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EFFECTS OF STOMACH INFLATION ON CARDIOPULMONARY FUNCTION AND SURVIVAL DURING HEMORRHAGIC SHOCK: A RANDOMIZED, CONTROLLED, PORCINE STUDY

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ABSTRACT—**Background:** Ventilation of an unprotected airway may result in stomach inflation. The purpose of this study was to evaluate the effect of clinically realistic stomach inflation on cardiopulmonary function during hemorrhagic shock in a porcine model. **Methods:** Pigs were randomized to a sham control group (n = 9), hemorrhagic shock (35 mL kg^{-1} over 15 min [n = 9]), and hemorrhagic shock combined with stomach inflation (35 mL kg^{-1} over 15 min and 5 L stomach inflation [n = 10]). **Results:** When compared with the control group, hemorrhagic shock (n = 9) increased heart rate (103 ± 11 vs. 146 ± 37 beats min^{-1}; $P = 0.002$) and lactate (1.4 ± 0.5 vs. 4.0 ± 1.9 mmol L^{-1}; $P < 0.001$), and decreased mean arterial blood pressure (81.3 ± 12.8 vs. 35.4 ± 8.1 mmHg; $P < 0.001$) and stroke-volume index (38.1 ± 6.4 vs. 13.6 ± 4.8 mL min^{-1} m^{-2}; $P < 0.001$). Hemorrhagic shock combined with stomach inflation (n = 10) versus hemorrhagic shock only (n = 9) increased intra-abdominal pressure (27.0 ± 9.3 vs. 1.1 ± 1.0 mmHg; $P < 0.001$), and decreased stroke-volume index (9.9 ± 6.0 vs. 20.8 ± 8.5 mL min^{-1} m^{-2}; $P = 0.007$), and dynamic respiratory system compliance (10.8 ± 4.5 vs. 38.1 ± 6.1 mL cmH_2O^{-1}; $P < 0.001$). Before versus after stomach evacuation during hemorrhagic shock, intra-abdominal pressure decreased (27.0 ± 9.3 vs. 9.8 ± 5.4 mmHg; $P = 0.042$). Survival in the sham control and hemorrhagic shock group was 9 of 9, respectively, and 3 of 10 after hemorrhagic shock and stomach inflation ($P < 0.001$). **Conclusions:** During hemorrhagic shock stomach inflation caused an abdominal compartment syndrome and thereby impaired cardiopulmonary function and aerobic metabolism, and increased mortality. Subsequent stomach evacuation partly reversed adverse stomach-inflation triggered effects.

KEYWORDS—Abdominal compartment syndrome, hemorrhagic, near-infrared, resuscitation, shock, spectroscopy, stomach, ventilation

INTRODUCTION

When ventilating a patient with an unprotected airway, the combination of pulmonary compliance, resistance of the respiratory system, and peak airway pressure during bag-valve-mask ventilation determines whether air reaches the lungs or enters the stomach (1). If stomach inflation occurs, a vicious circle of decreasing pulmonary compliance, increasing peak airway pressure and further increasing stomach inflation may result (2). In one case of undetected esophageal intubation in a normovolemic trauma patient, stomach inflation resulted in massive abdominal distension and gut ischemia, but cardiocirculatory function remained stable (3). In a subsequent laboratory study, increasing stomach inflation resulted in decreasing diameter of the inferior vena cava, the abdominal aorta, and impaired cardiopulmonary function and even death (4). However, these pigs were normovolemic, suggesting that stomach inflation in a setting with severe hemorrhagic shock may disproportionally compromise cardiocirculatory function. Because lower esophageal sphincter pressure may collapse earlier than cardiocirculatory function in shock (5), substantial stomach inflation is actually likely when ventilating a severely injured patient with an unprotected airway.

Current guidelines for management of patients with hemorrhagic shock following major trauma focus on circulation and coagulation management (6), but interactions of stomach inflation with cardiopulmonary function are not discussed. This is unfortunate because prevention of or evacuation of stomach inflation may be a simple and inexpensive maneuver that could be performed immediately after endotracheal intubation to

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Authors' contributions: PP had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis, takes the responsibility for the content of the manuscript, including the data and analysis. PB, GP, GS, AW, HS, and HA conducted the experiments, collected the data, and contributed substantially to data analysis and interpretation and writing the manuscript. SN substantially contributed to study design, data analysis and interpretation, and writing the manuscript. HB, VW, and PP organized funding, and substantially contributed to design, data analysis and interpretation, and writing the manuscript.

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ensure that cardiopulmonary function is not inadvertently compromised. This may be even more important when considering that even transient posttraumatic stomach inflation-induced hypotension may have adverse effects on outcome (7, 8).

In this study, we sought to determine the effect of increasing levels of stomach inflation on cardiocirculatory function during severe hemorrhagic shock. Our null hypothesis was that stomach inflation would have no effect on cardiocirculatory function and survival in a porcine preparation of controlled hemorrhagic shock.

MATERIALS AND METHODS

This research project was approved by the Austrian Federal Animal Investigation Committee (33/2011). Animals were managed in agreement with institutional, national, and international guidelines (9–11). On the basis of previous studies we performed this study in an established porcine model (4, 12, 13) on 28 healthy, 12- to 16-week-old domestic pigs of both sexes(17 female, 11 male), weighing 29 to 44 kg.

The animals were fasted overnight, but had free access to water. The pigs were premedicated with Azaperone (4 mg kg^{-1} i.m.; Janssen, Vienna, Austria) and Atropine (0.01 mg kg^{-1} i.m.) 1 h before surgery. Anesthesia was induced with a single bolus dose of Ketamine (25 mg kg^{-1} i.m.), Propofol (1–2 mg kg^{-1} i.v.), and Piritramide (30 mg i.v.; Janssen) given via an ear vein. The animals were placed in supine position, and their trachea and esophagus were intubated with 7.0 mm internal diameter tracheal tubes (Rüsch, Kernen, Germany) during spontaneous ventilation. The tracheal cuff was blocked to 30 cmH_2O, and the esophageal tube was blocked to 100 cmH_2O; cuff pressures were monitored continuously. After tracheal intubation, pigs were ventilated volume controlled (Evita 2; Draeger, Lübeck, Germany) with 21% inspiratory oxygen at 20 ventilations min^{-1}. Tidal volume was adjusted between 6 and 8 mL kg^{-1} body weight to maintain normocapnia (35–45 mmHg), and positive end-expiratory pressure was set to 5 cmH_2O in the preparation phase. Anesthesia was maintained with Propofol (6–8 mg kg^{-1} h^{-1} i.v.) and repetitive injections of Piritramide (30 mg i.v.). If clinical assessment indicated superficial anesthesia, more Piritramide (7.5–15 mg) was injected. Body temperature was maintained between 38.0°C and 39.0°C (normal values in pigs). A standard lead II electrocardiogram was used to monitor cardiac rhythm, and a pulsoxymeter was placed on the tail. Near-infrared spectroscopy (NIRS; Invos System, Somanetics, Troy, Mich) probes were placed on the forehead and the right thigh. Saline solution (0.9%, 6 mL kg^{-1} h^{-1} i.v.) was administered in the preparation phase. A 7.0 Fr saline-filled pulmonary artery catheter (Edwards Life Sciences, Irvine, Calif) was placed in the pulmonary artery via jugular vein to measure right atrial and pulmonary artery pressure, cardiac output, and core temperature. One 6.0 Fr saline-filled arterial catheter (Arrow, Reading, Pa) was placed in the right femoral artery to measure aortic blood pressure, and another 6.0 Fr saline-filled arterial catheter was placed in the left femoral artery to induce hemorrhagic shock. The bladder was cannulated with a 16G (1.8 × 45 mm) peripheral venous catheter. Urine was removed, and 100 mL saline solution was injected into the bladder to measure intra-abdominal pressure. All catheters were placed with ultrasound guidance (Titan, SonoSite, Bothell, Wash). The intravasal and bladder catheters were attached to pressure transducers (1290A; Hewlett Packard, Böblingen, Germany), which were calibrated at the level of the right atrium. Hemodynamic and respiratory variables were measured and analyzed using an AS/3-Monitor (Datex-Ohmeda AS/3; GE Healthcare, Buckinghamshire, UK). NIRS probes (INVOS, Covidien, Medtronic, Dublin, Ireland) were attached to the right forehead and thigh.

Blood gases were analyzed with a blood gas analyzer (ABL 800 Flex; Radiometer Copenhagen, Wiener Neudorf, Austria). After preparation 5,000 IU of unfractioned heparin were administered, and after 15 min stabilization phase the experimental protocol was started. After assessing baseline parameters for circulation, ventilation, and blood gases, positive end-expiratory pressure was reduced to 0 cmH_2O. Saline solution infusion was then stopped. Overall 500 mL were administered.

To simulate a scenario of hemorrhagic shock with inadvertent excessive stomach inflation, the animals were randomly assigned to three groups. The control group received no hemorrhagic shock and no stomach inflation (n = 9); in the hemorrhagic shock group 35 mL kg^{-1} blood were drained via a central venous line over 15 min (n = 9), and in the hemorrhagic shock and stomach inflation 35 mL kg^{-1} blood were drained via a central venous line over 15 min. Thereafter stomach inflation with 5 L ambient air was induced in 5 min (n = 10). After blood drain, inspiratory oxygen fraction was increased to 80% to simulate oxygenation during airway management. Stomach inflation was performed using a calibrated syringe (Rudolph, Kansas City, Mo), connected to the proximal end of the esophageal tube. The esophageal tube was clamped after stomach inflation. Cuff pressures were monitored continuously (Pressure Gauge; VBM, Sulz, Germany). Hemodynamic and respiratory parameters were measured at baseline, after shock induction, after gastric inflation, and subsequently every 5 min. Arterial and mixed venous blood gases were collected at the same time. After 45 min, the esophageal tube was removed, a 12 Ch gastric tube was inserted, and a suction device was connected to aspirate gastric air. To standardize external compression of the stomach, a 5 kg sandbag was then placed on the epigastrium to support gastric suction. After 75 min, measurements were taken every 15 min. The experimental protocol ended after 135 min, or when an animal died (Fig. 1). Animals were then euthanized with an overdose of Piritramide, Propofol, and potassium chloride.

Statistical analysis

Data were recorded every 5 min from the start of stomach inflation until 30 min after stomach deflation, thereafter every 10 min. Data of multiple data acquisition time points were merged for the analysis of the time points "Stomach inflation" and "Stomach deflation" to reduce the number of comparisons. All values are expressed as mean ± SD. Intergroup comparisons were performed using the nonparametric Mann-Whitney *U* test, whereas the Wilcoxon test was used for intragroup comparisons. Survival rates were compared using Kaplan-Meier methods with log-rank (Mantel Cox) comparison of cumulative survival by treatment group. Statistical hypothesis testing was performed two-tailed, and *P* values <0.05 were considered statistically significant. Regarding multiple intergroup comparisons, a correction for multiple testing according to Bonferroni was performed. Therefore, *P* values <0.0042 were considered statistically significant for multiple intergroup comparisons. IBM SPSS Statistics for Windows, Version 21.0, was used to perform statistical analysis (IBM Corp, Armonk, NY).

RESULTS

Results are displayed for baseline, hemorrhagic shock, stomach inflation, and stomach deflation. After 75 min there was no further significant change to any parameter. Therefore, we did not extend the display of data beyond 75 min.

Before hemorrhagic shock and stomach inflation, hemodynamic and respiratory parameters, blood gases, and NIRS values were comparable between groups (Figs. 2–5; Supplemental Figs. 1 and 2, at http://links.lww.com/SHK/A370). Hemoglobin levels in the sham control group ranged around the normal physiologic value of 9, whereas hemoglobin levels in hemorrhagic shock animals decreased to approximately 7 g dL^{-1} (results not depicted) (14). Induction of hemorrhagic

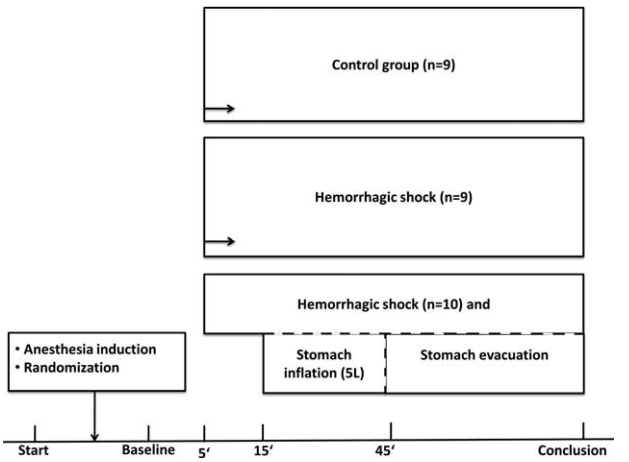

FIG. 1. **Flowchart of the experimental protocol.** Piglets were anesthetized and randomized into on of three groups, i.e. i) control, ii) hemorrhagic shock or iii) hemorrhagic shock and stomach inflation.

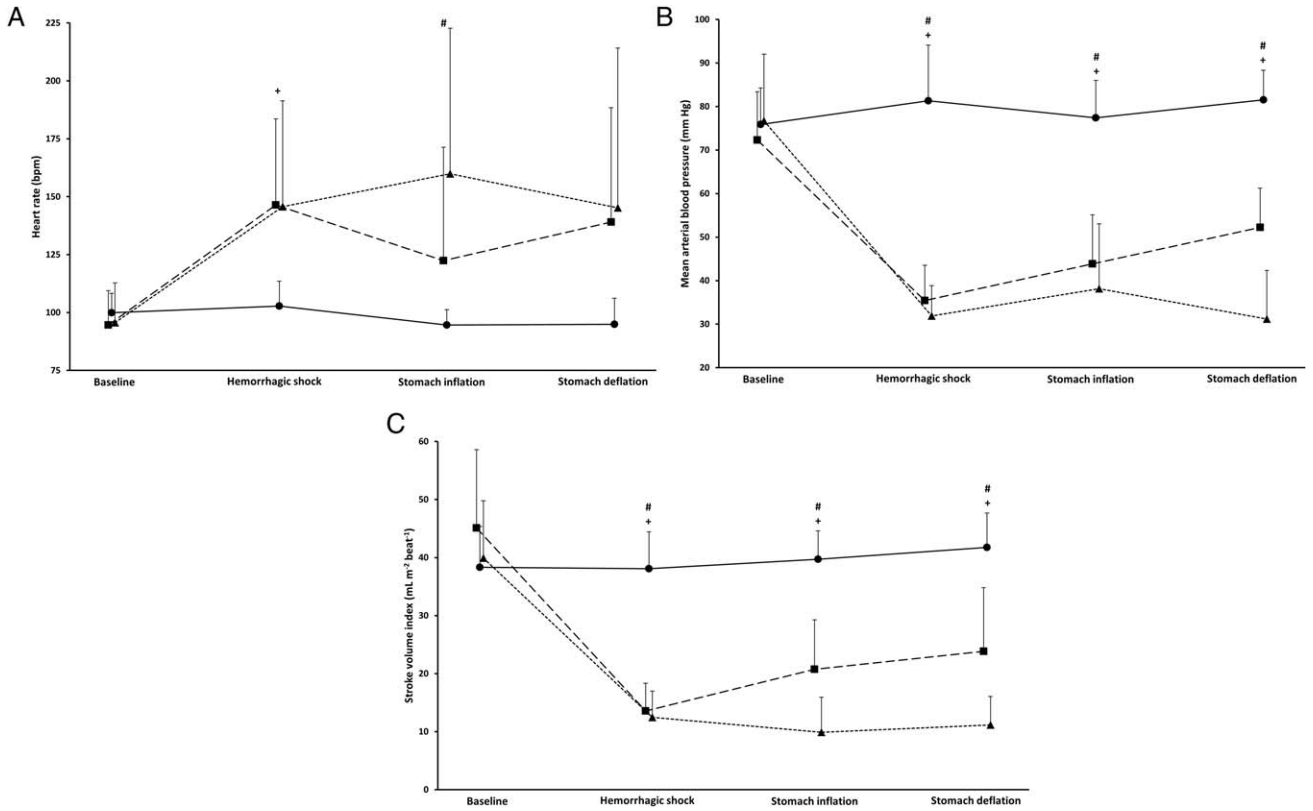

FIG. 2. **(A–C) Heart rate (bpm), mean arterial blood pressure (mmHg), stroke-volume index (mL m^{-2} $beat^{-1}$).** Note that scales do not start at zero. ●, sham control; ■, hemorrhagic shock; ▲, hemorrhagic shock combined with stomach inflation. +, $P < 0.0042$ ● sham control vs. ■ hemorrhagic shock; #, $P < 0.0042$ ● sham control vs. ▲ hemorrhagic shock combined with stomach inflation; §, $P < 0.0042$ ■ hemorrhagic shock vs. ▲ hemorrhagic shock combined with stomach inflation.

shock increased heart rate (103 ± 11 vs. 146 ± 37 beats min^{-1}; $P = 0.002$) and lactate (1.4 ± 0.5 vs. 4.0 ± 1.9 mmol L^{-1}; $P < 0.001$), and decreased mean arterial blood pressure (81.3 ± 12.8 vs. 35.4 ± 8.1 mmHg; $P < 0.001$), central venous pressure (9.0 ± 1.2 vs. 5.2 ± 1.9 mmHg; $P = 0.001$), mean pulmonary artery pressure (21.8 ± 2.3 vs. 14 ± 2.4 mmHg; $P < 0.001$), stroke-volume index (38.1 ± 6.4 vs. 13.6 ± 4.8 mL min^{-1} m^{-2}; $P < 0.001$), oxygen delivery (49.1 ± 14.5 vs. 22 ± 5.7 mL min^{-1}; $P < 0.001$), cerebral (52 ± 10 vs. 28 ± 6; $P = 0.001$), and thigh NIRS (60 ± 4 vs. 45 ± 4; $P = 0.001$). Stomach inflation increased intra-abdominal pressure (1.1 ± 1.0 vs. 27.0 ± 9.3 mmHg; $P < 0.001$), and further advanced the shock state as shown by an additional increase in the heart rate (122 ± 51 vs. 160 ± 58 beats min^{-1}; $P = 0.066$), and further decreases in mean arterial blood pressure (43.9 ± 11.3 vs. 38.2 ± 14.9 mmHg; $P = 0.414$) and stroke-volume index (20.8 ± 8.5 vs. 9.9 ± 6.0 mL min^{-1} m^{-2}; $P = 0.007$). Stomach inflation also decreased pulmonary function as shown by decreases in dynamic respiratory system compliance (38.1 ± 6.1 vs. 10.8 ± 4.5 mL cmH_2O^{-1}; $P < 0.001$). In addition, oxygen delivery (26.4 ± 6.8 vs. 13.7 ± 8.4 mL min^{-1}; $P = 0.009$), pH (7.44 ± 0.04 vs. 7.41 ± 0.07; $P = 0.157$), and base excess diminished (0.4 ± 2.6 vs. -2.4 ± 3.7; $P = 0.052$), whereas lactate increased (5.0 ± 2.5 vs. 6.8 ± 3.0 mmol L^{-1}; $P = 0.122$). Cerebral and thigh NIRS values decreased in hemorrhagic shock. After stomach inflation, cerebral NIRS tended to increase (36 ± 5 vs. 39 ± 16; $P = 0.629$), whereas thigh NIRS values further decreased (45 ± 9 vs. 24 ± 9; $P = 0.010$).

Stomach evacuation decreased intra-abdominal pressure (27.0 ± 9.3 vs. 9.8 ± 5.4 mmHg; $P = 0.042$) and increased dynamic respiratory system compliance (10.8 ± 4.5 vs. 23 ± 4.4 mL cmH_2O^{-1}; $P = 0.042$), whereas heart rate (160 ± 58 vs. 145 ± 52 beats min^{-1}; $P = 0.345$), mean arterial pressure (38.1 ± 14.9 vs. 31.2 ± 11.2 mmHg; $P = 0.078$), stroke-volume index (9.8 ± 6.0 vs. 11.2 ± 4.9 mL min^{-1} m^{-2}; $P = 0.893$), oxygen delivery (13.7 ± 8.4 vs. 19.8 ± 11.6 mL min^{-1}, $P = 0.893$), and cerebral (39 ± 16 vs. 44 ± 13; $P = 0.655$) and thigh NIRS (24 ± 9 vs. 44 ± 7; $P = 0.180$) did not change significantly. Survival in the control and hemorrhagic shock group was 9 of 9, respectively, whereas 7 of 10 animals in the hemorrhagic and stomach inflation group died (Fig. 5; $P < 0.001$).

DISCUSSION

During hemorrhagic shock stomach inflation caused an abdominal compartment syndrome and thereby impaired cardiopulmonary function and metabolism, and increased mortality. Subsequent stomach evacuation partly reversed the adverse stomach-inflation triggered effects. Our findings indicate that the combination of hemorrhagic shock and stomach inflation could be a lethal combination in severely traumatized patients, and stomach evacuation is not able to reduce intra-abdominal pressure to base line.

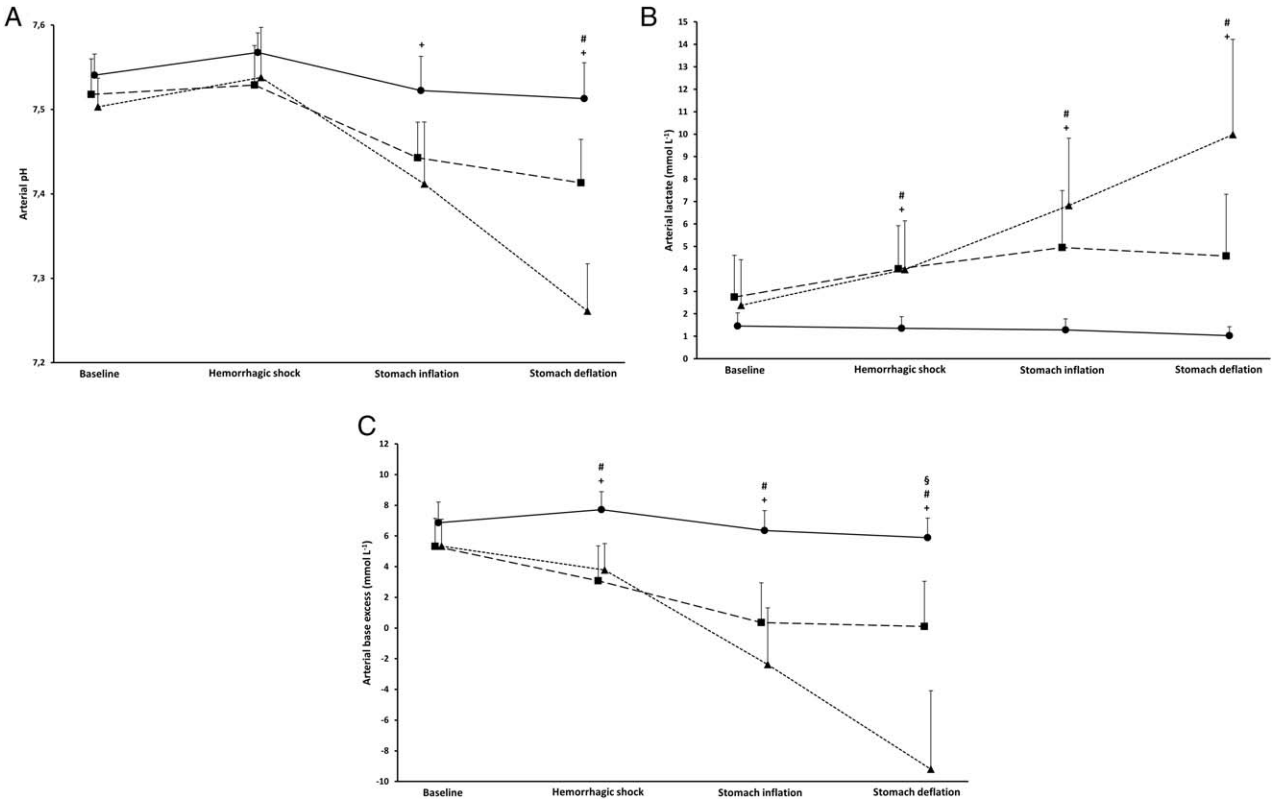

FIG. 3. **(A–C) Arterial pH, arterial lactate (mmol L^{-1}), and arterial base excess (mmol L^{-1}).** Note that scales do not start at zero. ●, sham control; ■, hemorrhagic shock; ▲, hemorrhagic shock combined with stomach inflation. +, $P < 0.0042$ ● vs. ■; #, $P < 0.0042$ ● vs. ▲; §, $P < 0.0042$ ■ vs. ▲.

In a hemorrhagic shock patient inadvertent stomach inflation may occur by different mechanisms: first, in the case of undetected, inadvertent esophageal intubation and subsequent positive pressure ventilation. Second, facemask ventilation of a severely traumatized patient may result in a significant incidence of stomach inflation, even when performed with a peak airway pressure as low as 15 cmH_2O (15). In both situations, large amounts of gas may pass the pylorus and mostly remain in the gut (3), but substantial stomach distention resulted in stomach rupture as well (16–18). Although the problem of substantial stomach inflation following inadvertent esophageal intubation may be obvious, stomach inflation following bag-valve-mask ventilation may be unanticipated, but treacherous (3, 4, 19). When ventilating an unprotected airway, approximately 50% of applied minute ventilation may enter the stomach (approximately 2.5 L min^{-1} in a 70 kg adult) (1). In addition, the upper and lower esophageal sphincter barrier may be impaired by several factors such as age, anesthesia induction, gastric content, obesity, preexisting gastric reflux, and shock (1, 20–22). When extrapolating these observations to our 40 kg pigs, several liters of stomach inflation may be reached easily during bag-valve-mask ventilation before endotracheal intubation. Stomach and intestinal air evacuation was performed with a gastric tube and external compression of the bloated belly with a standardized weight of 5 kg, similarly to attempts performed in clinical practice with a gastric tube and gentle external compression of the distended abdomen with a hand. Accordingly, we suggest that the 5 L stomach inflation used in our experimental model-simulating inadvertent stomach inflation during hemorrhagic shock may serve as a realistic tool to assess effects of stomach inflation on hemodynamic and pulmonary function in shock.

In our model, experimental stomach inflation decreased pulmonary compliance dramatically to approximately one-third of baseline values while reaching intra-abdominal pressures of approximately 30 mmHg. This produced corresponding cardiocirculatory function deterioration, similar to a previous canine model (23), but especially in the hemorrhagic shock animals, indicating that the model is a valid tool to assess effects of stomach inflation during shock. Interestingly, pigs suffering from hemorrhagic shock only recovered at least in part by reaching a mean arterial blood pressure of approximately 52 mmHg, whereas pigs with additional stomach inflation remained in shock with less than 50% of preshock mean arterial blood pressure respectively, less than 30% of preshock stroke-volume index, and even developed further increasing dramatic lactate levels of approximately 10 mmol L^{-1}. Not surprisingly, this resulted in statistically significant increased mortality in hemorrhagic shock pigs with stomach inflation. If these observations can be extrapolated into clinical practice, even professional rescuers may further deteriorate circulatory shock by hyperventilating the lungs, and thereby inadvertently inflating the stomach during their bag-valve-mask ventilation maneuvers instead of safely ventilating and oxygenating the patient (24). Circulatory decompensation and death were caused by the combined effects of

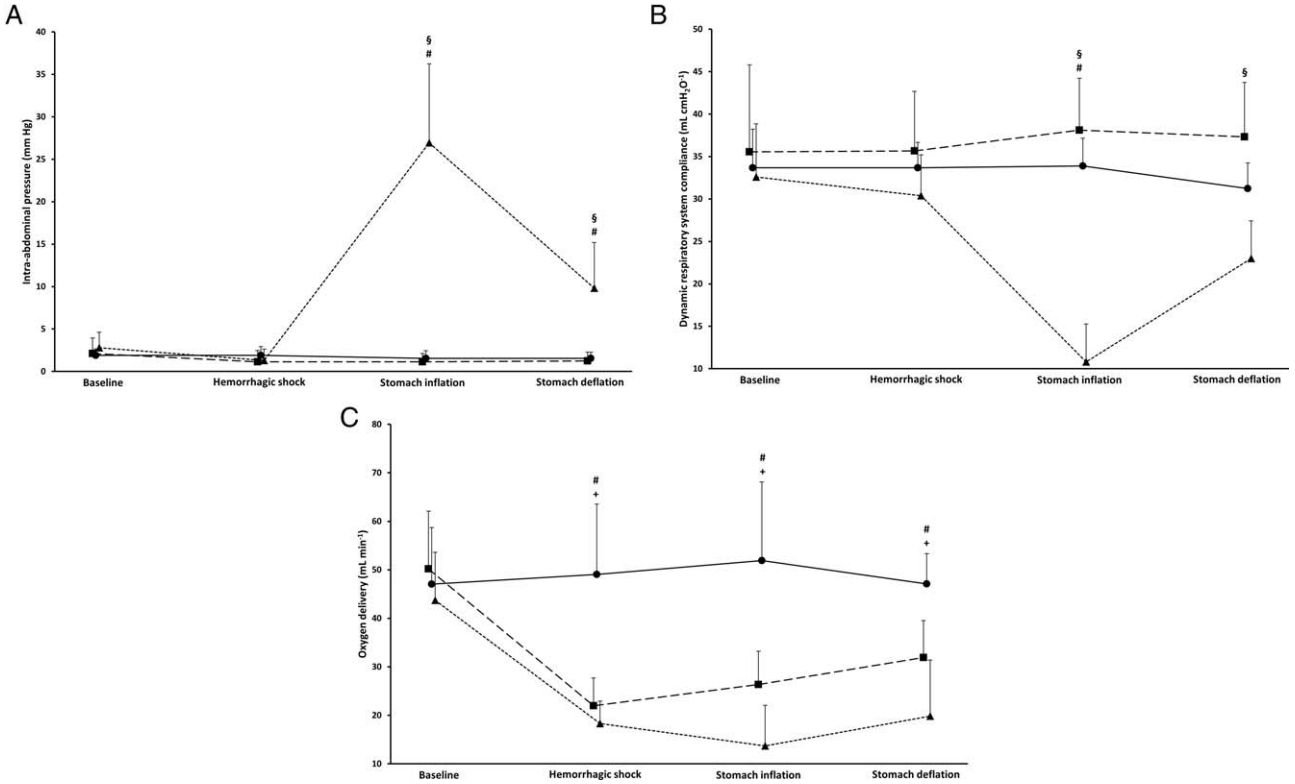

FIG. 4. **(A–C) Intra-abdominal pressure (mmHg), dynamic respiratory system compliance (mL cmH_2O^{-1}), and oxygen delivery (mL min^{-1}).** Note that scales do not start at zero. ●, sham control; ■, hemorrhagic shock; ▲, hemorrhagic shock combined with stomach inflation. +, $P < 0.0042$ ● vs. ■; #, $P < 0.0042$ ● vs. ▲; §, $P < 0.0042$ ■ vs. ▲.

hypovolemic shock and distributive shock. The latter was induced by the stomach inflation-triggered abdominal compartment syndrome.

An interesting finding of our study is the measurement of excessively high intra-abdominal pressures, reaching almost 30 mmHg during stomach inflation. The detrimental effects of elevated intra-abdominal pressures (>12–15 mmHg) and the abdominal compartment syndrome (i.e., sustained intra-abdominal pressure >20 mmHg and associated new organ dysfunction) on cardiopulmonary and abdominal organ function have been recognized only in the last years (25, 26). Intra-abdominal hypertension and abdominal compartment syndrome may increase morbidity and mortality in critically ill patients (25, 26). Although an abdominal compartment syndrome in critically ill patients usually develops over hours or days due to hemorrhage or fluid over-resuscitation, this phenomenon occurred in our animals rapidly following stomach inflation. In another report, stomach inflation resembled functional clamping of the inferior vena cava and the aorta, as demonstrated by computed tomography scans (3). In the present study, this is impressively underlined by decreases in stroke-volume index and a more than 50% decrease of thigh NIRS values due to reduced perfusion. These findings may be explained by several mechanisms. First, the stomach inflation triggered abdominal compartment syndrome, impaired perfusion in the caudal part of the body as demonstrated by low thigh NIRS measurements, thus reducing right ventricular preload and subsequently stroke-volume index. Second, the increase in airway pressure contributed to a compression of pulmonary capillaries as demonstrated by increases in pulmonary artery pressure. Third, hypercarbia and hypoxia induced pulmonary vasoconstriction, thus further increasing pulmonary artery pressure-mediated right heart failure. Furthermore, cardiac failure impaired oxygen delivery, as demonstrated by the development of a mixed respiratory and metabolic acidosis in the hemorrhagic shock plus stomach inflation group; 7 out of 10 hemorrhagic shock pigs with stomach inflation died within 60 min after the onset of the abdominal compartment syndrome.

Extrapolating our data from a 40 kg pig to a 70 kg hemorrhagic shock patient, possibly less than 10 L, cumulative stomach inflation may be a critical limit. As mentioned earlier, up to 50% of minute ventilation during bag-valve-mask ventilation may enter the stomach; accordingly, in a hemorrhagic shock patient, life-threatening stomach inflation may result within 4 min of mouth-to-mouth, bag-valve-mask ventilation, or in some cases, when using supra-glottic airway devices (27). As a consequence, during management of hemorrhagic shock, early intubation instead of prolonged bag-valve-mask ventilation may be more important than currently anticipated, even in corresponding guidelines (28, 29).

An increase in pulmonary airway pressures proposes that even a relatively moderate amount of approximately 5 L stomach inflation may trigger rapid pulmonary failure. Decreased respiratory system compliance on one hand, and increased airway pressures on the other hand indicate severe

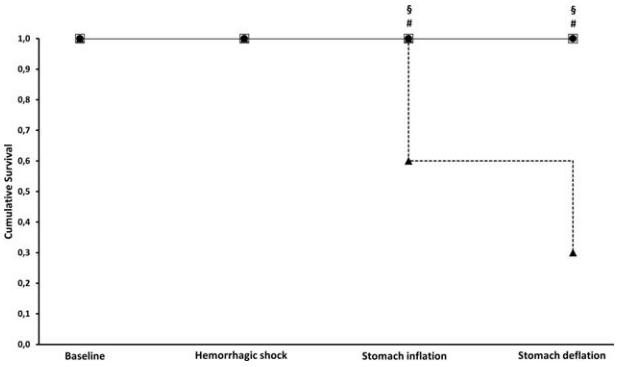

FIG. 5. **Kaplan-Meier survival analysis.** ●, sham control; ■, hemorrhagic shock; ▲, hemorrhagic shock combined with stomach inflation. +, $P < 0.05$ ● vs. ■; #, $P < 0.05$ ● vs. ▲; §, $P < 0.05$ ■ vs. ▲ (18).

compression of pulmonary tissue. In our model, volume-controlled ventilation ensured constant tidal volumes, but increased peak airway pressures up to approximately 40 cmH_2O. However, high peak airway pressure may lead to even more stomach inflation, severe barotrauma of the lung, and may trigger respiratory distress syndrome, with subsequent multiorgan failure (30, 31). Otherwise, with pressure-limited ventilation or bag-valve-mask ventilation, insufficient ventilation may result. Because oxygen content remained comparable between groups while oxygen delivery dropped acutely by approximately 65% following stomach inflation, this indicates primarily a hemodynamic problem, although the pulmonary function remained almost unchanged.

Previous studies of stomach inflation in pigs with spontaneous circulation and during cardiopulmonary resuscitation showed a deterioration in pulmonary function and circulation (3, 4), and also in survival in one model (3). In our model, we evacuated the stomach in all animals 30 min after inducing stomach inflation, which reduced intra-abdominal pressure by more than 50% and increased respiratory system compliance by more than 100% within minutes. However, these dramatic improvements did not correspond with cardiocirculatory variables. A subtle signal may indicate which body region may benefit the most of stomach evacuation, namely subdiaphragmatic parts. In fact, NIRS values of the thigh region almost doubled after stomach evacuation; corresponding increases in arterial lactate suggest a washout effect of subdiaphragmatic regions, an effect that may affect outcome (32). If our observations can be extrapolated to shock patients who underwent bag-valve-mask ventilation before endotracheal intubation, it may be prudent to evacuate the stomach early after invasive airway management to improve proper distribution of organ blood flow. This maneuver is currently not recommended by any clinical advanced life support guideline, but it is inexpensive, minimally invasive, and may decrease morbidity. Likewise our findings indicate that it may be reasonable to avoid ventilating an unprotected airway in a hemorrhagic shock patient early as this may kick off a vicious circle of increasing stomach inflation and decreasing cardiopulmonary function. On the basis of prior experimental studies on stomach inflation (3, 4), we had expected a cardiocirculatory collapse as shown by increasing heart rate and lactate and decreasing stroke-volume index and pH. We also had expected that stomach evacuation would considerably improve cardiocirculatory function and aerobic metabolic function. However, our findings of increased mortality in the stomach inflation group, and insufficient cardiocirculatory improvement after stomach evacuation seem to indicate that the combination of hemorrhagic shock and stomach inflation could be a lethal combination in severely traumatized patients, and stomach evacuation by suction is not able to reduce intra-abdominal pressure to baseline as some air may be trapped in the intestine.

Some limitations should be noted. First, the use of potent anesthetics may have influenced hemodynamic and pulmonary parameters. Second, we used a model with stable hemorrhagic shock, which may not mimic traumatic bleeding in humans. However, there are clinical scenarios were bleeding can be stopped, e.g., if it is amenable to compression. We considered this model more controllable than one with ongoing bleeding. Third, we are unable to determine whether these results of a porcine study can be directly extrapolated to stomach inflation in humans; however, such a clinical study may be extremely difficult to conduct due to ethical considerations. We also did not standardize stomach inflation to the body weight; this may have precipitated adverse events in smaller animals. In addition, we clamped the esophageal tube to allow for standardization of the stomach inflation. As the lower esophageal sphincter is not a one-way valve, probably only parts of the air may be trapped in the stomach or in the gut, and higher peak airway pressures may be required than used in this model. Lastly, no fluid resuscitation was performed in this scenario. We considered an out-of-hospital scenario with basic life support-skilled emergency medical technicians who were performing mask ventilation without fluid resuscitation. A scenario, which does happen commonly in Central Europe before arrival of an advanced life support team.

In conclusion, in this porcine hemorrhagic shock model, stomach inflation impaired cardiopulmonary function, metabolism, and resulted in increased mortality. Stomach evacuation partly reversed adverse stomach-inflation triggered effects.

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