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 studied during ventilation with 0, 15 and 25 cmH₂O (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

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 (Pavlunol®, 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 (FiO₂ > 0.5) except during the surgical preparation when nitrous oxide in oxygen (0.7/0.3) was used. Minute volumes were adjusted to maintain

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 cmH₂O 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 cmH₂O PEEP. We therefore applied 25 cmH₂O 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 cmH₂O 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 cmH₂O while Jardin et al. [12] applied up to 30 cmH₂O 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.
a stable PaCO₂ 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.

**Echocardiographic 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 crescent 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, Welwyn 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 (AT11 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-diastolic (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 cmH₂O of PEEP, ZEEP (0II), 25 cmH₂O PEEP and again ZEEP (0III).

**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 cmH₂O PEEP resulted in a higher HR (34±25 beats/min) which was even more pronounced (37±29 beats/min) at 25 cmH₂O 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 cmH₂O