Automated ventilation with a novel small device based upon expiratory ventilation assistance improves hemodynamics in hemorrhagic shock

Paul M. Heerdt MD, PhD, David A. Berlin MD, Harrison Peprah-Mensah BS, Seth Manoach MD
Departments of Anesthesiology, Pharmacology, and Medicine (Critical Care), Weill Cornell Medical College, New York, NY
Sponsor: Dolphys Medical BV, Eindhoven, NL

INTRODUCTION

Positive pressure ventilation (PPV) impairs venous return and augments hypotension during hemorrhagic shock, especially if end-expiratory pressure (EEP) is applied. Previous data indicate that negative airway pressure during the exhalation phase of PPV may improve hemodynamics in hemorrhagic shock (1). A hand-held device (Ventrain, Dolphys Medical BV, Eindhoven, NL) was recently released that uses high flow oxygen passing through a T-piece to first deliver gas via a small bore cannula for inhalation and then create a Bernoulli-effect suction to facilitate exhalation, a process known as Expiratory Ventilation Assistance (EVA) (2). *The present pilot study tested the hypothesis that a prototypical small automated ventilator based upon the EVA principle could be used to generate a controlled period of negative EEP and improve hemodynamics relative to PPV in hemorrhagic shock.*

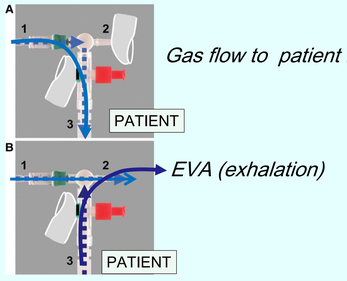
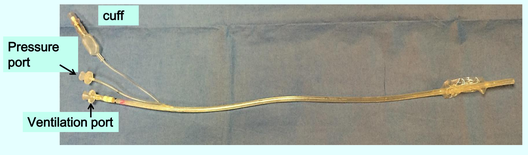


Figure 1. Representation of the EVA principal in the commercially available device. The central feature is regulation of flow thru a t-piece.

(from: Hamaekers AE, van der Beek T, Theunissen M, Enk D. *Rescue ventilation through a small-bore transtracheal cannula in severe hypoxic pigs using expiratory ventilation assistance.* Anesth Analg. 2015 Apr;120(4):890-4

METHODS

Under an IACUC-approved protocol, data from 8 anesthetized pigs were used for the study. Animals were instrumented to measure mean systemic (mAP) and pulmonary arterial (mPAP) pressures, central venous pressure (CVP), left ventricular (LV) pressure and volume, and cardiac output (CO) both by bolus thermodilution (measured at 20 min intervals during shock and regarded as the standard) and continuous NICOM (Cheetah Medical). After preparation, 40 ml/kg blood was removed over 30 minutes to produce a mAP of ~25 mmHg. Half the animals were then maintained on PPV (9 cc/kg tidal volume, O EEP) while the other half were changed to EVA delivered via a cuffed, 2 mm internal diameter endotracheal catheter (figure 2) by a small prototype ventilator controlled with a hand-held tablet (Microsoft Surface Pro). With the device, EEP was progressive decreased to – 8 cmH_2O and peak inspiratory pressure (PIP) adjusted to maintain tidal volume. Minute ventilation was matched between groups, and arterial blood gases (ABG) drawn at regular intervals. Shock was maintained for 60 minutes before resuscitation with shed blood and crystalloid. After resuscitation, ventilation was then maintained with PPV (9 cc/kg tidal volume, O EEP) in all animals. Between groups, mAP, CO, and ABG data before, during and after hemorrhage were compared by t-test as the primary outcome variables.



➢ **Figure 2**. The "EVAcath", a customized, 2 mm internal diameter (4 mm OD), cuffed catheter with a distal port for monitoring airway pressure.

RESULTS

SUMMARY: Even at baseline, a transition from positive to negative EEP augmented ventricular filling and pressure (figure 3 A). After hemorrhage, EVA with negative EEP substantially increased left ventricular volume and pressure (figure 3 B). As shown in figure 4, following hemorrhage sequential reductions in PIP and EEP produced sequential increases in cardiac output (Figure 4). Overall, there were no control vs EVA differences in mAP or CO at baseline, the end of blood removal, or following resuscitation. During shock, mAP and CO in the EVA group were increased relative to control after 20 minutes with these differences even more prominent after 60 minutes (figure 5). With minute ventilation matched, arterial pH, PaO_2/FiO_2, and $PaCO_2$ were not different between control and EVA at the end of hemorrhage, but were after the 60 minute shock interval (table 1).

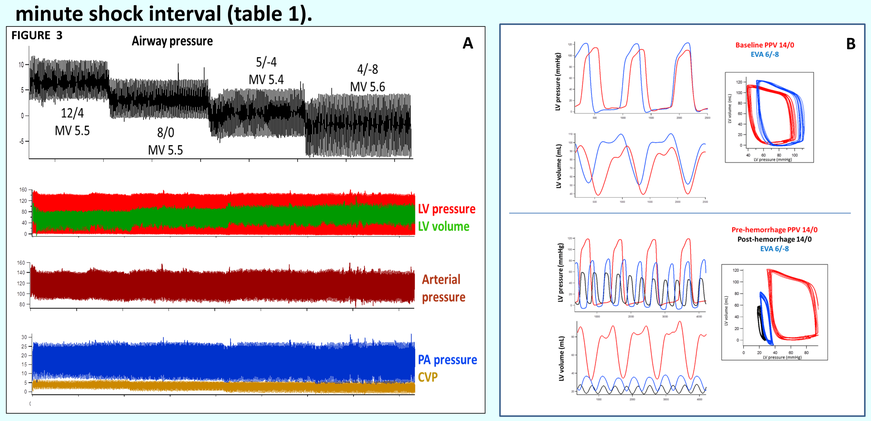


Figure 3. ***Panel A***. Representative recordings during ~10 minutes of automated EVA ventilation showing the effect of decreasing **airway pressure in black** (cmH_2O) on **left ventricular (LV) pressure in red** (mmHg), **LV volume in green** (mL), **systemic arterial pressure in brown** (mmHg), **pulmonary arterial (PA) pressure in blue** (mmHg), and **central venous pressure (CVP) in tan** (mmHg). ***Panel B***. Comparative pressure/volume measurements with different modes of ventilation, before and after hemorrhage with positive pressure ventilation (PPV) and EVA (PIP/EEP).

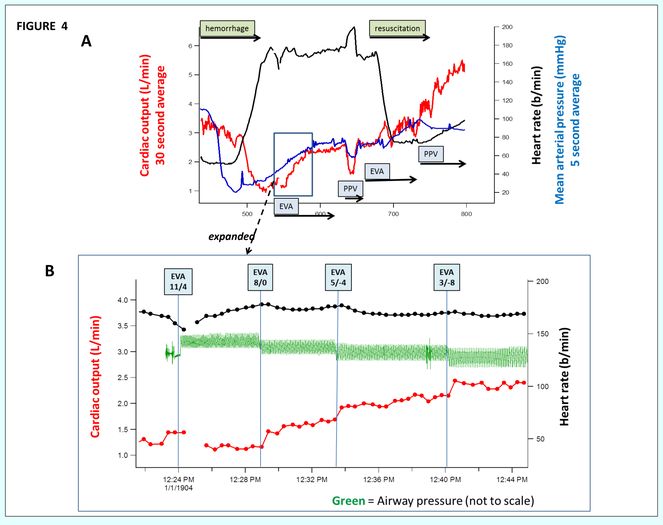


Figure 4. ***Panel A:*** The effect of hemorrhage and conversion to EVA on NICOM **cardiac output**, **heart rate**, and **mean arterial pressure**. With conversion from EVA to positive pressure ventilation (PPV) during shock, there was a decline in blood pressure and cardiac output, and a rise in heart rate. ***Panel B*** depicts the expanded effect of progressively decreasing inspiratory and expiratory pressures (shown as x/y) during initiation of EVA.

Table 1. Ventilation and arterial blood gas data comparing positive pressure ventilation (PPV) to expiratory ventilation assistance (EVA) before, during, and after hemorrhage shock. At baseline and post-resuscitation, both groups received PPV. MV= minute ventilation; TV = tidal volume; PIP = peak inspiratory pressure; EEP = end expiratory pressure; BE = base excess. Values in **red** indicate EVA difference from PPV ($p \leq 0.05$).

	baseline				end hemorhage				shock 60 min				end resuscitation				30 min post resusc			
	PPV		EVA		PPV		EVA		PPV		EVA		PPV		EVA		PPV		EVA	
	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD
MV (L/min)	5.2	0.5	5.1	0.5	5.2	0.5	5.1	0.5	5.2	0.4	5.3	0.8	5.3	0.4	5.3	0.9	5.4	0.2	5.4	0.7
TV (mL)	420	26	434	40	420	26	428	38	415	17	427	140	418	20	409	132	416	17	445	14
Rate (b/min)	12.3	0.5	11.8	0.5	12.3	0.5	12.0	0.0	12.5	0.6	13.3	3.9	12.5	0.6	14.0	3.9	13.0	1.0	12.5	2.1
PIP (cmH2O)	19.3	1.0	19.0	0.8	19.3	1.0	18.5	0.6	20.0	2.2	**5.8**	2.4	20.3	1.0	**5.8**	2.4	20.7	0.6	20.3	3.3
EEP (cmH2O)	4.0	0.0	4.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	**-8.8**	1.0	0.0	0.0	**-8.8**	1.0	4.0	0.0	4.0	0.0
pH	7.48	0.01	7.50	0.03	7.40	0.06	7.44	0.04	7.41	0.08	**7.29**	0.14	7.35	0.17	7.30	0.13	7.45	0.06	7.50	0.0
PCO2 (mmHg)	46	2	42	4	49	5	43	5	44	10	**66**	17	57	12	64	14	45	3	42	4
PO2/FiO2	556	46	555	32	451	91	410	42	485	83	**382**	214	499	92	354	54	265	114	323	191
BE (mEq/L)	10.7	1.0	10.1	1.2	5.7	6.5	5.0	2.4	4.1	11.3	6.3	2.3	6.2	8.7	6.5	4.0	7.8	3.0	9.3	4.1
Hemoglobin (g/dL)	10.3	0.9	10.1	1.3	9.0	0.5	9.2	0.7	8.5	0.6	9.8	0.8	10.1	1.4	10.4	1.2	9.3	2.4	8.8	1.2
Lactate (mEq/L)	2.5	0.8	2.2	1.3	5.3	4.4	4.8	1.7	7.6	7.6	5.1	1.8	6.0	6.7	5.4	2.6	2.2	1.3	2.4	1.1

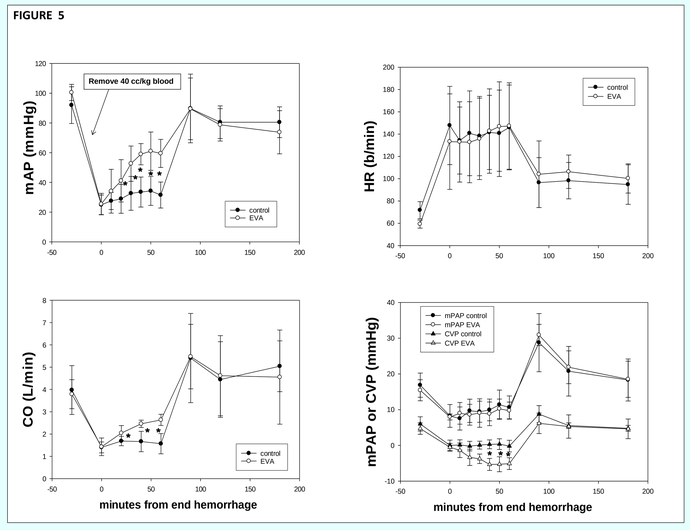


Figure 5. Comparison of mean arterial pressure (mAP), thermodilution cardiac output (CO), heart rate (HR), mean pulmonary arterial pressure (mPAP), and central venous pressure (CVP) during PPV and EVA. During shock, cardiac output measurements were made at 20 minute intervals. * designates a difference between PPV and EVA at the same time point.

CONCLUSIONS

These pilot data support the hypothesis that a small, automated ventilator based upon the EVA concept can improve hemodynamics in hemorrhagic shock. However, the data also indicate that this mode of ventilation may be less efficient than conventional PPV, thus requiring a higher minute ventilation for CO_2 removal.

References

1. Crit Care Med. 2006;34:2175-81
2. Anesth Analg. 2015;120:890-4