ORIGINAL ARTICLE

Emergent cardiopulmonary bypass in canines with penetrating cardiac wounds caused by gunshot

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Background: Most patients with penetrating cardiac wounds die within minutes of injury from uncontrolled haemorrhage and acute cardiac dysfunction. Thus, sustaining sufficient circulation rapidly is crucial to saving lives. Emergent cardiopulmonary bypass (CPB) is a potential intervention to maintain circulation after penetrating cardiac wounds from a gunshot.

Methods: Canines were wounded with a bullet and randomly split into one of three treatment groups. Animals in group 1 (Gp1) were treated with conventional methods. Animals in group 2 (Gp2) received emergent CPB for 180 min and autologous blood transfusion. Animals in group 3 (Gp3) received emergent CPB for 30 min followed by surgical repair. Animal survival, haemodynamics and blood chemistry were measured, and lung water content was evaluated at the end of the experiment.

Results: The right ventricle was the most severely wounded cardiac chamber. In Gp1, mean arterial pressure and central venous pressure were dramatically decreased 8 min after injury, and all animals died within 18 min. In Gp2 and Gp3, mean arterial pressure ranged from 60–90 mm Hg during CPB. 60 min after terminating CPB in Gp2, mean arterial pressure and heart rate were decreased compared to Gp3. In Gp3, most animals maintained haemodynamic stability. 60 min after CPB, free haemoglobin in circulating blood was elevated compared to pre-trauma levels. Pulmonary water content was significantly higher in Gp2 and Gp3 than in Gp1.

Conclusions: Emergent CPB in the field can maintain haemodynamic stability and supply vital organs with sufficient blood flow, but surgery following CPB is essential to rescue patients with penetrating cardiac wounds.

Penetrating cardiac wounds (PCW) cause immediate fatal injuries in 80–94% of cases. Victims of PCW must reach professional medical care with sufficient vital functions to survive.[1 2] Though great progress has been made in emergent medicine and surgical techniques, only minor improvements in the survival rates following PCW have been made in decades.[3 4] PCW represents an ongoing clinical challenge to trauma surgeons.

Cardiopulmonary bypass (CPB), a milestone in cardiac surgery, temporarily replaces the function of the heart and lungs and supplies vital organs with oxygenated blood flow. Emergent CPB used with surgery after PCW has been successful in saving lives, provided the CPB was initiated in a timely manner.[5] Previous research has studied the role of CPB during surgical repair, but has not investigated whether timely application of CPB in the field is useful in maintaining haemodynamic stability and functioning vital organs. Here, we describe the effects of using emergent CPB on canines immediately after a PCW caused by a gunshot wound.

MATERIALS AND METHODS

All research was conducted in compliance with the Animal Welfare Act and the Guide for the Care and Use of Laboratory Animals. All surgeries were performed in designated veterinary surgical suites using sterile instruments and procedures.

A total of 36 healthy hybrid canines, weighing 15 (4) kg, were randomly assigned to one of three groups (n = 12/group) irrespective of gender. Animals were wounded as described below. Following injury, group 1 (Gp1), the control group, received conventional treatments. Group 2 (Gp2) received conventional treatments and emergent CPB. Group 3 (Gp3) received conventional treatments, emergent CPB and surgical repair.

The animals were fasted overnight and sedated with an intramuscular injection of ketamine (10 mg/kg). General anaesthesia was administered with intravenous pentobarbital sodium (30 mg/kg). Each animal underwent endotracheal intubation, was prepared with povidone iodine and draped with sterile sheets. A 20 gauge angiocatheter was inserted into the left subclavian artery for continuous blood pressure monitoring and sample blood collection. Animals were placed in an erect position with the head facing down and turned to the left 20°. A small diameter rifle and bullets weighing 2.57 g with an initial velocity of 350 m/s were used to create the PCW. From a distance of 25 m, each animal was shot, by a professional, at the intersection of the sternal midline and the fourth intercostal space. A velocity detector was placed to measure the entrance and exit velocity of the bullet. The conventional treatments of laceration closure, infusion of lactated Ringers solution (100–250 ml/h depending on bleeding rate), thoracic cavity drainage and mechanical ventilation (volume control plus positive end expiratory pressure 8.16 cm H_2O, tidal volume 15 ml/kg, breath rate 11–16/min) were immediately performed after the injury. Gp1 received no further care and repair of myocardial injury was not performed.

Gp2 received a single intravenous bolus of heparin (2.5 mg/kg). Four doctors were divided into two groups, respectively, for emergency insertion of femoral artery perfusion cannulae and jugular vein drainage cannulae. A 22 gauge French arterial perfusion cannula (Jostra, Hirrlingen, Germany) (with the tip

Abbreviations: CPB, cardiopulmonary bypass; PAWP, pulmonary artery wedge pressure; PCW, penetrating cardiac wounds; SOL, signs of life

Table 1 Treatments after injury on each group

Group	Treatments
Gp1	Conventional treatments
Gp2	Conventional treatments+CPB
Gp3	Conventional treatments+CPB+surgical repair

CPB, cardiopulmonary bypass.
Conventional treatments: laceration closure + infusion of lactated Ringers solution + thoracic cavity drainage + mechanical ventilation.
Surgical repair: cardiac wound repair + pulmonary lobectomy.

cut off) was inserted 5–7 cm through the left femoral artery towards the heart. A 20 gauge French venous drainage cannula was inserted 8–10 cm towards the heart through the right jugular vein. The cannulae were ligated with No. 10 sutures. After gas was driven out, these cannulae were connected to an emergent CPB system, autologous blood infusion system (developed and manufactured by our institute), and membrane oxygenator, and the surgeons then began the extracorporeal circulation perfusion at room temperature. If necessary, lactated Ringers solution (250–500 ml) was added to maintain the volume of the CPB reservoir. Volume of the balanced crystalloid/colloid priming solution was 100 ml/kg. Flow rate was 800–1500 ml/min. Haemorrhaged blood was collected through the chest tube into the CPB reservoir and mixed with heparin (3 mg/ml). The procedures were performed by two teams (two surgeons in each team). CPB was generally established in 5–7 min post-trauma and terminated after 3 h.

Gp3 received treatment as described above for Gp2 until 30 min of CPB had been performed. At this point, Gp3 animals underwent surgery to repair cardiac wounds and to receive a pulmonary lobectomy. Protamine in a 2:3 ratio was used to neutralise the heparin. When these operations were completed the animal was removed from CPB and monitored for 3 h. The different treatments performed on different groups are shown in table 1.

Mean arterial blood pressure and heart rate were measured at the left subclavian artery at pre-trauma, 5, 30, 60, 90, 120, 150, and 180 min post-trauma. Arterial blood was sampled to analyse blood gases with a Roche OMNI C analyzer (Roche Diagnostics GmbH, Mannheim, Germany) at pre-trauma and after 60 and 180 min of CPB. Venous blood was collected from the right jugular vein to measure alanine aminotransferase, blood urea nitrogen, carninine and creatine kinase with enzyme kinetics using Olympus AU2700 autoanalyzer (Olympus, Tokyo, Japan) (corrected by haematocrit). Before the injury, a Swan-Ganz catheter was inserted through the right jugular vein of animals in Gp3 to measure pulmonary artery wedge pressure (PAWP) and cardiac output at pre-trauma, 120 min of CPB, 5 min after terminating CPB and at the end of experiment. The samples collected from dead animals were assigned to the adjacent time point.

In Gp1, tissue was collected when animals died. Gp2 animals were exsanguinated after 3 h of CPB. Gp3 animals were exsanguinated 3 h after surgery was completed. After animals were sacrificed, the heart and lungs were collected from six animals in each group. Cardiac or pulmonary tissue was treated by conventional methods for haematoxylin and eosin staining, and then examined under a light microscope. The remaining left lung was weighed and heated at 75℃ for 72 h to a constant weight to calculate the water content of the lung. The measurements performed on each group are shown in table 2.

All values are expressed as mean (SD). Completely random design analysis of variance (ANOVA) and methods of the least significant difference were performed to make two-two comparisons. Values of $p < 0.05$ were considered significant.

RESULTS

The difference in energy transmitted from pellet to animal was not different in the three groups (table 3). Wound tracts were observed in the skin, thorax and lung, and rib fractures were common. Pulmonary wound tracts were located in the hilum of the middle or inferior lobe of the right lung. Injury to the associated pulmonary vessels was common. The tracts ranged from 3–4 cm in length and 1–2 cm in diameter. In some animals, pulmonary contusion, haematoma and oedema were seen in both lungs. At 2 h post-trauma, the difference in chest drainage of the three groups was not significant (table 4, total blood volume of a canine 74 ml/kg and circulating blood volume 37 ml/kg).

As shown in table 4, the right ventricle was the wounded chamber of the heart most involved in all groups. The rates of wounded right ventricle were 75% in Gp1, 75% in Gp2, and 83.3% in Gp3, and there was no statistical significance among the groups ($p>0.05$). In Gp1, the right atrium of two animals

Table 2 Measurements performed on each group

	Gp1	Gp2	Gp3
Pre-trauma	MAP/HR	MAP/HR/ABG/Hb/BC	MAP/HR/ABG/Hb/BC/PAWP/CO
5 min	MAP/HR	MAP/HR	MAP/HR
30 min		MAP/HR	MAP/HR
60 min		MAP/HR/ABG	MAP/HR/ABG
90 min		MAP/HR	MAP/HR
120 min		MAP/HR/Hb	MAP/HR/PAWP/CO/Hb
150 min		MAP/HR	MAP/HR
180 min		MAP/HR/Hb/ABG/BC	MAP/HR/Hb/ABG/PAWP/CO/BC

ABG, arterial blood gas; BC, blood chemistry; CO, cardiac output; Hb, haemoglobin; HR, heart rate; MAP, mean arterial blood pressure; PAWP, pulmonary artery wedge pressure.

Table 3 Energy transmitted from pellet to animal

Group (n = 12)	Diameter (mm)	Weight (g)	Velocity (m/s)	Energy (J)	Absorbed energy (J)	Absorbed efficiency
1	5.56	2.57	355 (18.7)	162 (7.3)	100.3 (17.4)	61.9%
2	5.56	2.57	349 (22.7)	157 (9.4)	103.6 (18.3)	66.2%
3	5.56	2.57	347 (15.5)	155 (6.6)	99.1 (13.3)	64.1%

Table 4 Cardiac wound location and chest drainage volume

Group	Wound location RA	RV	LV	Chest drainage 200–300 ml	300–400 ml	400–500 ml
1	2	9	1	3	6	3
2	0	9	3	7	5	
3	0	10	2	3	9	

LV, left ventricle; RA, right atrium; RV, right ventricle.

was wounded, and the left ventricle of one animal was wounded. The animals whose right atria were wounded died 14 min and 18 min after being wounded, whereas the animals whose left ventricle was wounded died 8 min later. In Gp2, the left ventricle of three animals was wounded, and those animals died 16 min, 23 min and 37 min later. In Gp3, the left ventricle of two animals was wounded, and those animals died 18 min and 34 min later. In Gp1, regardless of whether the wounded organ was cardiac atrium or ventricle, the survival time could not be prolonged by conventional treatment, as the survival rate was zero 3 h later. Although the animals in Gp2 and Gp3 whose left ventricle was wounded died after cardiopulmonary bypass was established, the survival time was obviously prolonged. The survival rates were 75% (9/12) in Gp2 and 83.3% (10/12) in Gp3 at 3 h after injury; both were significantly higher than that of Gp1 ($p<0.01$), but there was no significant difference between Gp2 and 3 ($p>0.05$).

Haemodynamics

In Gp1, except for two animals wounded in the right atrium, mean (SD) arterial pressure (152.3 (94.5) mm Hg vs 20.25 (6.75) mm Hg), heart rate (193 (136)/min vs 22 (27)/min) and central venous pressure (4.5 (1.5) mm Hg vs 3.0 (1.5) mm Hg) were dramatically decreased at 8 min post-trauma (figs 1 and 2). The animals in Gp1 died between 9.6–18 min post-trauma. Due to the rapid use of emergent CPB, animals in Gp2 and Gp3 maintained mean arterial pressure in the range of 60–90 mm Hg for the duration of CPB. However, following the separation of CPB, animals in Gp2 presented with progressively decreasing mean arterial pressure and heart rate. Animals in Gp3, except for the two animals that had a wound in the left ventricle and a perforated ventricular septum and died at 18 min and 34 min post-trauma, maintained haemodynamic stability. Sixty minutes after CPB separation, mean arterial pressure in Gp2 was lower than that in Gp3 ($p<0.05$) (fig 1), and heart rate in Gp2 was much lower than that in Gp3 ($p<0.05$) (fig 2).

Cardiac output and pulmonary artery wedge pressure

Cardiac output in Gp3 animals was much lower immediately after CPB separation compared to pre-trauma values. However, 120 min after CPB cardiac output remarkably went up and at 180 min slightly decreased. PAWP was higher than pre-trauma after CPB separation and was slightly reduced at 120 min and 180 min (table 5).

Free haemoglobin

The chest drainage at 2 h post-trauma was 360 (86) ml, 399 (46) ml, and 403 (51) ml in groups 1, 2 and 3, respectively. The differences between the groups were not significant. In Gp1, blood in the chest drainage showed an increased number of red blood cells and haemoglobin ($p<0.01$ and $p<0.05$, respectively) and a decreased number of platelets ($p<0.01$) after the trauma. In Gp2 and Gp3, the free haemoglobin was significantly increased compared to pre-trauma levels at 1 h (2.82 (1.34) g/l) and 3 h (2.97 (0.66) g/l) post-trauma (table 6).

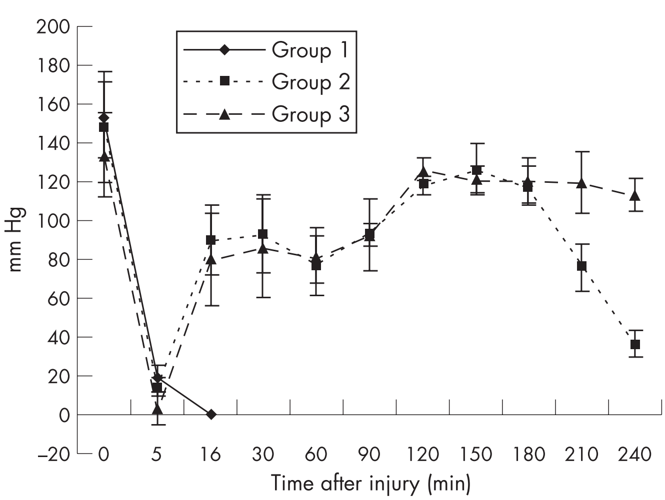

Figure 1 Mean arterial pressure measured in the subclavian artery after gunshot inflicted penetrating cardiac wounds. Mean arterial pressure decreased in Gp1 and animals died within 18 min after injury. Animals in Gp2 and Gp3 maintained mean arterial pressure ranging from 60–90 mm Hg during emergent cardiopulmonary bypass (CPB). However, following the termination of CPB, mean arterial pressure decreased in Gp2. In Gp3, after terminating CPB, most animals maintained haemodynamic stability. Mean arterial pressure was lower in Gp2 compared to Gp3 ($p<0.05$) at 60 min following termination ($p<0.05$).

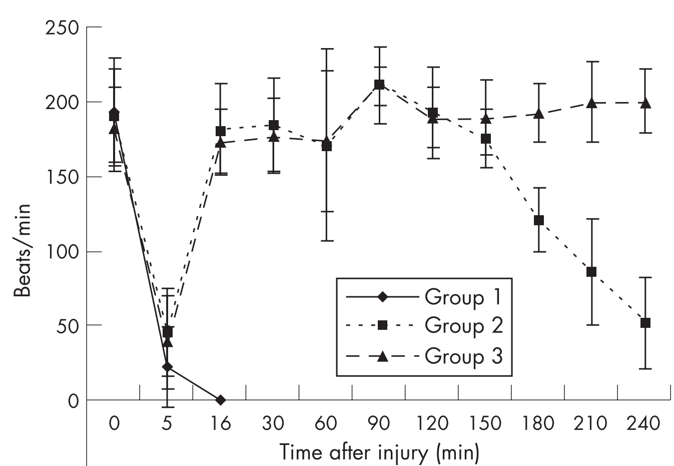

Figure 2 Heart rate measured after gunshot inflicted penetrating cardiac wounds. In Gp1, mean (SD) heart rate decreased from 193 (136) beats/min to 22 (27) beats/min and animals died between 9.6–18 min post-trauma. Animals in Gp2 and Gp3 maintained heart rate within a normal range. Following the termination of cardiopulmonary bypass (CPB), animals in Gp2 showed decreasing heart rate, while in Gp3, heart rate was maintained. Heart rate in Gp2 was lower than in Gp3 at 60 min following termination of CPB ($p<0.05$).

Table 5 Mean (SD) cardiac output and pulmonary artery wedge pressure in Gp3

	Pre-trauma	Weaning off CPB	120 min post-trauma	180 min post-trauma
CO (litres/min)	2.28 (0.26)	1.25 (0.22)*	2.04 (0.15)	1.95 (0.27)
PAWP (mm Hg)	15.53 (2.93)	17.1 (5.40)	13.73 (4.20)	11.85 (3.23)

CO, cardiac output; CPB, cardiopulmonary bypass; PAWP, pulmonary artery wedge pressure.
*$p<0.01$ vs pre-trauma.

Arterial blood gas

After injury, oxygen and carbon dioxide pressure changed in all three groups. In Gp1, oxygen decreased and carbon dioxide increased at 8 min post-trauma. Similarly, the oxygen pressure decreased and the carbon dioxide pressure increased after injury in both Gp2 and 3, but there were no differences between Gp2 and 3 (table 7).

Pulmonary water content

Pulmonary water content was greater in Gp2 (84 (5)%) and Gp3 (81 (6)%) compared with Gp1 (74 (5)%) ($p<0.01$). The decrease from Gp2 to Gp3 was also significant ($p<0.05$).

Enzyme labelled compound assay

Creatine kinase increased after trauma ($p<0.01$), but no differences between pre- and post-trauma in glutamic oxalacetic transaminase, urea nitrogen and creatinine were found in Gp2 and 3 (table 8).

Histological observations

Large amounts of serum and red blood cells permeated into the alveolar cavity in both the right and left lungs. The myocardium ruptured and both red blood cells and inflammatory cells were observed in the myocardium interstitium.

DISCUSSION

PCW presents a significant surgical challenge because of the unique clinical course and the need for emergent surgery. Often, surgery, which may include CPB, must be initiated in a prompt yet careful fashion to optimise outcome and minimise morbidity.[5] In patients with PCW, the most common initial clinical presentation is cardiac tamponade, which together with uncontrolled haemorrhage, results in 17% of patients with no blood pressure and 54–69% of patients with hypotension (systolic blood pressure 30–90 mm Hg).[6 7] Rhee reported in patients with PCW that one of the important factors influencing outcome was signs of life (SOL). If SOL were present on arrival at the hospital, 11.5% of patients survived in contrast to a 2.6% survival rate if no SOL were present. If SOL were present during transport, 8.9% of patients survived, while the absence of SOL in the field yielded a survival rate of only 1.2%.[8] Therefore we hypothesised that maintaining haemodynamic stability and preventing hypotension after PCW would increase patient survival rate.

The use of CPB was a milestone in the development of cardiac surgery because it takes the place of the heart and lungs and supplies blood to the body during surgery. CPB was first used in trauma cases in 1972. Emergent CPB was used effectively, in the course of cardiac repair, to maintain heart and lung function during haemorrhagic shock from right ventricle rupture.[9] However, because the use of heparin during CPB increases the risk of haemorrhaging, CPB is difficult after trauma and generally performed only in the operating room. Recently, newly developed heparin-coated conduits and percutaneous puncturation have made the use of CPB in the field possible.[10] Ullrich reported that emergent CPB was successfully used on a patient with PCW and cardiac arrest, and that the extra-organ pipeline coated with heparin circumvented the need to use systemic heparin, and thus avoided exacerbating haemorrhage.[11] Finally, Webb described the successful surgical repair of PCW with emergent CPB, and pointed out that timely application of emergent CPB was essential in life threatening cases of PCW.[8]

Nonetheless, despite the introduction of CPB, PCW represents an ongoing clinical challenge to trauma surgeons. Reported mortality rates range from 35–81%.[2 3 6 12 13] PCW commonly occurs as a result of gunshot or stab wounds,[14] with gunshot wounds causing more fatalities than stab wounds.[6 15 16] Approximately 62–84% of patients with gunshot inflicted PCW die in the field or during transport.[1–3] In one retrospective analysis, 19.5% of patients arriving at the hospital with SOL after all categories of PCW died while 24% of patients with gunshot inflicted PCW died. This suggests PCW from gunshot wounds are the most lethal form of cardiothoracic trauma.[17] Despite the high mortality rate from gunshot inflicted PCW, no animal model has been developed. The only data available are retrospective analyses of clinical cases of gunshot wounds in humans. Thus, no significant progress in care has been made in the last few decades.[4]

Previously, we successfully established an animal model of thoracic gunshot wounds in canines by decreasing the initial velocity of a bullet and thereby reducing the kinetic energy.[18] In this study, we used these established parameters to cause a PCW with a gunshot. Similar to other studies, the right ventricle was the most severely wounded cardiac chamber,[19] and as in human patients with PCW, the primary cause of death was severe hypotension caused by uncontrolled haemorrhage.[20] Conventional treatments after the gunshot inflicted PCW failed to save the lives of the canines (Gp1). Thus, in order to save lives after PCW, it is critical to maintain haemodynamic stability and support circulation as quickly as possible.

We hypothesised that prompt establishment of CPB could substitute the heart's function in sustaining SOL. Indeed, the evaluation of blood enzymes in our study suggests that in Gp2 and Gp3 hepatic and renal functions were maintained, and therefore that sufficient systemic circulation was maintained. Beside conventional treatments, emergent CPB was established in Gp2 and Gp3 within 10 min. As shown in our results, mean arterial pressure ranged from 60–90 mm Hg; additionally, in Gp3 cardiac output index varied from 1.95–2.04 litres/min and pulmonary arterial wedge pressure was below 11.85 mm Hg.

Table 6 Free haemoglobin in plasma

Group	Pre-trauma	120 min post-trauma	180 min post-trauma
2	0.31 (0.20)	2.82 (1.34)	6.68 (1.66)*
3	0.34 (0.12)	2.97 (0.66)	3.85 (0.99)

*$p<0.01$ vs pre-trauma.

Table 7 Arterial oxygen (Pa_{O_2}) and carbon dioxide (Pa_{CO_2}) pressure of Gp2 and Gp3 (mm Hg)

Group		Pre-trauma	60 min post-trauma	180 min post-trauma
2	Pa_{O_2}	111.23 (15.23)	88.73 (15.08)*	84.08 (17.33)*
	Pa_{CO_2}	34.2 (6.9)	43.58 (21.23)	36.38 (7.73)
3	Pa_{O_2}	112.65 (28.65)	96.9 (10.35)	75.15 (17.33)†
	Pa_{CO_2}	36.15 (13.73)	36.15 (9.9)	39.9 (13.65)

* $p<0.05$, †$p<0.01$ vs pre-trauma.

Table 8 Trauma effects on blood chemistry

		ALT (IU/l)	CK (IU/l)	BUN (mmol/l)	Cr (IU/l)
2	Pre-trauma	31 (16)	210 (186)	2.6 (1.1)	61 (13)
	Pre-death	60 (35)	1204 (686)	2.7 (1.5)	57 (14)
3	Pre-trauma	28 (13)	195 (199)	2.9 (1.3)	51 (14)
	Pre-death	55 (37)	1098 (658)*	3.0 (0.9)	56 (13)

ALT, glutamic oxalacetic transaminase; BUN, blood urea nitrogen; CK, creatine kinase; Cr, creatinine.
*$p<0.01$ vs pre-trauma.

However, pulmonary water content was greater in Gp2 and Gp3 compared to Gp1, which indicates that CPB leads to greater retention of intrapulmonary water and might impair pulmonary function. Oxygen pressure in arterial blood was higher than 75 mm Hg and carbon dioxide was lower than 45 mm Hg. These findings suggest that CPB might meet the physiological requirements of vital organs. Moreover, these data suggest that when initiated quickly, CPB might be helpful in patients with PCW from gunshots, especially when combined with surgery. Compared to Gp2, shorter duration of CPB in Gp3 resulted in less retention of intrapulmonary water, which could indicate that earlier separation of CPB might be helpful for pulmonary recovery.

This is an animal study and extrapolation into clinical practice is not easy. But PCW is a lethal injury, there has been little advance in care and reduction in mortality following gunshot inflicted PCW, and any work in this area is welcomed. This study shows that the establishment of CPB in the field might be effective in sustaining haemodynamic stability and meeting the physiological requirements of vital organs. Under the conditions of emergent CPB, surgical repair might save more lives.

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