Title: THE EFFICACY OF HIGH FREQUENCY VENTILATION IN CARDIAC TAMPONADE

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Introduction: Cardiac tamponade is characterized by a reduction in stroke volume and cardiac output caused by increased intrapericardial pressure from an accumulation of fluid in the pericardial space. Positive pressure ventilation may further decrease cardiac output by decreasing venous return to the heart and ventricular filling (1). High frequency ventilation (HFV) is associated with lower peak airway pressures, and may be beneficial to the patient with cardiac tamponade. However, the effects of HFV on tamponade have never been investigated. This study examined the hemodynamic changes in anesthetized dogs with cardiac tamponade using two different modes of mechanical ventilation, HFV and conventional mechanical ventilation (CMV).

Methods: Seven mongrel dogs were anesthetized with sodium pentobarbital, paralyzed, intubated and ventilated with 100% oxygen. Arterial and Swan-Ganz catheters were inserted. Monitored parameters included heart rate (HR), arterial pressure (AP), pulmonary artery pressure (PAP), pulmonary capillary wedge pressure (PCWP), central venous pressure (CVP), thermodilution cardiac output (CO), and pH and blood gas tensions. Airway pressure (Paw) was also monitored using a saline-filled catheter with its tip located 1 cm distal to the end of the endotracheal tube. A short thoracotomy incision was made and a pediatric nasogastric feeding tube was inserted into the pericardial cavity to produce tamponade and to measure intrapericardial pressures (IPP). After insertion of a closed pleural drain and closure of the chest wound, cardiac tamponade was induced by infusing 120-180 ml of saline, producing a resting IPP of 12 mmHg measured at apnea by disconnecting the respiratory circuit for a few seconds. After 60 min of tamponade each animal underwent four alternating, randomized trials of CMV and HFV. CMV was performed using a volume cycled ventilator at respiratory rates (f) equal to 10 and 20 breaths per minute (bpm). The tidal volume (Vt) was adjusted to maintain $PaCO_2$ between 35-40 mmHg. HFV was performed using a high frequency jet ventilator at f equal to 60 and 120 bpm, and the same steady state $PaCO_2$ was maintained by altering the driving pressure. Inspiratory-to-expiratory time (I:E) ratio was kept constant at 1:2. Arterial blood pH and base excess were maintained at 7.40±0.05 and 0±3 mmol/L by administering sodium bicarbonate. Hemodynamic parameters and blood gas values were then measured after 15 minutes in each ventilatory mode. Data from the four groups were compared using analysis of variance for repeated measurements followed by Tukey's HSD test, with p values less than 0.05 considered significant. All values are expressed as mean ± SD.

Results: Peak Paw and IPP were significantly lower and cardiac index (CI) and stroke volume index (SI) were significantly greater during HFV than they were during CMV (Table 1, Fig 1). Fig 2 shows a significant negative linear relationship between CI and peak IPP.

Discussion: The major functional disturbance caused by cardiac tamponade is a mechanical restriction of ventricular filling during diastole. This results in a reduction in stroke volume and cardiac output. In this experiment, cardiac tamponade was produced by increasing intrapericardial pressure to 12 mmHg since this pressure was found to be the maximal pressure tolerated before cardiac arrest occurred. As opposed to spontaneous ventilation, CMV increases airway pressure, which can be transmitted to the intrapericardial space, making cardiac tamponade worse. This was clearly shown in this study, since higher airway pressure due to CMV caused higher peak intrapericardial pressure, resulting in lower stroke volume. As compared to CMV, HFV did not increase airway pressure significantly since tidal volumes were smaller, resulting in better hemodynamics. However, a higher frequency (f=120) was not necessarily more beneficial for cardiac tamponade. This is probably because increasing frequency will cause an auto-PEEP effect, due to a shorter expiratory phase between respiratory cycles. The results indicate that high frequency ventilation may be superior to conventional mechanical ventilation in cases of cardiac tamponade.

Table 1

	CMV(f=10)	CMV(f=20)	HFV(f=60)	HFV(f=120)
HR (/min)	199±15	203±11	202±12	202±12
mean AP (mmHg)	92±26	109±29	119±17*	107±23
mean PAP(mmHg)	17.3±2.2	17.9±2.7	18.3±2.0	18.7±2.8
CVP (mmHg)	11.1±1.2	11.4±0.5	10.9±0.9	11.1±1.3
PCWP (mmHg)	11.9±1.7	12.3±1.0	12.9±1.2	13.3±1.3*
V_T (ml/kg)	22.5±1.7	15.5±1.6	9.1±1.3**	6.8±1.1**#
peak P_{aw} (mmHg)	14.1±3.6	11.1±3.4	7.9±2.2**	7.0±1.5**
peak IPP (mmHg)	18.7±2.4	17.0±1.4	14.6±1.8*	14.7±2.0*
CI ($L/min/m^2$)	0.8±0.4	1.0±0.2	1.5±0.3**#	1.4±0.3*

IPP = intrapericardial pressure, CI = cardiac index
*(p<0.05) **(p<0.01) as compared with CMV(f=10)
#(p<0.05) as compared with CMV(f=20)

Figure 1

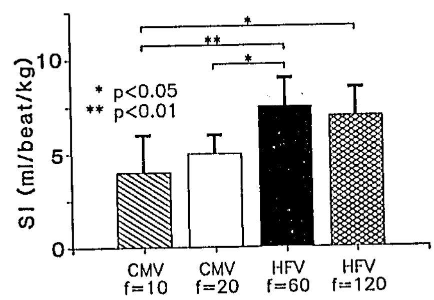
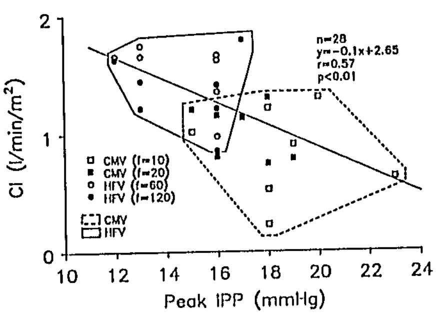


Figure 2



Reference: (1) Moller CT et al: Haemodynamics of cardiac tamponade during various modes of ventilation. Br J Anaesth 51:409-414, 1979