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Echocardiographic analysis of cardiac function during high PEEP ventilation

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Abstract. *Objective:* Does positive end-expiratory pressure ventilation (PEEP) deteriorate cardiac contractility?
Design: By means of echocardiography nine piglets were studies during ventilation with 0, 15 and 25 cmH_2O (PEEP). Recordings were made before and after 500 ml of 6% dextran 70.
Measurement and results: Right and left ventricular end-diastolic diameters were plotted against the stroke volume determined by the thermodilution technique. By combining observations made before and after volume expansion during the different ventilation modes, a ventricular function curve was obtained. The slopes of the curves were similar during all three ventilation modes, both on the left and on the right side.
Conclusion: This study indicates undisturbed myocardial contractility during PEEP ventilation. We infer that the cardiac output deterioration in the intact animal is caused entirely by impairment of venous return.

Key words: Cardiac output – Ventricular function – Positive end-expiratory pressure – Echocardiography

Part of the decrease in CO seen during positive end-expiratory pressure (PEEP) ventilation is said to be caused by a decrease in myocardial contractility [1–6]. In two of our previous studies, one with closed chest [7] and the other with open chest design [8], 15 cmH_2O PEEP did not reveal any sign of cardiac depression. It has, however, been argued by Henning [9] and Biondi et al. [10] that the depression becomes evident at levels above 15 cmH_2O PEEP. We therefore applied 25 cmH_2O PEEP in another open chest study [11]. This time signs of a decline in right ventricular ejection fraction were observed, an event generally considered to reflect myocardial depression. But rather than a primary myocardial dysfunction induced by the PEEP itself, the decline was thought to be seen as secondary to the strain imposed by the marked elevation in right ventricular outflow impedance with a mean pulmonary arterial pressure exceeding 50 mmHg. The right ventricular end-diastolic volume as well as end-systolic volume seemed to have increased.

These observations were made in an open chest preparation with a widely open pericardium. Whether these findings apply to the intact animal remains unknown. We therefore decided to study cardiac dimensions in the intact pig during 15 as well as 25 cmH_2O PEEP.

Cardiac dimensions during PEEP ventilation have earlier been studied by Jardin et al. [12], Terai et al. [13] and Mitaka et al. [14]. The two latter investigators used PEEP of 15 cmH_2O while Jardin et al. [12] applied up to 30 cmH_2O PEEP. This study was done in patients with stiffened lungs, evidenced by the moderate drop in CO (35%) which suggests that the extreme PEEP level was only partially transmitted to the circulatory system.

Cardiac dimensions are conveniently analyzed by means of echocardiography. The technique was used in the three studies mentioned above as well as in the present one.

Material and methods

Animals and anesthesia

This study was approved by the Animal Ethics Committee of the Uppsala University. Nine pigs of Swedish native breed, 24–30 kg, 10–12 weeks old and of both sexes were used. Anaesthesia was induced with pentobarbital (Mebumal® Vet, ACO), 25 mg/kg i.v. Atropine, 0.5 mg i.v., was given to avoid salivation. Anaesthesia was maintained by a continuous infusion of methomidate (Hypnodil®, Jansen) 7.5 mg/kg/h. Pancuronium bromide (Pavulon®, Organon), 0.18 mg/kg/h i.v., was used as the muscle relaxant. Glucose in saline (Rehydrex®, Pharmacia) 15 ml/kg/h was given for hydration.

The pigs were placed on their back, tracheotomized and connected to a volume controlled ventilator (Servo Ventilator 900C, Siemens) set to 30 breaths/minute. Ventilation was given with oxygen in air ($FIO_2 > 0.5$) except during the surgical preparation when nitrous oxide in oxygen (0.7/0.3) was used. Minute volumes were adjusted to maintain

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a stable $PaCO_2$ throughout the experimental period. To maintain normal acid-base balance sodium bicarbonate was given as a continuous infusion.

Surgical procedures

A polyethylene catheter was inserted in the right carotid artery and the tip positioned in the aortic arch. Two polyethylene catheters were inserted in the left external jugular vein with the tips located in the superior vena cava, and a balloon-tipped, thermistor-probed, 7Fr, Swan-Ganz catheter was positioned in the pulmonary artery guided by pressure wave recordings.

Echocardiograhic procedure

The echocardiographic probe (5.0 MHz phased array transducer) was mounted on the tip of 120 cm long oesophagoscope (Hewlett-Packard Inc.) and connected to a Hewlett-Packard phased array ultrasound system 77020 AC, version K.

A skin incision was made to the left of the sterno-xiphoid joint and a pocket was established for the probe. The pocket extended 5 cm under the left lateral side of the sternum and was made wide enough to ensure a fair amount of motion with the echocardiographic probe.

An optimum short axis view of the heart was obtained, usually at the level of the papillary muscle (or slightly above or below), sometimes slightly oblique. This view gave a good representation of the left ventricular short axis view with the right heart cresent to the left in the picture as in a normal transthoracic short axis scan. At each experimental step (see below) the best obtainable short axis scans were sought, using the manipulative controls of the oesophagoscope. This was necessary since the various ventilation modes tended to move the heart relative to the probe by hyperinflating the lungs. All scans were videotaped, using a Panasonic AG 6200, VHS video recorder, with the PAL/Secam video system.

Measurements

Mean arterial pressure (MAP), mean right (MRAP) and mean pulmonary arterial pressure (MPAP) were measured by connecting the catheters to appropriate pressure transducers (EM 751 A, Elcomatic, Glasgow, UK). The zero reference level was 8 cm below the sternum.

The signals were amplified using a pressure similar amplifier (BAP 001, S&W, Albertslund, Denmark), read on a digital display unit (DDP 602, S&W, Albertslund, Denmark) and recorded by means of a 4-channel rectilinear heatpen recorder (MX 412, Devices, Wellwyn Garden City, UK). Mean pressures were obtained by electronic dampening of the signals. Heart rate (HR) was recorded from the ECG monitor.

Cardiac output was determined by the thermodilution technique, using a computer device (9520 A, Edwards laboratories, Santa Ana, CA). A bolus of 5 ml saline at room temperature was injected by means of an automatic syringe (ATI 1 Ulab, Sweden), starting at end-expiration. The mean value of five determinations was used.

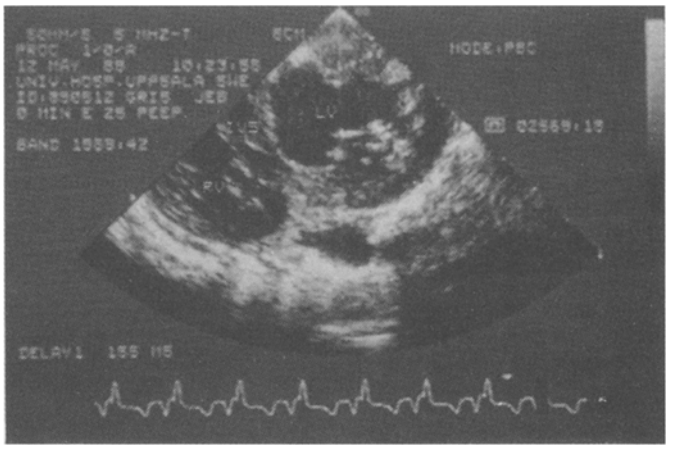
All echocardiographic measurements were done with a tracer on manually frozen frames of end-diastole (beginning of the Q-wave of the ECG) and end-systole (smallest systolic left ventricular cavity area). The following measurements were made and given as the mean of three individual measures. Left and right ventricular end-diastolic and end-systolic diameters and left ventricular end-diastolic and end-systolic areas as shown in Fig. 1. The end-diastolic interventricular septal and left ventricular free wall thicknesses were measured as well.

From the ventricular diameter, fractional shortening (FS) was calculated for the left ventricle as LV end-diastolic diameter (LVedd)-LV end-systolic diameter (LVesd)/LV end-diastolic diameter.

Experimental protocol

After preparation, the animals were allowed to stabilize for 30 min at zero end-expiratory pressure (ZEEP) ventilation.

After the stabilization a set of baseline measurements was recorded during ZEEP (denoted 0I). Recordings were then made after 10 min at each of the following ventilator settings: 15 cmH_2O of PEEP, ZEEP (0II), 25 cmH_2O PEEP and again ZEEP (0III).



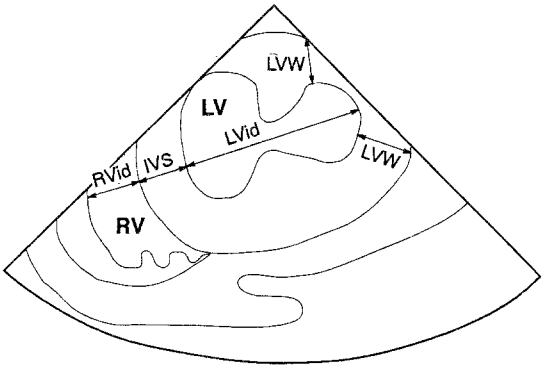


Fig. 1. Typical short axis view of the pig's heart obtained through the substernal pouch. Above: Real-time, 2-dimensional, still frame. Below: Drawn picture of the same still frame as above. *RV* = right ventricle; *LV* = left ventricle; *RVid* = right ventricular internal dimension; *IVS* = interventricular septum; *LVid* = left ventricular internal dimension; *LVW* = left ventricular wall

Thereafter the animals were primed with twenty milliliters of haptendextran (Promitén®, Pharmacia AB) and 500 ml of 6% dextran 70 (Macrodex®, Pharmacia AB) was then infused in 5 min. After another 5 min the previous protocol was repeated, resulting in a second series of recordings.

Abbreviations, calculations and statistical arrangements

The abbreviations and the formulae used for derived variables are presented in Table 1.

ANOVA for repeated measurements and the Bonferroni-Dunn test was used to evaluate differences over the observation time. Statistical significance was considered as $p<0.05$.

Results

Hemodynamic measures

The results of the hemodynamic variables are presented in Tables 2 and 3. Before volume expansion (Table 2) cardiac output (CO) was higher during 0III compared 0I (14%). The application of 15 cmH_2O PEEP resulted in a higher HR (34±25 beats/min) which was even more pronounced (89±29 beats/min) at 25 cmH_2O PEEP. Mean arterial pressure (MAP) decreased during both PEEP levels by 31±18 mmHg (4.12±2.39 kPa) and 36±18 mmHg (4.79±2.39 kPa), respectively. Decreases were also observed in CO and stroke volume (SV) during 15 cmH_2O

Table 1. Abbreviations and formulae

Hemodynamic measures	
HR	Heart rate, beats/minute
MAP	Mean arterial pressure, mmHg
CO	Cardiac output measured by thermodilution, litres/minute
SV	Stroke volume, (CO·1000/HR), ml/beat
End-diastolic measures	
RVedd	Right ventricular end-diastolic diameter, mm
LVedd	Left ventricular end-diastolic diameter, mm
LVaed	Left ventricular end-diastolic area, sq.mm
End-systolic measures	
LVesd	Left ventricular end-systolic diameter, mm
LVAes	Left ventricular end-systolic area, sq.mm
Fractional changes	
LVFS	(LVedd-LVesd)/LVedd

PEEP (51% and 59% resp.), as well as during 25 cmH_2O PEEP (62% and 78% resp.).

Before volume expansion HR was higher (55±41 beats/min) and SV was lower (43%) during 25 cmH_2O PEEP compared to 15 cmH_2O PEEP.

After volume expansion (Table 3) no differences in either HR or MAP were observed between the ZEEP measures (0I, 0II, 0III). CO was lower during 0II (10%) and 0III (18%) compared to 0I. SV was also lower during 0III compared to 0I (15%). When 15 cmH_2O PEEP was applied, no difference was noticed in the HR, but during 25 cmH_2O PEEP HR increased (22±29 beats/min). MAP decreased during both PEEP levels, by 22±12 mmHg (2.92±1.60 kPa) during 15 cmH_2O PEEP and by 35±19 mmHg (4.66±2.53 kPa) during 25 cmH_2O. The decreases in CO and SV during 15 cmH_2O PEEP were 39% and 36%, respectively, with the corresponding differences during 25 cmH_2O PEEP of 60% and 63%, respectively.

After volume expansion HR was higher (27±35 beats/min) while MAP, CO and SV were all lower

Table 2. Hemodynamic measures before volume expansion

PEEP level	0I	15	0II	25	0III
HR, beat/min	125 ±17	159 ±38 **	126 ±20 ##	214 ±28 $$$ xxx	142 ±16 £££
MAP, mmHg	98 ±18	67 ±4 ***	105 ±17 ###	69 ±9 $$$	111 ±14 £££
(kPa)	(13.0 ±2.4)	(8.9 ±0.5)	(14.0 ±2.3)	(9.2 ±1.2)	(14.8 ±1.9)
CO, l/min	4.13±0.38	2.04±36 ***	4.31±0.63 ###	1.62±0.22 $$$	4.69±0.81 £££ c
SV, ml/beat	33.5 ±5.7	13.6 ±4.7 ***	34.9 ±7.4 ###	7.7 ±1.4 $$$ x	34.1 ±5.2 £££

The hemodynamics during the different ventilation modes before volume expansion. Parameters and units according to Table 1. Mean±SD, $n = 9$. Statistically significant differences between 0I and 15 PEEP are denoted by *, between 15 PEEP and 0II by #, between 0II and 25 PEEP by $, between 25 PEEP and 0III by £, between 0I and 0III by c and between 15 PEEP and 25 PEEP by x. One symbol for $p<0.05$, two symbols for $p<0.01$ and three symbols for $p<0.001$

Table 3. Hemodynamic measures after volume expansion

PEEP level	0I	15	0II	25	0III
HR, beat/min	150 ±13	145 ±13	149 ±12	172 ±39 x	150 ±32
MAP, mmHg	115 ±10	93 ±7 **	114 ±13 ##	79 ±13 $$$ x	119 ±23 £££
(kPa)	(15.3 ±1.3)	(12.4 ±0.9)	(15.2 ±1.7)	(10.5 ±1.7)	(15.8 ±3.1)
CO, l/min	7.02±0.85	4.30±0.64 ***	6.33±0.56 ### a	2.55±0.56 $$$ xxx	5.79±0.74 £££ ccc
SV, ml/beat	46.8 ±3.1	29.8 ±4.5 ***	42.6 ±5.5 ###	15.8 ±5.1 $$$ xxx	40.0 ±8.6 £££c

The hemodynamics during the different ventilation modes after volume expansion. Parameters and units according to Table 1. Mean±SD, $n = 9$. Statistically significant differences between 0I and 15 PEEP are denoted by *, between 15 PEEP and 0II by #, between 0II and 25 PEEP by $, between 25 PEEP and 0III by £, between 0I and 0II by a, between 0I and 0III by c and between 15 PEEP and 25 PEEP by x. One symbol for $p<0.05$, two symbols for $p<0.01$ and three symbols for $p<0.001$

Table 4. Echocardiographic measures before volume expansion

PEEP level	0I	15	0II	25	0III
RVedd, mm	16.7 ±4.0	10.0 ±3.8 ***	15.9 ±3.6 ###	7.2 ±2.9 $$$	15.4 ±3.0 £££
LVedd, mm	38.0 ±4.4	26.7 ±5.3 ***	37.2 ±3.0 ###	20.4 ±3.5 $$$ xx	37.3 ±2.6 £££
LVAed, sq.mm	1059 ±234	502 ±175 ***	993 ±153 ###	233 ±76 $$$ xx	1037 ±154 £££
LVesd, mm	29.1 ±3.0	21.4 ±5.4 ***	28.6 ±2.6 ###	16.2 ±5.1 $$$ xx	28.3 ±2.7 £££
LVAes, sq.mm	525 ±123	288 ±148 ***	528 ±123 ###	148 ±72 $$$ x	515 ±125 £££
LVFS	0.23±0.07	0.20±0.08	0.23±0.05	0.21±0.15	0.24±0.05

The echocardiographic measures during the different ventilation modes before volume expansion. Parameters and units according to Table 1. Mean ± SD, $n = 9$. Statistically significant differences between 0I and 15 PEEP are denoted by *, between 15 PEEP and 0II by #, between 0II and 25 PEEP by $, between 25 PEEP and 0III by £ and between 15 PEEP and 25 PEEP by x. One symbol for $p<0.05$, two symbols for $p<0.01$ and three symbols for $p<0.001$

during 25 cmH_2O PEEP compared to 15 cmH_2O PEEP, by 14±12 mmHg (1.86±1.60 kPa), 41% and 47%, respectively.

Echocardiographic measures

All echocardiographic measures are recorded in Tables 4 and 5.

End-diastolic measures. Before volume expansion (Table 4), no differences between the baseline measures was obtained. After volume expansion (Table 5) the left ventricular end-diastolic area (LVAed) was lower during 0III (151±92 mm^2) than 0I.

Before volume expansion, RVedd (right ventricular end-diastolic diameter) and LVedd (left ventricular end-diastolic diameter) decreased during 15 cmH_2O PEEP by 40% and 30%, respectively, and during 25 cmH_2O PEEP by 55% and 45%, respectively. After volume expansion, the corresponding decreases during 15 cmH_2O PEEP were 31% and 24%, respectively, and 46% and 38%, respectively, during 25 cmH_2O PEEP.

When comparing 15 and 25 cmH_2O PEEP before volume expansion LVedd as well as LVAed (left ventricular area end-diastolic) decreased during 25 cmH_2O PEEP by 24% and 54%, respectively. After volume expansion decreases were observed in RVedd, LVedd and LVAed by 26%, 18% and 39%, respectively.

End-systolic measures. When comparing the different baseline measures, no differences were observed. Before volume expansion (Table 4) left ventricular end-systolic diameter (LVesd) decreased during 15 cmH_2O PEEP by 26% and during 25 cmH_2O PEEP the corresponding decrease was 43%.

After volume expansion (Table 5) decreases were noticed in LVesd during 15 cmH_2O by 20% and during 25 cmH_2O by 32%.

Before volume expansion decreases were observed during 25 cmH_2O PEEP in LVesd (24%) and LVAes

Table 5. Echocardiographic measures after volume expansion

PEEP level	0I	15	0II	25	0III
RVedd, mm	20.0 ±2.5	13.9 ±2.5 ***	18.9 ±3.4 ##	10.3 ±3.2 $$$ x	18.0 ±2.9 £££
LVedd, mm	40.7 ±2.0	31.0 ±4.1 ***	40.6 ±1.9 ###	25.3 ±5.8 $$$ xx	39.9 ±3.1 £££
LVAed, sq.mm	1275 ±114	697 ±113 ***	1209 ±110 ###	424 ±157 $$$ xxx	1125 ±117 £££ c
LVesd, mm	29.7 ±1.1	23.9 ±4.5 **	29.9 ±1.7 ##	20.3 ±6.5 $$$	29.1 ±2.4 £££
LVAes, sq.mm	585 ±93	361 ±97 ***	548 ±82 ###	255 ±160 $$$ x	551 ±105 £££
LVFS	0.28±0.04	0.23±0.07	0.26±0.03	0.21±0.10	0.27±0.03

The echocardiographic measures during the different ventilation modes after volume expansion. Parameters and units according to Table 1. Mean ± SD, $n = 9$. Statistically significant differences between 0I and 15 PEEP are denoted by *, between 15 PEEP and 0II by #, between 0II and 25 PEEP by $, between 25 PEEP and 0III by £, between 0I and 0III by c and between 15 PEEP and 25 PEEP by x. One symbol for $p<0.05$, two symbols for $p<0.01$ and three symbols for $p<0.001$

(49%) when compared to 15 cmH_2O PEEP. After volume expansion a decrease was observed only in LVAes by 29%.

Fractional changes. No statistically significant changes in left ventricular fractional shortening (LVFS) were observed throughout the experiment.

Correlations. LVedd and RVedd were found to be correlated to stroke volume with individual correlation coefficients of 0.83–0.97 on the left side and 0.77–0.97 on the right (Figs. 2 and 3). The difference between end systolic and end diastolic cross sectional areas correlated to SV measured by thermodilution, $r = 0.79$ (Fig. 5).

Discussion

The experimental design of the present study was essentially the same as we have used earlier [7, 8, 11]. Myocardial contractility was evaluated by observing the change in stroke volume (SV) following blood volume expansion. This time, however, the procedure for volume expansion was modified and we used 500 ml 6% dextran 70 instead of the same amount of autologous blood. The modification lead to some differences which could be seen even before volume expansion. In the previous studies the animals were depleted of 500 ml of blood prior to baseline measurements. Supposedly this explain the substantially lower CO levels observed in the earlier studies.

Furthermore, the modification influenced the response to volume load. In the present study the augmentation in CO was much more pronounced. This probably reflects the difference in volume expanding ability between whole blood and 6% dextran 70.

We have previously not applied as much as 25 cmH_2O PEEP in a closed chest setting. According to the acid-base balance the animals sustained this pressure level well inspite of the rather excessive decreases in CO

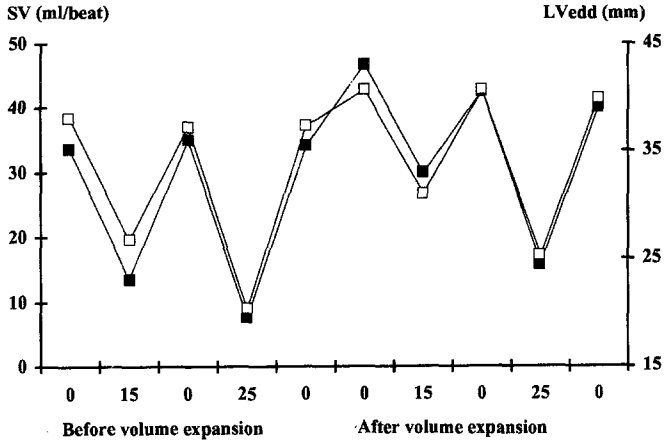


Fig. 2. The stroke volume (SV, ml/beat) and left ventricular end diastolic diameter (LVedd, mm), before and after volume expansion, during the different ventilation modes. Mean values, $n = 9$. *SV*: —■—. *LVedd*: –□–

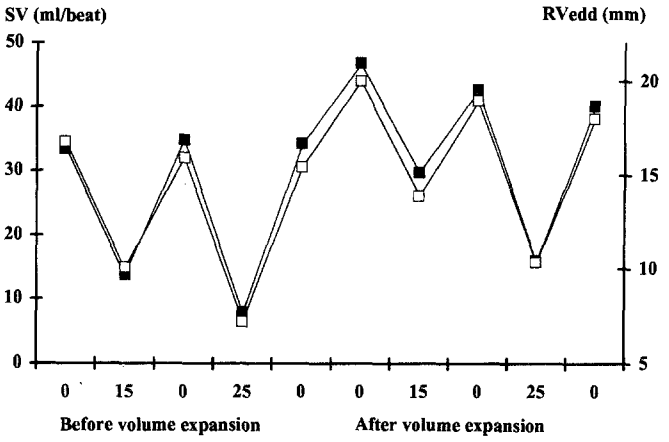


Fig. 3. The stroke volume (SV, ml/beat) and right ventricular end diastolic diameter (RVedd, mm), before and after volume expansion, during the different ventilation modes. Mean values, $n = 9$. *SV*: —■—. *RVedd*: –□–

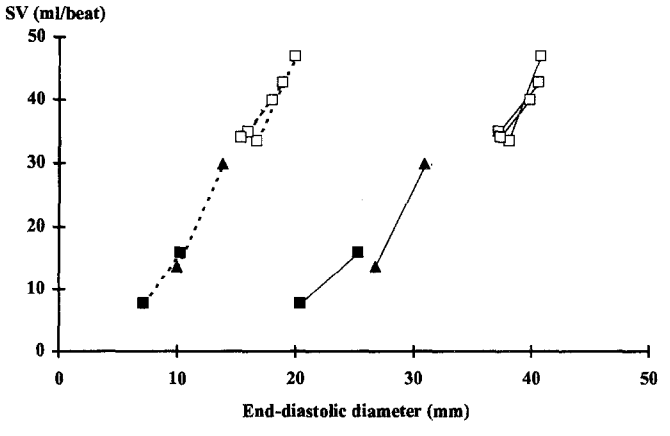


Fig. 4. The left (LVedd) and the right (RVedd) ventricular end-diastolic diameter plotted versus the corresponding SV, before and after volume expansion during the different ventilation modes. –□– 0 cmH_2O PEEP/*LVedd*. —▲— 15 cmH_2O PEEP/*LVedd*. —■— 25 cmH_2O PEEP/*LVedd*. –□– 0 cmH_2O PEEP/*RVedd*. –▲– 15 cmH_2O PEEP/*RVedd*. –■– 25 cmH_2O PEEP/*RVedd*

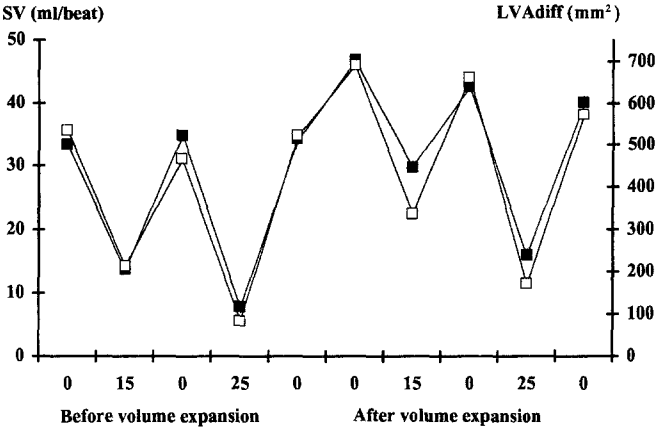


Fig. 5. The stroke volumes (SV, ml/beat) and differences between left ventricular end diastolic and end systolic areas (LVAdiff, mm^2), before and after volume expansion, during the different ventilation modes. Mean values, $n = 9$. *SV*: —■—. *LVAdiff*: –□–

and SV. Compared to 0II, the figures before volume expansion were 62% and 78%, respectively, and after volume expansion 60% and 63%, respectively. There were also marked decreases in MAP presumably activating the baroreceptors and thereby the sympathetic nervous system [15, 16]. The increases in HR in the present study may serve as an indicator of the activation.

The use of echocardiography involved some technical problems. The conventional precordial position of the probe did not work due to interposition of lung tissues between the heart and the probe. Neither did the transoesophageal approach work, again because of interposition of lung tissues. We solved the problem by preparing a substernal pouch where we placed the tip of the transoesophageal probe.

A further problem concerns the geometry of the right ventricle. Its crescent-shape, partially overlapping the right atrium as well as the left ventricle, makes it difficult to obtain a stable two dimensional scan. Already a slight shift in the position of the heart relative to the probe may change the echocardiographic presentation of the right ventricle. Thus, we occasionally observed cycles with larger end-systolic than end-diastolic right ventricular diameters (RVedd). This is also the reason why we confined the presentation of the right heart dimensions to RVedd. The conditions on the left side are more favourable due to the spherical shape of the left ventricle. Here we found it appropriate to use end-diastolic and end-systolic measures as well as the differences and ratios between them.

End-diastolic to end-systolic differences in cross sectional areas were also computed. They correlated well with SV:s obtained with thermodilution ($r = 0.79$) and are illustrated in Fig. 5. Similar good correlations have also been reported by Terai [13] and Mitaka [14].

Several investigators have paid great attention to the behaviour of the interventricular septum during PEEP ventilation, i.e. the ventricular interdependence. Jardin et al. [12] and Guzman et al. [17] found that right ventricular strain during PEEP ventilation resulted in a leftward shift of the interventricular septum, causing a decrease in SV. Similar observations were reported by Badke et al. [18]. Displacement of the interventricular septum was occasionally observed even in the present study but the inconsistency precluded meaningful conclusions.

In our previous studies on CO deterioration during PEEP ventilation we have used the transmural mean atrial pressures to represent the ventricular end-diastolic fiber length [7, 8, 11]. By plotting these pressures against the corresponding SV before as well as after volume load, a type of ventricular function curve was obtained. A change in the slope of this curve would indicate a change in contractility.

In the present study the transmural mean atrial pressures are replaced by the end-diastolic diameters as a measure of the end-diastolic fiber length.

As seen in Figs. 2 and 3 there is a good correlation between end-diastolic diameters and the corresponding SV, with individual correlation coefficients varying between 0.83–0.97 on the left side and between 0.77–0.97 on the right. It may be noted that for a certain change in SV, the change in RVedd is half of the change in left ventricular end-diastolic diameter.

The data in Figs. 2 and 3 could be transformed to ventricular function plots similar to those used in our earlier studies (Fig. 4). SV:s before and after volume expansion were combined for the three ventilation modes and plotted against the corresponding end-diastolic diameters.

On the right side, the three lines fall along a common slope indicating an unaltered contractility during all three ventilatory conditions.

On the left side (Fig. 4), the line obtained during 15 cmH_2O PEEP appears to be steeper but statistical analysis does not reveal any significant deviation from the other two ventilatory conditions, again indicating unaltered contractility. Further support for this is provided by the left ventricular fractional shortening index, LVFS. This variable, which is derived according to the formula in Table 1, corresponds to ejection fraction and remained unchanged throughout the experiment (Tables 4 and 5).

Therefore, we could not reproduce the apparent right ventricular dysfunction observed in our previous high PEEP open chest preparation [11]. A fact which may possibly explain this discrepancy is that this time we did not reach the same excessive mean pulmonary arterial pressure. The right ventricle was not subjected to the same degree of afterload. Hence, we believe that in the intact animal, the decrease in CO during PEEP ventilation up to 25 cmH_2O can be entirely explained by impeded venous return.

Obviously, this challenges the observations made by Liebmann et al. [1], Grindlinger et al. [6], Dunham et al. [5], Hechtman et al. [19] and, especially, Patten et al. [2] in their cross circulation study.

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