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Effects of Ventilatory Techniques during Cardiopulmonary Bypass on Post-bypass and Postoperative Pulmonary Compliance and Shunt

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Pulmonary compliance and shunt were evaluated preoperatively, 30 minutes after cardiopulmonary bypass, and two hours postoperatively in 132 calves undergoing open-heart surgery with halothane and oxygen anesthesia. The calves were divided into 11 groups with respect to maintenance of the lungs during bypass. In Group 1 the lungs were collapsed during bypass. In all other groups the lungs were mechanically ventilated, statically inflated, or both, with either pure oxygen or nitrous oxide, 50 per cent, in oxygen. All groups had similar compliance and shunt values before operation and sustained significant decreases in compliance and increases in shunt 30 minutes after bypass. Calves exposed to positive-pressure breathing during bypass had higher shunt and lower compliance values after bypass and postoperatively than those not exposed to mechanical ventilation, irrespective of the inflating gas or presence or absence of any amount of static airway pressure. Animals not ventilated during bypass had compliance and shunt values that were not significantly different from preoperative values, while calves that were ventilated had compliance and shunt values that were still significantly altered two hours postoperatively. These data demonstrate that positive-pressure breathing during bypass decreases pulmonary compliance after bypass and postoperatively and increases intrapulmonary shunt, but that the gas inflating the lungs during bypass does not influence either of these variables. The findings also suggest that static pulmonary inflation during bypass offers no advantage over allowing the lungs to remain collapsed. (Key words: Surgery, cardiovascular; Lung, compliance; Lung, function: Lung, shunting; Ventilation, positive-pressure breathing.)

RESPIRATORY DISTRESS after open-heart surgery remains a problem in spite of adequate venting of the left side of the heart during cardiopulmonary bypass, improved pump oxygenators, and better preoperative preparation and intraoperative management.[1–3] Some of the postoperative respiratory distress is related to the primary pathologic process[4,5] and some to invasion of the pleural cavities.[1] However, another source of postoperative respiratory dysfunction may be improper management of the lungs during bypass.

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The ideal method of maintaining the lungs during cardiopulmonary bypass to optimize post-bypass and postoperative pulmonary function is controversial. The reasons for this are that carefully controlled studies are difficult to perform in patients undergoing open-heart surgery, and the unavailability of animal data. In this study we evaluated the effects of a number of methods of maintaining the lungs of calves during cardiopulmonary bypass on pulmonary compliance (C) and pulmonary shunting ($\dot{Q}_S/\dot{Q}_T$) immediately following discontinuation of bypass and postoperatively.

Methods

The experimental subjects were 132 healthy, 90–98 kg Holstein bull calves undergoing implantation of pneumatically driven artificial hearts. Each animal was anesthetized with sodium methohexital, 3 to 5 mg/kg, intravenously, the trachea was intubated, and anesthesia was maintained with halothane, 1 to 2 per cent, in oxygen. Ventilation was maintained with a volume-limited respirator through a humidified nonrebreathing circuit. The respirator was adjusted to produce a tidal volume of 15 ml/kg at a rate of 10–15 breaths/min to keep arterial carbon dioxide tension between 30 and 35 torr. Following this, a 10-gauge needle was placed percutaneously into the right external jugular vein. With continuous pressure recording, a 7-Fr Swan-Ganz triple-lumen catheter was threaded through the needle, through the right ventricle, and into the proximal pulmonary artery. A second catheter (17 Fr) was threaded into the central aorta from the left femoral artery.

Following a 30-minute period of equilibration with the calf in the supine position, a sternal splitting thoracotomy was performed. Catheters were placed in the right carotid artery and inferior and superior vena cava, and cardiopulmonary bypass was initiated. The extracorporeal system (Bently oxygenator; Sarns roller pump) was primed with lactated Ringer's solution, 2.5 liters, and low-molecular-weight dextran, 0.5 liters. Anticoagulation was maintained with heparin, 5 mg/kg, and anesthesia with halothane, 0.5–1.0 per cent, delivered into the oxygenator. Mixed venous blood samples were obtained at the venous inflow to the oxygenator every 15 minutes and were analyzed on a Radiometer acid–base analyzer for oxygen

TABLE 1. Properative, Post-bypass, and Postoperative Compliance (Mean ± SD)

	Ventilatory Status	Ventilatory or CPAP Gas During Bypass	Compliance (ml/cm H_2O)		
			Preoperative	Post-bypass	Postoperative
Group 1	No ventilation No CPAP	None	94 ± 8	75† ± 10	90 ± 9
Group 2	No ventilation 4 cm H_2O CPAP	Oxygen	93 ± 7	72† ± 11	89 ± 11
Group 3	No ventilation 8 cm H_2O CPAP	Oxygen	95 8	70† ± 12	88 ± 10
Group 4	PPB No CPAP	Oxygen	94 ± 8	58‡§ ± 13	78*§ ± 12
Group 5	PPB 4 cm H_2O CPAP	Oxygen	94 ± 8	59‡§ ± 12	79*§ ± 11
Group 6	PPB 8 cm H_2O CPAP	Oxygen	94 ± 9	56‡§ ± 14	77*§ ± 11

* $P < .05$, † $P < .025$, ‡ $P < .01$, Student's t test for paired data, compared with preoperative data.
§ $P < .05$, Student's t test for unpaired data, compared with values of Groups 1, 2 and 3 during this period.

TABLE 2. Preoperative, Post-bypass, and Postoperative Compliance (Mean ± SD)

	Ventilatory Status	Ventilatory or CPAP Gas during Bypass	Compliance (ml/cm H_2O)		
			Preoperative	Post-bypass	Postoperative
Group 1	No ventilation No CPAP	None	94 ± 8	75† ± 10	90 ± 9
Group 2N	No ventilation 4 cm H_2O CPAP	50 per cent N_2O in oxygen	94 ± 8	71† ± 12	87 ± 12
Group 3N	No ventilation 8 cm H_2O CPAP	50 per cent N_2O in oxygen	95 ± 8	70† ± 13	89 ± 11
Group 4N	PPB No CPAP	50 per cent N_2O in oxygen	94 ± 7	56‡§ ± 11	76*§ ± 10
Group 5N	PPB 4 cm H_2O CPAP	50 per cent N_2O in oxygen	93 ± 7	58‡§ ± 11	77*§ ± 12
Group 6N	PPB 8 cm H_2O CPAP	50 per cent N_2O in oxygen	95 ± 9	58‡§ ± 13	77*§ ± 10

* $P < .05$, † $P < .025$, ‡ $P < .01$, Student's t test for paired data, compared with preoperative data.
§ $P < .05$, Student's t test for unpaired data, compared with values of groups 1, 2N and 3N during this period.

and carbon dioxide tensions and pH. Bypass flows were maintained between 50 and 70 ml/kg/min to keep mixed venous oxygen tension between 38 and 42 torr. Cow blood was administered as needed to maintain bypass flows greater than 50 ml/kg/min. Esophageal temperature was monitored with a Yellow Springs temperature probe. All animals were cooled to 28 C during bypass and rewarmed to 39 C (normal for a calf) at its conclusion. The calves were randomly placed into one of 11 study groups of 12 animals each prior to induction of anesthesia according to the method of maintaining the lungs during bypass. These included: nonventilation with the lungs collapsed (Group 1); nonventilation with the lungs exposed to a continuous positive airway pressure (CPAP) of 4 (Group 2) or 8 cm H_2O (Group 3) using pure oxygen; nonventilation with the lungs exposed to CPAP of 4 (Group 2N) or 8 cm H_2O (Group 3N) using nitrous oxide, 50 per cent, in oxygen; ventilation or positive-pressure breathing (PPB) at a rate of 6 breaths/min and tidal volume of 10 ml/kg without CPAP, using pure oxygen (Group 4) and nitrous oxide, 50 per cent, in oxygen (Group 4N); PPB with 4 (Group 5) or 8 cm H_2O (Group 6), using pure oxygen; PPB with 4 (Group 5N) or 8 cm H_2O (Group 6 N) CPAP using nitrous oxide, 50 per cent, in oxygen.

A silicone rubber or polyurethane elliptical type of artificial heart was used in these investigations. This type of artificial heart, and its driving and control systems, have been described.[6-8] The arti-

ficial heart is attached to the natural pulmonary artery and aorta via standard Dacron arterial grafts. These grafts contain taps to which pieces of high-pressure intravenous tubing are connected and brought outside of the chest for blood sampling and pressure measurement.

Following implantation of the artificial heart, bypass was terminated and artificial heart output adjusted via heart rate and air driving pressure manipulations to maintain pulmonary arterial oxygen tension within normal limits (38–42 torr).[6–8] Following this, tubes for drainage of both pleural cavities were implanted, the thoracotomy incision was closed, and the animal was placed in the prone position. Ventilation after bypass was accomplished with humidified halothane, 0.5 per cent, in oxygen and postoperatively with humidified pure oxygen.

C and $\dot{Q}_S/\dot{Q}_T$ were measured 60–75 minutes after anesthetic induction and intubation but before thoracotomy, 30 minutes following bypass but before chest closure, and two hours after anesthesia and operation. Volume data were obtained with a giant syringe by static inflation of the lungs from functional residual capacity with 15 ml/kg of the inspired mixture of gases. The animals then exhaled passively into a waterless spirometer. Exhaled volumes measured by the spirometer were corrected to BTPS. Airway pressure was measured at the endotracheal tube utilizing a specially designed adapter. $\dot{Q}_S/\dot{Q}_T$ was calculated using methods previously reported.[9]

TABLE 3. Properative, Post-bypass, and Postoperative Pulmonary Shunt (Mean ± SD)

	Ventilatory Status	Ventilatory or CPAP Gas during Bypass	Pulmonary Shunt (Per Cent)		
			Preoperative	Post-bypass	Postoperative
Group 1	No ventilation No CPAP	None	14 ± 3	25† ± 7	17 ± 4
Group 2	No ventilation 4 cm H_2O CPAP	Oxygen	13 ± 4	28† ± 5	16 ± 4
Group 3	No ventilation 8 cm H_2O CPAP	Oxygen	13 ± 4	27† ± 5	16 ± 5
Group 4	PPB No CPAP	Oxygen	13 ± 3	41‡§ ± 10	24†§ ± 3
Group 5	PPB 4 cm H_2O CPAP	Oxygen	12 ± 3	45‡§ ± 10	27†§ ± 4
Group 6	PPB 8 cm H_2O CPAP	Oxygen	12 ± 4	44‡§ ± 9	26†§ ± 4

† $P < .025$, ‡ $P < .01$, Student's t test for paired data, compared with preoperative data.
§ $P < .05$, Student's t test for unpaired data, compared with values of Groups 1, 2 and 3 during this period.

TABLE 4. Preoperative, Post-bypass, and Postoperative Pulmonary Shunt (Mean ± SD)

	Ventilatory Status	Ventilatory or CPAP Gas during Bypass	Pulmonary Shunt (Per Cent)		
			Preoperative	Post-bypass	Postoperative
Group 1	No ventilation No CPAP	None	14 ± 3	25† ± 7	17 ± 4
Group 2N	No ventilation 4 cm H_2O CPAP	50 per cent N_2O in oxygen	12 ± 3	29† ± 6	17 ± 7
Group 3N	No ventilation 8 cm H_2O CPAP	50 per cent N_2O in oxygen	14 ± 4	28† ± 7	18 ± 6
Group 4N	PPB No CPAP	50 per cent N_2O in oxygen	13 ± 3	43‡§ ± 11	27†§ ± 7
Group 5N	PPB 4 cm H_2O CPAP	50 per cent N_2O in oxygen	12 ± 3	45‡§ ± 12	24†§ ± 6
Group 6N	PPB 8 cm H_2O CPAP	50 per cent N_2O in oxygen	14 ± 4	43‡§ ± 12	26†§ ± 5

† $P < .025$, ‡ $P < .01$, Student's t test for paired data, compared with preoperative data.
§ $P < .05$, Student's t test for unpaired data compared with values of Groups 1, 2N and 3N during this period.

Mean weight, time of bypass and blood loss and replacement during and after bypass and for two hours postoperatively were similar in all groups. Both pleurae were entered prior to bypass in every animal.

Results

C and $\dot{Q}_S/\dot{Q}_T$ of calves in Groups 1–6 are given in tables 1 and 3, respectively. C and $\dot{Q}_S/\dot{Q}_T$ of calves in Groups 2N–6N, as well as Group 1, are given in tables 2 and 4, respectively. All groups of calves had similar C and $\dot{Q}_S/\dot{Q}_T$ values prior to operation and sustained significant decreases in C and increases in $\dot{Q}_S/\dot{Q}_T$ 30 minutes after bypass. Calves whose lungs were ventilated during bypass (Groups 4–6 and 4N–6N) had higher $\dot{Q}_S/\dot{Q}_T$ and lower C values 30 minutes after bypass and two hours postoperatively than those whose lungs were not ventilated (Groups 1–3, 2N, and 3N), irrespective of the bypass inflating gas or presence or absence of any amount of CPAP. Animals whose lungs were not ventilated during bypass had C and $\dot{Q}_S/\dot{Q}_T$ values that were not significantly different from preoperative values two hours postoperatively, while calves whose lungs were ventilated had C and $\dot{Q}_S/\dot{Q}_T$ values that were still significantly altered at this time.

Discussion

In a recent report,[10] we demonstrated that 5 cm H_2O CPAP during bypass had little influence but PPB during bypass decreased post-bypass and postoperative C and increased $\dot{Q}_S/\dot{Q}_T$ in calves undergoing lateral thoracotomy with ketamine and succinylcholine anesthesia. We were unable to evaluate different concentrations of oxygen and different levels of CPAP during bypass in that study. We also could not be sure that continuous muscle relaxation with succinylcholine and ketamine anesthesia had not influenced our results. The findings of this investigation confirm that ventilation during bypass is detrimental to post-bypass and postoperative C and $\dot{Q}_S/\dot{Q}_T$, and that CPAP, irrespective of the level employed, offers no advantage over allowing the lungs to remain collapsed. In addition, our data suggest that the operative position and incision, anesthetic agent employed, inspired concentration of oxygen during bypass, and presence or absence of a muscle relaxant probably do not play major roles in altering post-bypass and postoperative C or $\dot{Q}_S/\dot{Q}_T$, at least in healthy calves.

That ventilation during cardiopulmonary bypass may be detrimental to pulmonary function after bypass has been suggested by a number of reports.[11–14] Cartwright and associates[12] showed that canine static pulmonary compliance remained unchanged during bypass when the lungs were passively deflated or statically inflated, but was decreased after PPB with room air. Neville *et al.*[13] demonstrated that ventilation with nitrous oxide and oxygen during bypass increased pulmonary resistance and decreased dynamic compliance in patients during open-heart surgery. Mandelbaum and Giammona[14] found that ventilating the lungs of dogs with oxygen during extracorporeal circulation decreased dynamic pulmonary compliance 15 per cent after two hours of bypass and 45 per cent after four hours.

The mechanism(s) responsible for the above mentioned changes, as well as those found in our study, is unclear. Neville and co-workers[13] suggested that ventilation during bypass results in airway hypocapnia and bronchoconstriction, and the latter is the cause of impaired pulmonary mechanics after bypass and postoperatively. They found that adding carbon dioxide to the inspired mixture of gases during bypass so that end-expiratory carbon dioxide was restored to pre-bypass levels prevented all changes in pulmonary compliance and resistance during extracorporeal circulation. Unfortunately, Neville did not measure pulmonary mechanics after bypass or postoperatively. Recent work from our laboratory (Stanley TH, Lunn JK, Liu WS, unpublished data) suggests that airway normocarbia does not influence alterations produced by ventilation during cardiopulmonary bypass on post-bypass and postoperative $\dot{Q}_S/\dot{Q}_T$ and C in healthy calves.

The report of Mandelbaum and Giammona[14] indicated that ventilation during bypass depletes pulmonary surfactant and promotes aletectasis. Additional support for surfactant depletion as the mechanism by which ventilation during bypass leads to increased pulmonary dysfunction after bypass comes from the work of Faraday and colleagues.[15] These investigators found that ventilation of excised unperfused dog lungs increased the surface tension of extracts of these lungs in direct proportion to tidal volume and duration of ventilation. They also demonstrated that the detrimental effects of PPB could be completely reversed by terminating ventilation, filling the lungs with air and maintaining them at a constant volume for several hours. Unfortunately, it was not possible to measure pulmonary surface tension in this study and, therefore, it is difficult to say what part pulmonary surfactant played in the generation of our results. However, preliminary data from another group of calves having their lower tracheobronchial tree aspirated after bypass and the secretions analyzed for surface activity, using a method previously reported,[16] suggests that PPB during bypass does indeed increase pulmonary surface tension after bypass (Stanley, unpublished data).

CPAP is an effective means of increasing functional residual capacity, preventing alveolar closure, and reducing $\dot{Q}_S/\dot{Q}_T$ in patients who have atelectasis

secondary to a variety of causes.[17–19] Use of CPAP without PPB is a common method of maintaining the lungs during bypass,[20] yet there is no available evidence that its employment prevents alveolar closure and atelectasis either during or after bypass compared with allowing the lungs to remain deflated and unventilated. Our data in this study demonstrate that post-bypass and postoperative $\dot{Q}_S/\dot{Q}_T$ and C are uninfluenced by CPAP during bypass. While $\dot{Q}_S/\dot{Q}_T$ and C may not reveal subtle forms of change in the lungs, *i.e.*, alterations in closing volume and $\dot{V}/\dot{Q}$ distribution, they are sensitive indices of alveolar collapse and atelectasis.[21] Our findings suggest, therefore, that nonventilation with either deflated or statically inflated lungs appears at this time to be the optimal method of maintaining the lungs during cardiopulmonary bypass, at least in terms of avoiding post-bypass and postoperative alveolar collapse and atelectasis.

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