

Do vented chest seals differ in efficacy? An experimental evaluation using a swine hemopneumothorax model

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Running Head: Efficacy of vented chest seals

Abstract

Objective: Hemopneumothorax (HPTX) is the second leading cause of potentially preventable death among combat casualties. We investigated the ability of five FDA-approved non-occlusive chest seals (CSs) to seal a bleeding chest wound and prevent tension HPTX in a swine model.

Methods: Following instrumentation, an open chest wound was created in the left thorax of spontaneously air-breathing anesthetized pigs (n=26, 43 Kg). Autologous fresh blood (226 mL) was then infused into the pleural cavity to produce HPTX. The chest wounds were then sealed with CSs. The sealant strength and venting function of CSs were challenged by infusion of 50 mL more blood directly into the chest wound and incremental air injections into the pleural cavity. Tension HPTX was defined as intrapleural (IP) pressure $\geq +1$ mmHg and $>20\%$ deviation in physiological measurements.

Results: An open chest wound with HPTX raised IP pressure (~ -0.7 mmHg) and caused labored breathing and reductions in PaO₂ and SvO₂ (P<0.01). Sealing the wounds with the CSs restored IP pressure, and improved breathing and oxygenation. Subsequent blood infusion into the wound and IP air injections produced CS-dependent responses. CSs with one-way valves (Bolin and SAM) did not evacuate the blood efficiently; pooled blood either detached the CSs from skin and leaked out (75%), or clotted and clogged the valve and led to tension HPTX (25%). Conversely, CSs with laminar venting channels allowed escape of blood and air from the pleural cavity and maintained IP pressure and oxygenation near normal levels. Success rates were 100% for

Sentinel and Russell (6/6); 67% for HyFin (4/6); 25% for SAM (1/4); and 0% for Bolin (0/4) CSs ($p=0.002$).

Conclusion: The sealant and valve function of vented CS differed widely in the presence of bleeding chest wounds. Medics should be equipped with more effective CSs for treating HPTX in the field.

Key words

Chest seal, pneumothorax, hemopneumothorax, tension hemopneumothorax, animal study, swine.

Introduction

During 10 years of war in Iraq and Afghanistan, thoracic injuries accounted for 8.6% of the total number of wounded US military personnel and resulted in an overall mortality rate of 8.3%. Penetrating trauma from explosive devices was the most common cause of injury and was mostly associated with pneumothorax (PTX) and pulmonary contusion (1). The mortality of these patients with chest injuries was higher than those injured in Korean and Vietnam wars (1). During the same decade, airway compromise was identified as the second leading cause of potentially preventable death (8%) among US casualties the majority of which (87.5%) was caused by airway obstruction and the rest resulted from tension PTX (2).

Until 2013 the Tactical Combat Casualty Care (TCCC) Guideline had recommended the use of occlusive (non-vented) chest seals (CSs) for treating an open pneumothorax (sucking chest wound) injury followed by close monitoring of the patient (3). No mention was made of whether or not a vented chest seal was preferable, because there was no evidence at the time to show that these one-way valves were effective at preventing the development of a tension pneumothorax. New experimental data collected in spontaneously breathing swine with an open chest wound showed persistent tension PTX development when the wound was sealed with an occlusive chest seal (4). In contrast, the use of a vented chest seal maintained IP pressure at normal levels and prevented tension PTX (4). These findings and earlier experimental (5, 6) and clinical evidence (7, 8) prompted revising TCCC guideline that now instructs “All open and/or sucking chest wounds should be treated by immediately applying a vented chest seal to cover the defect” (9).

Currently as many as seven non-occlusive CSs are cleared by the Food and Drug Administration for treatment of open chest wounds and prevention of tension physiology. The effectiveness of these chest seals partly depends on their adhesive properties especially in the presence of blood and other contaminants, and partly determined by their valve or vent function to evacuate air and blood from the pleural cavity and maintain IP pressure near normal level (i.e., ease respiration and improve oxygenation). The function of some of these CSs has been examined in earlier studies in swine models (6, 10). Those results indicated that all tested chest seals with either a laminar vent or a one-way valve were equally effective in evacuating blood and air from pleural space and preventing tension PTX.

In this study, we selected five non-occlusive CSs for further evaluation among seven choices based on their superior skin adhesion and valve function as reported previously (11) and the results of our own pilot test of all seven devices in pigs with bleeding chest wounds. The selected five CSs (Fig. 1A) were examined for their adhesion strength to seal a bleeding chest wound and their valve/vent function to allow release of air and blood from the pleural cavity and maintain IP pressure near normal level [i.e., prevent tension hemopneumothorax (HPTX)]. An open chest wound model with active bleeding was developed in pigs for measuring physiological changes that occur following CSs application and air and blood introduction into the pleural cavity.

MATERIAL AND METHODS

This study was approved by the Institutional Animal Care and Use Committee of the U.S. Army Institute of Surgical Research and conducted in compliance with the Animal Welfare Act and the implementing Animal Welfare Regulations. All animals received care and were used in accordance with the principles of the *Guide for the Care and Use of Laboratory Animals* (12).

Immature female Yorkshire pigs (~43 kg, n= 26) were induced with an IM injection of tiletamine-zolazepam (Telazol, 4-6 mg/kg), intubated, secured on the operating table in a supine position and mechanically ventilated with inhalation anesthesia (1% to 3% isoflurane) for instrumentation as described (4). Briefly, the right carotid artery was cannulated for continuous recording of vital signs, and blood sampling. The right external jugular vein was also accessed and a Swan-Ganz catheter (oximetric PA catheter) was inserted for continuous measurement of central venous pressure, (CVP), pulmonary arterial pressure (PAP), mixed venous oxygen saturation (SvO₂) and cardiac output (CO). The left femoral artery was cannulated for collecting fresh blood used. Maintenance fluid (LR at 3-5 ml/kg/hr) was administered through an ear vein catheter and urine output was measured *via* a Foley catheter. Body temperature was maintained at 37° to 39°C. The skin on the chest area where the chest wound was created and sealed with CSs was shaved, cleaned with alcohol and dried before proceeding with the injury.

Following instrumentation, the animal was placed in a sternal recumbent position, mechanical ventilation and isoflurane anesthesia was discontinued. The animal was allowed to breathe spontaneously (medical air) for the rest of the experiment. Intravenous anesthesia was provided

by continuous infusion of propofol (5- 10 mg/kg/hr) with buprenorphine (2-10 µg/kg/hr) through an ear vein.

Intrapleural (IP) cannulation and chest injury: Prior to chest injury, a 14 Ga central venous catheter was inserted percutaneously into the left pleural space at the 6th intercostal space. This IP catheter was used for incremental air injection into the pleural cavity and measurement of IP pressure with a digital manometer. The baseline IP pressure was measured immediately after placing this catheter.

A reproducible open chest wound (10.5 mm diameter) was created at the 5th intercostal space on the axillary line. An incision was made through the dermis and underlying muscles were dissected bluntly until the pleural space was entered. The hole was then expanded by inserting a small trocar (Throacoport™, 10.5 mm, Covidien) and maintained open by leaving the trocar sleeve in the wound track. The top portion of the sleeve was then cut off flush with the skin. Next, a 16 Ga venous catheter was inserted percutaneously into the chest wall 8-10 cm above the chest wound (without penetrating the pleural cavity) and extended parallel to the ribs until it entered into the wound track near skin surface. This catheter was used for slow infusion of fresh blood into the wound track to mimic a bleeding chest wound (Fig. 1B).

Experimental Design: Once the chest injury and cannulation were completed, the chest hole was plugged with a rubber stopper and excess air was evacuated with a syringe reestablishing previously measured baseline IP pressure (-7 to -8 mmHg inspiration). Next, a 15-min stabilization period was allowed and then baseline values for hemodynamic and respiratory

parameters [tidal volume (V_t) and respiration rate (RR)] were recorded. An arterial blood sample (baseline) was collected for blood gas analysis (ABG).

To induce HPTX, the chest plug was removed and approximately 10% of each pig's blood volume (average of 226 mL) was withdrawn from the femoral artery (at 25 ml/min without adding anticoagulant) and simultaneously infused into the pleural cavity through the chest wound using a Masterflex pump. Hemodynamic and respiratory parameters were then recorded (0 min time point) and the chest wound covered and sealed with one of the vented/valved CSs. **The skin around the chest wound cleaned with gauze and dried before CSs were placed.** Caution was made to place the valve(s) or central venting section of each chest seal directly over the open chest wound. Physiological changes were measured 10 minutes after placing the chest seals (10 min time point). Next, the first increment of air (0.25 L) was injected into the pleural space. The initial air infusion was followed by slow infusion (5 ml/min) of 50 mL additional fresh blood -- simultaneously withdrawn from the femoral artery-- into the wound track. This procedure was done to mimic a bleeding chest wound after it was covered and sealed with a chest seal. Subsequent physiological changes were recorded after the 50-mL blood infusion was completed (at 20 min time point). Arterial blood samples were collected for ABG analysis at 10 minute intervals. Air injections (0.25 L each time), blood sampling and data recording (10 minutes after each air injection) continued until either 2 liters of air (~ 100% of the total lung capacity) was injected into the pleural cavity without causing tension HPTX, or discontinued if the chest seal detached from the wound (became nonfunctional) or tension HPTX developed. Tension HPTX was diagnosed in pigs according to earlier studies (13, 4) when mean IP pressure > +1 mmHg and significant deviations (from baselines) were measured in 4 of the following 5 parameters:

30% rise of PAP, 30% fall of SvO₂, and 20% fall of V_t, MAP, or CO. Development of tension HPTX or lack of it was confirmed with a final chest X-ray. Animals were euthanized after obtaining final blood samples.

Based on previous adhesion data and our own pilot test on pig skin, five out of seven vented CSs were selected and tested in this study. The Bolin vented chest seal was included as a control because it is preferably carried by military first responders and was recognized as one of the highest-rated vented CSs in the Navy equipment survey (14). The physical characteristics of the five vented CSs are listed in table 1. Asherman™ and Fast Breathe Thoracic seal™ were not included in this study due to either poor skin adherence properties (9, 11) or having a valve similar to those of selected CSs. Each chest seal was intended to be tested in six animals, however, if after 4 experiments a chest seal failed to function properly twice, further testing of that device was discontinued.

Data Analysis

Normality of distribution of data was tested with the Kolmogorov-Smirnov test. Treatment effects at selected time points (baseline, HPTX, CSs placed, CS+1.25 L PTX) were analyzed by one-way analysis of variance (ANOVA) with pairwise comparisons using the Tukey test. Incidence of successful experiments comparing all five CSs was conducted using chi-square. Pair wise comparison of successful incidences (Bolin vs. other four CSs) was done using Fisher's Exact test with Bonferroni adjustment of p value. Physiological measurements and

laboratory data are expressed as mean \pm SEM and a $p < 0.05$ was considered statistically significant.

Results

Laboratory analysis of baseline blood samples and respiratory and circulatory measurements before the start of surgery did not differ among the five groups of pigs. These values were within normal ranges for Yorkshire crossbred pigs as determined in our laboratory.

Effects of HPTX and chest seal application on respiratory function: As expected, the open chest injury (PTX) and initial blood infusion (226 ± 7.5 ml; no difference among groups) into the pleural space producing HPTX significantly raised IP pressure during inspiration (from -7 to -0.7 mmHg, Fig. 2). This condition also had visible effect on the pigs' spontaneous breathing efforts (labored breathing). HPTX also resulted in a significant reduction in tidal volume (37%, 262 to 164 mL), a moderate increase in respiration rate (22%, 18 to 22 breath/min) and an overall 19.5% decrease ($p < 0.01$ vs. baselines) in minute volume (4.6 to 3.7 L/min) in all animals (Fig. 3) with no differences among groups. These respiratory changes in turn caused significant decreases in blood oxygenation as measured by a 29% drop in PaO₂ (148 to 105 mmHg, $p < 0.01$, Fig. 4) and a 16% (80.5% to 67.5%, $p < 0.01$) decrease in SvO₂ compared to baselines (Fig. 5). Elimination of CO₂ from blood, however, was less affected (PaCO₂ increased 14%, 52 to 59 mmHg, $p < 0.01$). **The deleterious changes in breathing and oxygenation measured here following HPTX represent the consequences of untreated chest wound (control) in this model.** Sealing the chest wounds with one of the vented/valved CSs immediately improved respiration and restored

IP pressure and respiratory minute volumes in all animals (no statistical difference compared with baselines, Fig. 2 and 3). However, at ten minutes after chest seal application an oxygenation deficit still persisted as PaO₂ rose only by 20% and SvO₂ did not change significantly (Figs. 4 and 5).

Subsequent 50 mL blood infusion into the wound track that mimicked a bleeding chest wound and IP air infusions produced different physiological effects depending on the adhesiveness and the valve/vent function of each chest seal. The laminar vent channels present on Sentinel, Russell and HyFin CSs, allowed blood and air to escape from the pleural space and prevented increase of IP pressure and development of tension HPTX in the treated animals (Fig. 2). Repeated measurements of PaO₂, PaCO₂, and SvO₂ also did not show any significant change throughout the experiment (Figs. 4 and 5). In case of HyFin CSs, however, the adhesive seal broke in two experiments after infusing ~ 1 liter air into the pleural cavity. These CSs partially detached from the skin (on the edge) and opened to the outside atmosphere, but their central section covering the chest wound continued to function covering the wound and preventing IP air entry during inspiration but detaching to allow air escape from pleural cavity. The adhesive failure of these two CSs accounts for the overall higher IP pressure (-3.5 mmHg) in the HyFin group (Fig. 2).

In contrast, flutter valves, present on Bolin and SAM CSs, were either inefficient (SAM) or impermeable (Bolin) to blood passage. Shortly after the start of blood infusion into the wound track and leakage of a few milliliters of blood, these valves became clogged with blood/ blood clots and stopped functioning. As a result when IP air injection started, IP pressure increased and animals' respiration became more labored. Accumulated blood and increasing IP pressure

behind the valves gradually loosened the adhesion and eventually separated the CSs from skin, producing a wide channel through which air and blood leaked out, thereby causing the IP pressure to return to zero mmHg. These experiments were, therefore, terminated because the CSs were no longer functional. In two cases wherein CSs adhesion held for longer periods, tension HPTX developed after about 1.25 liter IP air injection (one with Bolin and one with SAM use). Only one SAM CSs continued to seal the wound and its valve remained functional (partially clogged with blood clot) during the experiment (data for this animal is shown as a dotted line in the graphs). Premature detachment of Bolin and SAM CSs (poor adhesion properties) avoided development of tension HPTX in more animals treated with these products.

Effects of HPTX and chest seal application on circulatory function: Changes in circulatory function were less prominent than those measured in respiratory function. The more consistent changes that occurred following PTX and HPTX (before chest seal application) were the increases of CVP and PAP which coincided with ~ 20% reduction in CO in all animals. Sealing the chest wound with any of the CSs did not improve these parameters. Subsequent IP air infusions also had no additional detrimental effects except on the two animals that developed tension HPTX. The CVP and PAP sharply rose and CO further declined when tension physiology developed in those animals. No significant change in HR occurred in animals that were treated successfully with a vented CSs. HRs increased by 24% in the two animals that developed tension HPTX. MAP did not change after chest injury or during blood and air injection in most animals with the exception of a 10-15% increase at the time of tension HPTX development in the 2 pigs noted above.

Successful HPTX treatment with CSs was defined as: 1) the chest wound remained sealed with the device; and 2) IP pressure increase was prevented (stayed near normal level) throughout the experiment. Based on this definition, 100% of Sentinel and Russell (6/6), 67% of HyFin (4/6), 25% of SAM (1/4), and 0% of Bolin (0/4) CSs were successful in treating HPTX ($p=0.002$). Sentinel and Russell performance was superior to the control (Bolin) chest seal ($p=0.02$).

Discussion

The purpose of this study was to examine the effectiveness of five vented/valved CSs in sealing an open chest wound and preventing tension physiology in patients with an open and bleeding chest wound. This was tested in a HPTX model in spontaneously breathing swine (sternal position) with an open bleeding chest wound on their right hemithorax. The potential for preventing tension HPTX was tested by sealing the bleeding chest wound with a chest seal and then incrementally injecting air into the pleural cavity that also contained a pool of blood (HPTX). The results indicated that the CSs with laminar vent channels were much more successful in sealing the chest wound and evacuating air and blood from the pleural cavity than those with one-way valve(s) in their centers.

A previous study in swine reported no difference in adhesive properties of the four CSs (Bolin, Russell, HyFin, and SAM) that were tested here. All four were found to be adequately adherent and capable of sealing a hypothetical chest wound even in presence of blood and sand contaminants on the skin (11). Chest seal adherence was also examined in a human study with simulated chest wounds (15). TCSs were applied on the chest and back of the 31 adult

volunteers that were sprayed with condensed milk to simulate blood and then quantified for adherences. Although numerical differences were not statistically significant, the author concluded that SAM and Bolin chest seals were more effective in retaining adherence compared with other devices (Asherman, HyFin, and Russell). Our adherence observations were quite different than those reported in the above studies. We found that when an actively bleeding chest wound is covered with a chest seal with flutter valves (Bolin and SAM), blood cannot pass through the valves and accumulates blood behind the valves weakening the adhesiveness and eventually causes the CSs to detach from skin. Such devices therefore become nonfunctional.

Other experimental studies compared the efficacy of some non-occlusive CSs (Bolin, Asherman, HyFin, Sentinel, and SAM) in other PTX and HPTX swine models and found no differences among them (6, 10). Their data indicated that vented and valved CSs were equally effective in evacuating blood and air from a communicating PTX and HPTX and preventing tension physiology. The contrasts between the results of these studies and ours are due to differences in the experimental model of PTX and HPTX used and the physiological measurements that were made to determine tension physiology. In those studies: 1) the CSs were tested in non-bleeding chest wounds; 2) diagnosis of tension PTX was defined as a 20% decrease in MAP or a 20% increase in HR, which did not occur when the chest wounds were covered with CSs and challenged with IP air injections. In our experiments, HR increased >20% in the two pigs that developed tension HPTX, however, MAP did not decrease even when tension HPTX was apparent and animal breathing critically compromised (i.e., cyanosis). In our model, the change in IP pressure was the best indicator for determining how well the CSs' vents/valves were functioning. Other physiological measurements (V_t , PaO_2 , SvO_2) were also taken into account to

determine development of tension physiology; and 3) pigs were placed on their back (supine position) on the operating table during experimentation. This position prevented IP injected blood to come in contact with the valves and compromise their functions. This supine position was shown to be associated with hypoxia and increased pulmonary vascular shunt in spontaneously breathing four-legged animals (16).

Our earlier swine study (4) showed that unlike occlusive (non-vented) CSs, application of a chest seal with valve (Bolin) on an open non-bleeding chest wound (PTX) successfully maintained normal IP pressure (-5 mmHg) and prevented tension PTX following multiple air injections (up to 2 liters) into the pleural cavity. This chest seal securely sealed the wound for 1 hr and the flutter valve functioned properly in allowing one-way air escape from chest to outside in the absence of blood contamination. Infusion of blood (up to 10% of blood volume) into the pleural cavity also did not interfere with valve function as long as the blood remained in the pleural cavity and did not leak outwards (personal observation, 6, 10). However, depending on the chest wound and patient position the possibility of an open chest wound with bleeding (originating within the chest) that exits through the chest defect always exists. Therefore, in this study we created a HPTX model that included an actively bleeding wound to test the efficacy of different non-occlusive chest seals. To create a reproducible bleeding wound, we infused 50 ml fresh blood directly into the wound track to expose each chest seal to the same bleeding condition. The results clearly showed that unlike laminar vent channels, one-way valves in some CSs will soon clog with blood/blood clots preventing air escape from the pleural cavity and increasing the risk of tension HPTX. Tension HPTX occurred in two animals (one with Bolin and one with SAM) in this study. However, tension HPTX might have occurred more frequently had these CSs

remained attached to the skin and sealed the chest wound for longer times. It is important to mention that failure of valved CSs occurred approximately one hr after application and after one liter air infusion that significantly increased IP pressure. These chest seal may still be useful for managing HPTX in civilian patients with rapid access to hospitals but their use may be problematic for treating military casualties whom may need prolonged prehospital management.

Detachment of CSs from skin also occurred in a chest seal with vent channel (HyFin). The presence of blood or moisture seemed to weaken the adhesive material and eventually cause detachment of a small portion of this chest seal from the skin. This occurred in 2 out of 6 experiments with this chest seal after 0.75-1 liter air injection despite its strong attachment to dry skin. In fact removal of this chest seal from skin was very difficult and often caused pronounced irritation and redness on the skin. This chest seal is made of a relatively thick plastic sheet that restricts the up/down movement of its venting layer (central piece) during inspiration (covering the wound and preventing air entry into pleural cavity) and expiration (lifting and allowing IP air escape). Some blood clots also formed on the venting layer that further limited its movement. Passage of blood through the laminar vented chest seal was not always complete. Some blood clots were found in the wound track when Russell CSs were used (2 out of 6), however, these did not block air escape through the peripheral vent channels.

This study has some limitations that should be addressed. First, penetrating chest trauma often causes irregular (e.g., stellate and jagged) chest wounds and is associated with lung or airway injuries that cause air leakage and hemorrhage into the pleural cavity. To be reproducible, the chest wound in this experimental model was round in shape with a 10.5 mm diameter. The model also did not include lung injury and external blood and air was injected into the pleural

cavity. Although this model may not mimic clinical HPTX, it offers improvement over previous models and is valid for examining the efficacy of CSs to seal a bleeding chest wound and to prevent accumulation of air and blood in the pleural cavity. Second, the volume of blood injected into the wound track (50 mL) to simulate a bleeding chest wound was chosen arbitrarily and may be more than what may occur in real circumstances. However, the clogging of the valves and detachment of some CSs occurred shortly after start of blood infusion, suggesting the adhesive failure could occur even in the presence of less bleeding. Third, the adhesive function of the CSs was evaluated after applying on shaved and dry skin of pigs with subsequent bleeding. The results may be different if these CSs are placed on a moist (sweat or blood) and hairy chest of wounded patients with or without external bleeding. Fourth, there were differences in definition of tension PTX between our study and other clinical and experimental reports (7, 10, 14)

In summary, the beneficial effects of application of five non-occlusive CSs for management of HPTX and prevention of tension HPTX were evaluated in a swine model with an open bleeding chest wound. We observed covering a dry (non-bleeding) chest wound with any of the CSs restored normal IP pressure and improved respiration. However, subsequent blood exuding from the wound site did not pass through the one-way valve CSs. Accumulated blood clogged the air passage and led either to development of tension HPTX, or more often, caused detachment of CSs from skin and loss of its function. Conversely, the laminated vent channel on other CSs allowed effective evacuation of blood and air from the pleural cavity and prevented tension HPTX. Laminated vent channels were also instrumental in preventing adhesive failure because blood did not accumulate behind the CSs. Although further investigation is warranted for clinical

field application, we recommend in the interim that CSs with laminar vent channel be used preferably for treating combat casualties in the field, particularly prior to the arrival to higher echelon of care.

ACCEPTED

Authorship

All authors contributed in designing the study, developing methodology, interpretation of results and critical review of the manuscript. Surgical procedures, chest seal treatments, data analysis and manuscript preparation were done by Dr. Kheirabadi with technical assistance by Ms. Miranda, Terrazas and SPC Voelker. Dr. Klemcke assisted with statistical analysis of data and editing the manuscript. Drs. Arnaud and Butler contributed in critical review and discussion of the manuscript. The study was supervised and the manuscript was reviewed and edited by Dr. Dubick.

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Disclosure:

The authors have no conflict of interest to report. This manuscript is not an endorsement of any chest seals by the authors or the U.S. Army. It is simply a report of our observations after testing these devices in an experimental model. The funding for this work was provided by the US Army Medical Research and Materiel Command and the Defense Health Program.

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Figure legend:

Fig. 1: **A.** Photograph of the five vented and valved chest seals evaluated in this study.

B. Photograph of a pig in sternal position with hemopneumothorax (HPTX). The open chest wound is sealed with a venting chest seal (round object with large red arrows) and fresh blood that is infused into the chest wound track to simulate active bleeding using a percutaneous catheter. The wound remained sealed while blood and air released from pleural cavity through the vent channel. IP: Intrapleural.

Fig. 2: Inspiration intrapleural (IP) pressure of pigs measured before (baseline) and after HPTX, chest seal application, and IP air injections. Experiments with of all 4 Bolin and 3 of 4 SAM chest seals were terminated early due to either chest seal adhesive failure (detachment from skin) or development of tension HPTX. The dotted line represents a successful test with SAM chest seal. φ $p < 0.01$ vs. baseline; + $p < 0.05$ vs. Russell or Sentinel; * $p < 0.05$ vs. HyFin, Russell or Sentinel. HPTX: Hemopneumothorax; PTX: Pneumothorax; CS: chest seal.

Fig. 3: Respiration minute volumes ($V_t \times RR$) during each step of experiments as measured by the anesthesia machine. Measurements for Bolin and SAM chest seals (except one denoted by dotted line) was discontinued after 1.25-1.5 liter IP air injection due to adhesive failure or development of tension HPTX. φ $p < 0.01$ vs. baseline; * $p < 0.05$ vs. HyFin, Russell or Sentinel. V_t : tidal volume; RR: respiration rate;

Fig. 4: Arterial partial pressure of oxygen (PaO₂) of the pigs measured before (baseline) and 10 min after HPTX induction, chest seal application, and each intrapleural air injection. Measurements for Bolin and SAM chest seals (except one denoted by dotted line) discontinued after 1.25-1.5 liter IP air injection due to adhesive failure or development of tension HPTX. ^φ *p*<0.01 vs. baseline; + *p*<0.05 vs. Russell or Sentinel; **p*<0.05 vs. Russell, Sentinel, or HyFin.

Fig. 5: Mixed venous saturation percent (SvO₂) measured 10 min after each step of experiments. Note significant reduction of SvO₂ after HPTX induction and minimal change after sealing the chest wound with one of the chest seals. Measurements for Bolin and SAM chest seals (except one denoted by dotted line) terminated early due to adhesive failure or development of tension HPTX. ^φ *p*<0.01 vs. baseline; **p*<0.05 vs. Russell, Sentinel, or HyFin.

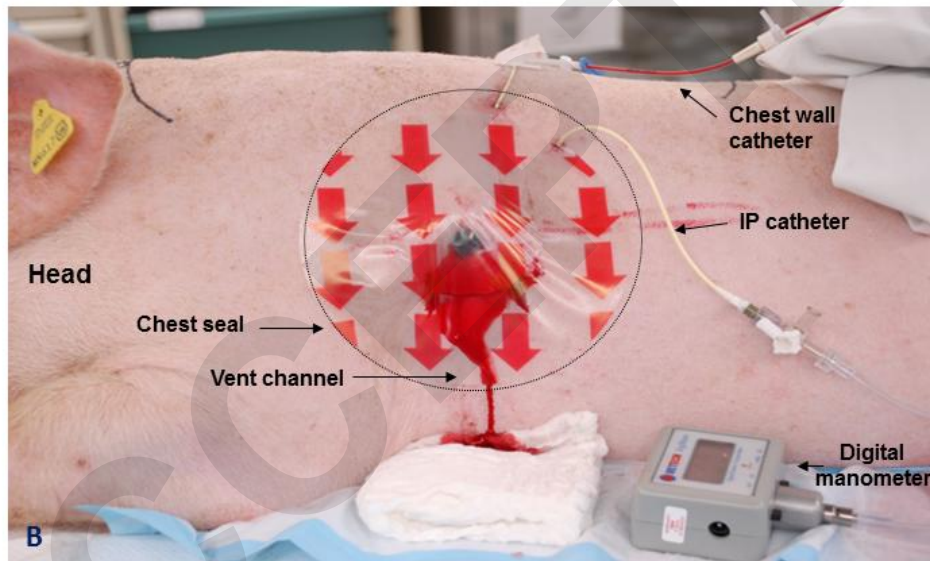
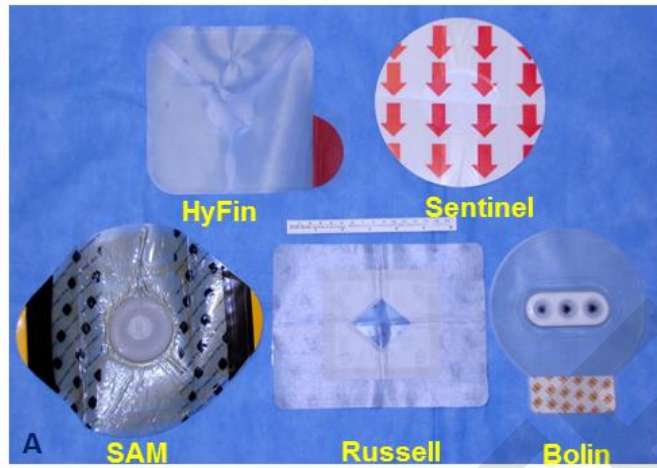


Fig. 1

IP Pressure (inspiration)

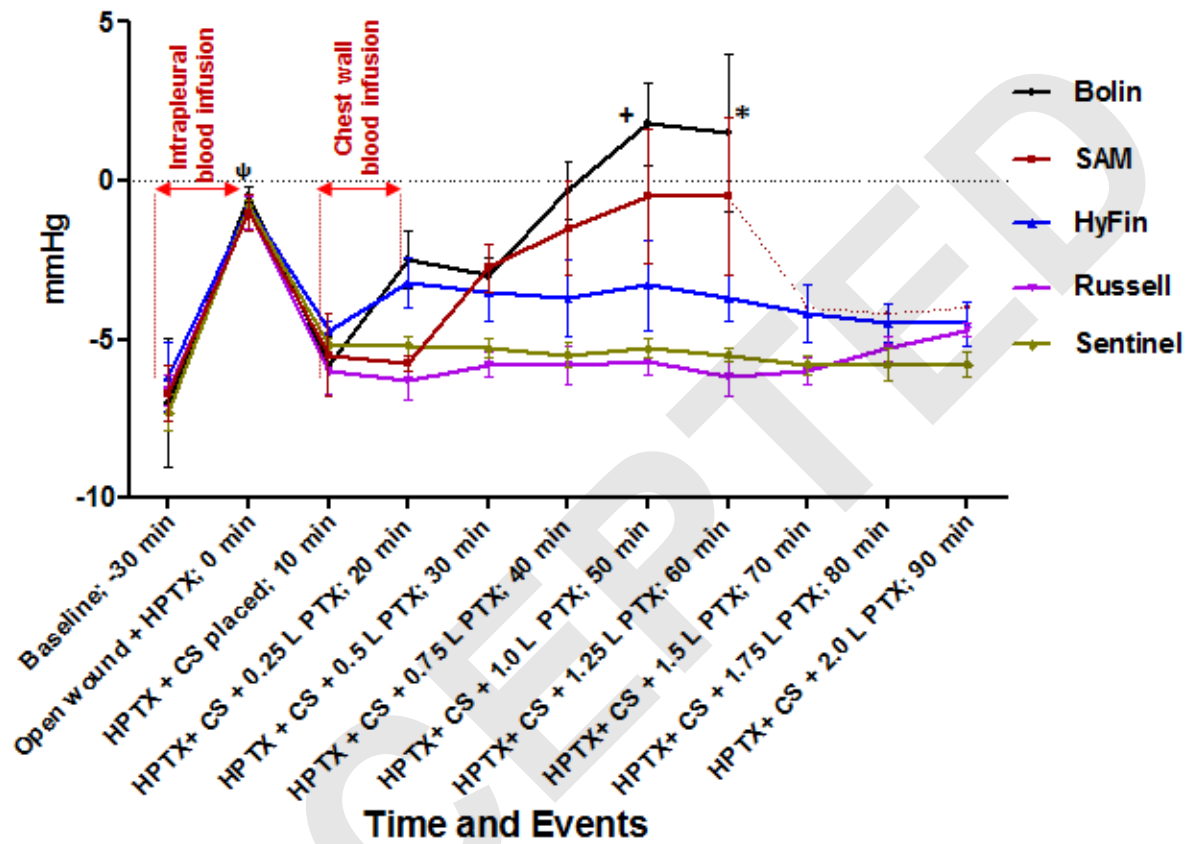


Fig. 2

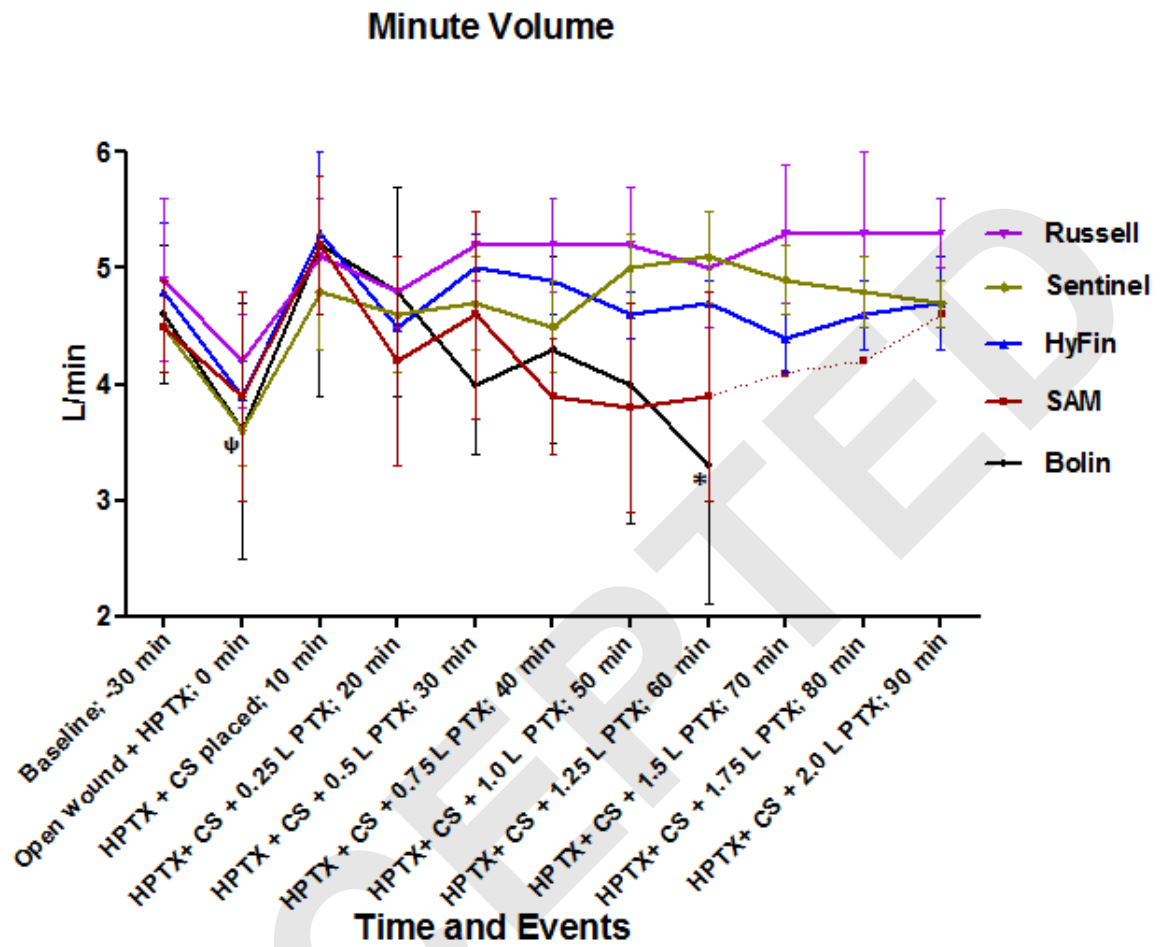


Fig. 3

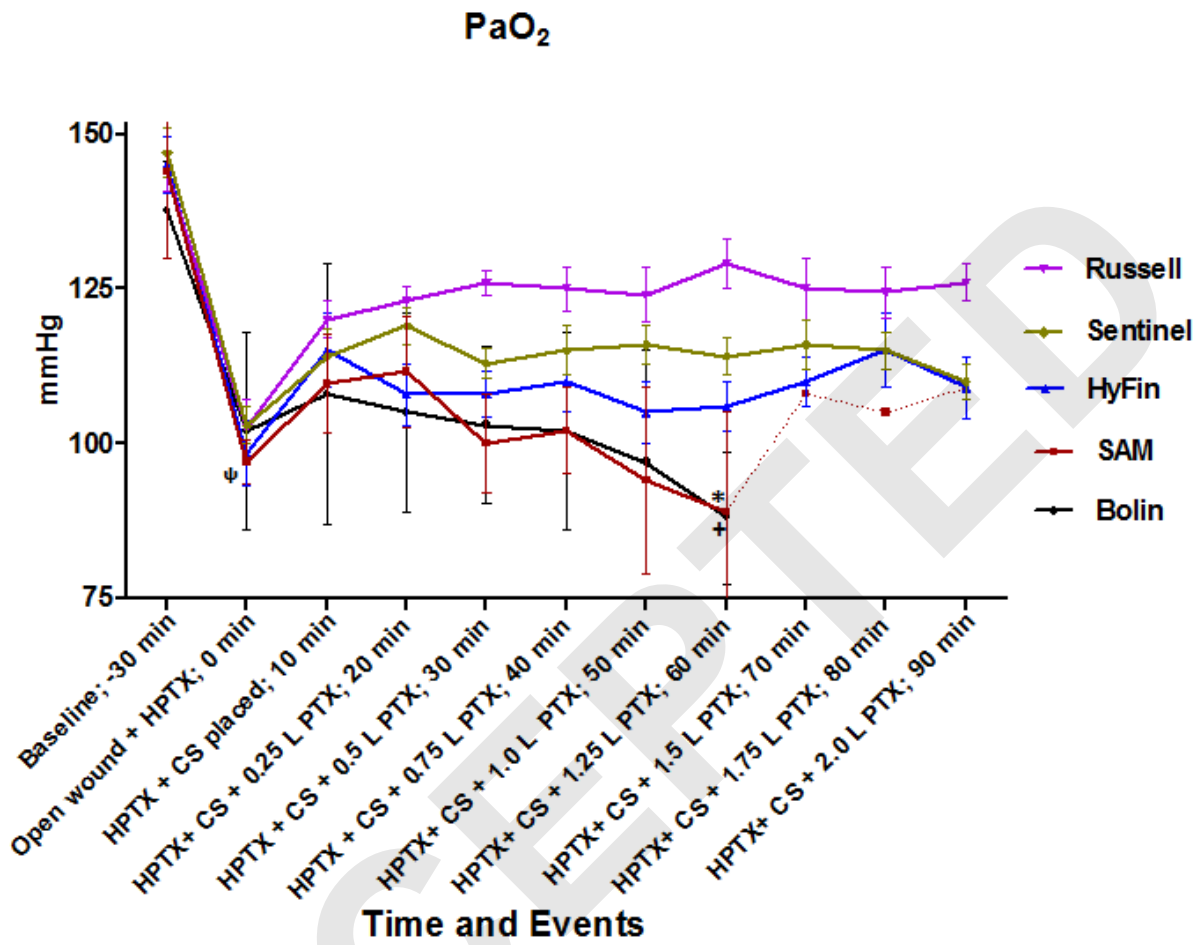


Fig. 4

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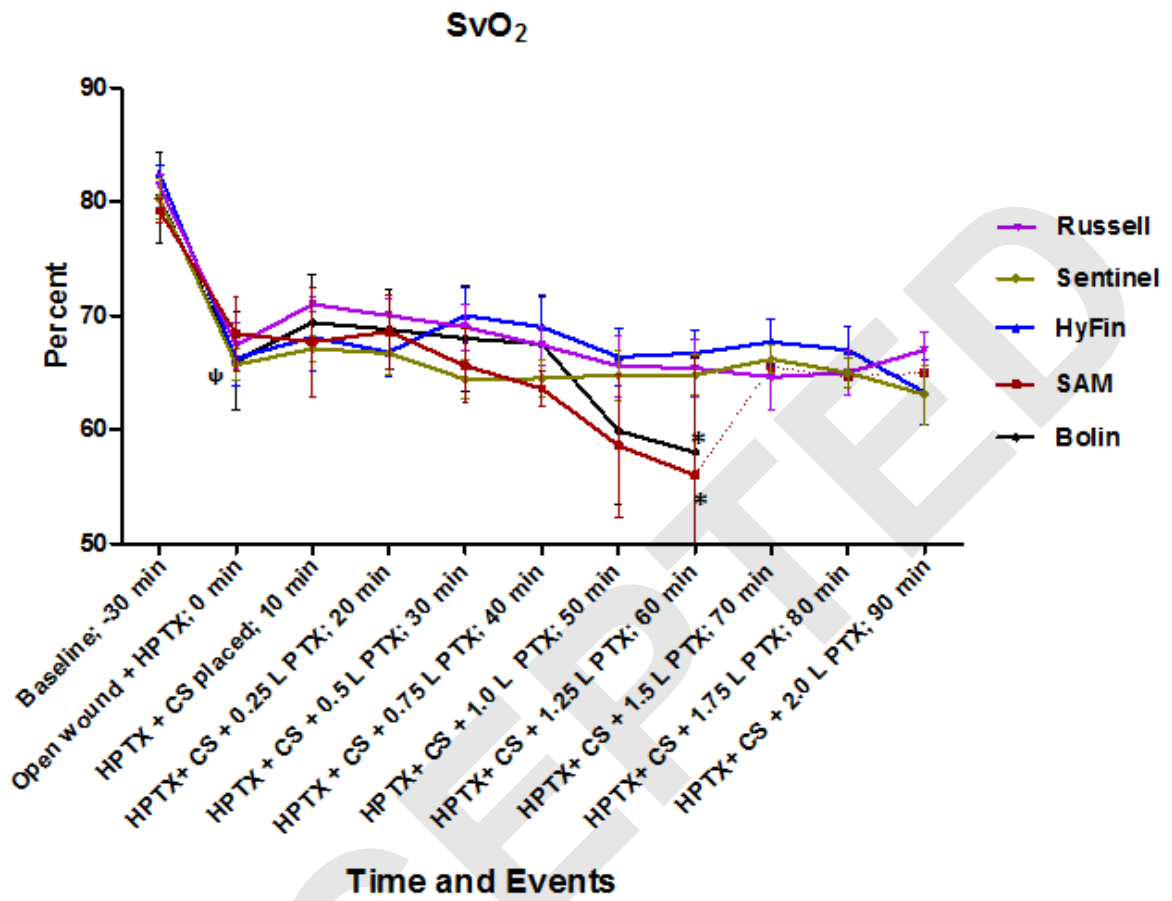


Fig. 5

Table 1. Chest Seal Specifications, Physical Characteristics and Price

Chest Seal Type	Package Weight (gr)	Package size (cm)	Chest seal Shape	Total surface area (cm ²)*	Adhesive surface area (cm ²)*	Valve/Vent type	Retail price of each (\$)
BOLIN	51.1	20.2 x 20.2	Circular	167.3	154.4	3 flutter valves	25.25
HYFIN	39.9	19.5 x 19.6	Square	257.6	214.7	3 laminar vent channels	18.99
RUSSELL	41.4	15 x 13.4	Rectangular	297	284.8	Laminar vent channel	22.47
SAM	65.4	25.4 x 16.1	Circular/oval	284	279	1 flutter valve	27.95
SENTINEL	21.8	19.1 x 20.4	Circular	206	168	2 laminar vent channels	16.70

*Estimated surface areas were calculated based on each chest seal dimensions; peeling tabs surface areas were not included in the total surface.