neostigmine during spontaneous ventilation. In part 2 experiments, after reversal of muscle paralysis, the animals were randomized to receive either spontaneous or mechanical ventilation. We found results similar to those of the part 1 experiments (see below); therefore, we do not believe that the order of ventilatory periods influenced our results. Finally, comparison of hemodynamic and gas exchange values between the initial and final periods (periods SV_O and SV_{OF} or periods MV_O and MV_{OF}) demonstrated that the model was stable over the time of the studies.

We used a balanced anesthetic (barbiturate/halothane) during our surgical preparation in amounts common to standard veterinary practice.¹⁸ During the baseline spontaneous ventilation period, initial values of CO, arterial blood gases, central venous pressure, and pulmonary and systemic arterial pressure were within expected norms of anesthetized dogs.¹⁹ During the experimental protocol, anesthesia was maintained with halothane. Halothane has been suggested as the anesthetic of choice in patients with a mediastinal mass to maintain spontaneous ventilation and minimize tracheal obstruction.¹ Although halothane may depress cardiac function²⁰ and potentially enhance the cardiopulmonary effects of the mediastinal mass, a similar situation would also exist in standard human anesthetic practice. It is possible that alternate anesthetic agents that depress cardiac function to a lesser degree would be preferable when the effects of an anterior mediastinal mass are predominantly hemodynamic.

PART 1 STUDIES

Group 1

We evaluated the hemodynamic effects of an anterior mediastinal mass and its interactions with mode of ventilation. We evaluated spontaneous ventilation with or without 5 cmH₂O CPAP and mechanical ventilation with or without 5 cmH₂O PEEP. Although we attempted to match factors that might independently influence cardiac performance, we found that there was a significant decrease in expired halothane concentrations during CPAP ventilation periods in group 1 studies. This might result in small changes, less than 1 or 2 mmHg,²¹ in atrial pressures and cannot explain our hemodynamic results.

We found that mass inflation resulted in a reproducible increase in esophageal pressure to 6 cmH₂O during either spontaneous or mechanical ventilation. Craven and Wood have previously shown that esophageal pressure changes accurately follow epicardial pressure changes induced by PEEP.²² In their model, esophageal pressure was measured with the patient in the lateral position as opposed to our supine positioning. Even if our supine measure-

ments of esophageal pressures overestimate pericardial pressure because of gravitational effects, this would result in an underestimation of transmural pressures during mass inflation, and therefore does not explain our observed decreases in hemodynamic parameters.

Cardiac index and mean arterial pressure decreased significantly during matched periods of mass inflation compared with mass deflation. This decrease in cardiac index occurred during either spontaneous or mechanical ventilation and was similar to that observed by Craven and Wood at values of 20 cmH₂O PEEP.²²

Our group 1 studies could not specifically determine the cause of the depression in cardiac index. Cardiac index is the product of stroke index and HR, and, because HRs were similar between periods, we concluded that mass inflation significantly decreased stroke index. Stroke index, in turn, is dependent on end-diastolic ventricular volume, outflow impedance, and cardiac contractility. Because we did not measure indices of ventricular contractility, we cannot comment directly on effects (*e.g.*, reflex changes) that mass inflation might produce on cardiac contractility. However, we can be certain that changes in impedance cannot explain our findings, because, if anything, mean aortic pressure was lower during mass inflation than during mass deflation. Similarly, we observed no differences in systemic vascular resistance (range, $1,862 \pm 352$ to $2,470 \pm 498$ dyn \cdot s \cdot cm⁻⁵) in any period. Ventricular volumes are often estimated from pressures, but this assumes that there have been no changes in ventricular chamber compliance between interventions. We measured transmural pressures as the difference between esophageal and intracavitary pressure and found that, although intracavitary pressures were increased during mass inflation, transmural pressures were similar between periods of mass inflation and deflation. Therefore, from these studies we concluded that either ventricular compliance decreased or that the inflation of the mass resulted in reflex depression of cardiac contractility.

We also compared the independent effects of CPAP and PEEP in our model during spontaneous and mechanical ventilation. Application of positive end-expired pressure did not result in significant changes in values of cardiac index and mean arterial pressure. At these low levels of applied airway pressure, we did not find significant increases in esophageal pressures either with or without the mass inflated. The effects of CPAP and PEEP were demonstrated in the increased end-expiratory pressure during spontaneous ventilation and increased peak inspiratory and end-expiratory pressures during mechanical ventilation. We initially wondered whether adding 5 cmH₂O pressure might further depress cardiac index during mass inflation because of an increase in right ventricular afterload.²³⁻²⁵ Alternately, with increased lung

inflation there might be a splint-like effect, limiting additional cardiac compression by the mediastinal mass. We found, however, that the addition of positive airway pressure neither augmented nor diminished cardiac index, and this was true during either spontaneous or mechanical ventilation. Although it is possible that greater levels of CPAP or PEEP might produce different effects, we did not test for this, and these interactions remain speculative at this time.

Group 2

We performed the group 2 studies to further define the cause of the decrease in stroke index associated with mass inflation. From group 1 results, we were certain that neither reductions in intraventricular pressures nor airway collapse could explain these effects. We decided to evaluate whether changes in ventricular compliance could account for our findings, using independent measurements of ventricular volume and pressure. Caneda *et al.* previously described a patient with an anterior mediastinal mass in whom echocardiograms demonstrated an enhanced leftward displacement of the interventricular septum and a subsequent decrease of left ventricular stroke index.¹⁷ These findings were similar to the effects of PEEP on left ventricular performance. Jardin *et al.*¹¹ demonstrated the decrease in CO with PEEP was associated with a nonuniform increase in right ventricular dimensions and enhanced leftward curvature of the interventricular septum. Using TEE, we measured the effect of mass inflation on right and left ventricular dimensions. Although, ideally, TEE should minimize the requirement for probe repositioning, we found that mass inflation resulted in a significant shift of the mediastinum with a resultant requirement to change probe positioning. Figure 1A demonstrates the position of the mediastinal mass in relation to the heart. To minimize repositioning of the probe adjustment, we limited mass inflation to volumes causing a 25% decrease of CO. With mediastinal shift there was foreshortening of the ventricular longitudinal axis, but ventricular short axis measurements were reproducible (fig. 1B). Although we could not accurately measure ventricular volumes, we have used the short axis changes as an index of volume changes.

We measured ventricular volumes at the same cardiac index before and after mass inflation. We infused autologous blood after mass inflation until CO returned to baseline values. In this manner, we compared ventricular dimensions required to produce a similar CO during periods of mass inflation and deflation. SAP, PAOP, PLAP, and PRAP were all increased with mass inflation during spontaneous or mechanical ventilation. The presence of the esophageal probe precluded measurement of esoph-

ageal pressure and calculation of transmural filling pressures, but, using the previously derived relationship between mediastinal mass and Pes, we were able to calculate estimates of transmural pressures. An estimation of transmural filling pressures was obtained by calculating Pes from the regression equation of $Pes = 0.012 \times \text{mass volume} - 0.11$ noted for group 1 experiments. Estimated transmural filling pressures were also greater with mass inflation compared with mass deflation during spontaneous or mechanical ventilation. This implies that a greater filling pressure was required to achieve similar COs with mass inflation because of decreased cardiac compliance. We found that, on inflating the mass, left ventricular diameter significantly decreased by 6 ± 2 mm during spontaneous ventilation and by 4 ± 1 mm during mechanical ventilation. Right ventricular diameter significantly increased during mass inflation by 2 ± 1 mm during spontaneous ventilation and by 3 ± 1 mm during mechanical ventilation. The changes in left and right ventricular dimensions were similar in comparison of mechanical ventilation and spontaneous ventilation. Figures 1C and D illustrate these differing changes in ventricular diameter with mass inflation. From these studies, we can conclude that inflation of the mediastinal mass caused decreased left ventricular compliance with a resultant decrease in stroke volume for a similar transmural pressure. The decrease in left ventricular compliance likely reflects the increased right ventricular volume resulting from right ventricular outflow obstruction by the mass. By interventricular interdependence, right ventricular enlargement could then result in decreased left ventricular compliance and decreased CO.²⁶

These results can be contrasted to the more global ventricular compromise seen with pericardial tamponade. In an acute canine tamponade model,²⁷ right ventricular collapse was seen when CO decreased by 21% and interpericardial pressure exceeded right ventricular diastolic pressure. The addition of an outflow tract obstruction (pulmonary artery band) impeded the collapse of the right ventricle by increasing right ventricular diastolic pressure. In our model of an anterior mediastinal mass, the right ventricle also is primarily affected, but it increases in volume rather than collapses.

PART 2 STUDIES

We wished to extend our initial findings and directly compare the differing effects of spontaneous and mechanical ventilation with muscle paralysis during mass inflation. All data points with mass inflation or deflation were grouped in either spontaneous or mechanical ventilation periods. The only significant differences that we found were in values of arterial oxygenation; we found

no difference in the independent variables of temperature, with values of PaO_2 greater during mechanical ventilation than during spontaneous ventilation. It is possible that this resulted from alveolar collapse caused by compression by the mediastinal mass. Similarly, there was a trend for shunt to be higher during spontaneous ventilation than during mechanical ventilation.

We analyzed the relationship between CO and volume of mass inflation for each dog during spontaneous or mechanical ventilation. The regression lines for spontaneous and mechanical ventilation are similar, and we can therefore conclude that the decrease in CO associated with various levels of mass inflation was similar during spontaneous or mechanical ventilation. This implies that the mode of ventilation did not independently affect the depression in CO associated with mass inflation. We therefore believe that the physiologic effects of the mediastinal mass on cardiac performance far outweigh the effects of spontaneous or mechanical ventilation.

We did not study therapeutic interventions that might improve the hemodynamic compromise observed with an anterior mediastinal mass. In another model of outflow obstruction (pulmonary artery band), right ventricular failure is improved by infusion of phenylephrine.²⁸ This increases aortic pressure and improves right ventricular coronary blood flow. As well, left ventricular diastolic pressure is increased, minimizing the septal shift secondary to a distended right ventricle. In contrast, continued infusion of volume expanders might potentially augment the distension of the right ventricle, aggravate the septal shift, and lead to additional reduction in left ventricular stroke volume.²⁹ Measurement of right ventricular end-diastolic volume with the use of a modified pulmonary artery catheter might allow for better detection and treatment of right ventricular distension.²⁵

In summary, we have studied the effects of an anterior mediastinal mass on the cardiac system in the absence of tracheal obstruction in dogs anesthetized with pentobarbital and halothane. We have shown that cardiac index decreased to a similar extent during both spontaneous and mechanical ventilation and that the addition of low levels of positive airway pressure significantly influenced the degree of cardiac impairment. We did find that positive-pressure ventilation was associated with improved oxygenation, likely on the basis of a reduction in intrapulmonary shunt. This implies that ventilatory interventions do not play a major role in management of hemodynamic compromise associated with an anterior mediastinal mass. Therefore, in the absence of tracheal obstruction, we cannot recommend one specific mode of ventilation as having superior characteristics in the treatment of patients with an anterior mediastinal mass. We did find that a large mediastinal mass causes a reduction

in cardiac index on the basis of a decrease in stroke index. The resultant reduction in stroke index is best explained by an increased right ventricular afterload causing right ventricular dilatation and secondarily increasing left ventricular stiffness. Based on these findings, therapy aimed at augmenting right ventricular performance with a resultant decrease in right ventricular chamber size might be clinically useful.

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