Effect of the Abdominal Aortic and Junctional Tourniquet on Chest Compressions in a Swine Model of Ventricular Fibrillation

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Keywords: Cardiopulmonary resuscitation, CPR, Ventricular Fibrillation, Abdominal Aortic and Junctional Tourniquet, Cardiac Arrest

The views expressed are those of the authors and do not reflect the official views of the Department of Defense or its Components. The experiments reported herein were conducted according to the principles set forth in the National Institute of Health Publication No. 80-23, Guide for the Care and Use of Laboratory Animals and the Animal Welfare Act of 1996, as amended.

Short Title: CPR with Abdominal Aortic and Junctional Tourniquet

Abstract

Introduction: Mortality for out-of-hospital cardiac arrest is high when traditional chest compressions are used without adjuncts. The abdominal aortic and junctional tourniquet (AAJT) is a device with a wedge-shaped air bladder that is used to occlude the aortic bifurcation. This augments blood flow to vital organs such as the heart and brain. We have previously shown that the addition of an AAJT to mechanical chest compressions leads to an increase in rate of survival in a model of traumatic cardiac arrest.

Hypothesis: This study was designed to determine if application of the AAJT would lead to more effective chest compressions as measured by improved hemodynamic parameters and an increased rate of return of spontaneous circulation (ROSC).

Methods: Yorkshire swine (n=6 per group) underwent general anesthesia and instrumentation. Ventricular fibrillation (Vfib) was electrically induced using spinal needles placed in contact with the left ventricle and allowed to progress untreated for eight minutes. After four minutes of chest compressions, animals were allocated into groups with or without the AAJT. The AAJT was inflated if selected, and chest compressions were continued for six minutes. Following a total of ten minutes of compressions, the animals entered into a ten-minute advanced cardiac life support phase. Hemodynamics and blood gas measurements were compared between groups.

Results: ROSC or cardioversion from Vfib was not achieved in either group. The AAJT group had improved hemodynamic parameters with significantly higher carotid diastolic pressure and higher blood flow in the carotid artery as compared with the non-AAJT group using repeated-measures ANOVA ($p = 0.016$ and 0.028 respectively). However, no significant differences were observed with coronary perfusion pressure or end tidal CO_2.

Conclusion: The AAJT did not confer a survival advantage during chest compressions in our swine model of cardiac arrest. However, there was an improvement in cerebral blood flow and carotid diastolic blood pressure while the AAJT was in place.

Keywords: Cardiopulmonary resuscitation, CPR, Ventricular Fibrillation, Abdominal Aortic and Junctional Tourniquet, Cardiac Arrest

1 Introduction

Cardiac arrest (CA) is a worldwide leading cause of death. The sudden loss of cardiac function leads to malperfusion of all vital organs. Out-of-hospital cardiac arrest (OHCA) incidence was reported to be 110.8 per 100,000 people in the United States [1]. Recovery from OHCA are low with a 10.8% survival rate and only a 9.0% return to good functional status for the survivors at hospital discharge [1]. Cardiopulmonary Resuscitation (CPR) is currently the main therapy to treat OHCA and improvement to the efficacy of CPR could lead to substantial reduction in cardiac arrest deaths and improve patients' functional status.

CA can be caused by myocardial infarction, physical stress, inherited disorders, and trauma. Most cases of OHCA are associated with ventricular fibrillation (Vfib). Vfib is characterized by high frequency, disorganized excitation of the ventricles which inhibits systemic perfusion. Current treatment of Vfib is CPR concurrent with the use of a defibrillator as part of Basic Life Support (BLS) and Advanced Cardiopulmonary Life Support (ACLS).

Various adjuncts and variations of CPR have been studied to treat CA and Vfib and include adjustments to the rate of chest compressions, depth of compressions, and focusing the location of compressions. These have been demonstrated to improve the efficacy of CPR in both the laboratory and patient setting[2-5]. Outcomes indicating improved CPR efficacy include increased coronary perfusion pressure and increased diastolic blood pressure. Application of tourniquets to all four limbs during CPR increased coronary perfusion pressure, a measure of successful chest compressions, for treating Vfib in swine [6]. Positioning compressions above the left ventricle led to an increase in both return of spontaneous circulation (ROSC) and survival in a CA model of Vfib [7,8]. Finally, the combination of compression-decompression with an impedance threshold device has shown some success, but has been subject to mixed results [9,10]. Unfortunately, none of these improvements have been widely adopted as they are difficult to apply in an expeditious matter.

The Abdominal Aortic and Junctional Tourniquet (AAJT) is a device that was originally designed to be placed around the abdomen over the umbilicus to occlude blood flow below the aortic bifurcation [11-13]. The AAJT has the potential to be used as an adjunct to improve the efficacy of chest compressions. Application of the AAJT could serve to increase cerebral and coronary blood delivery from each compression due to the sequestration of blood above the location of occlusion. Additionally, the AAJT may limit diaphragm movement during chest compressions leading to an increase in intrathoracic pressure and thereby heart compression [14]. The AAJT has previously shown efficacy in treating traumatic cardiac arrest following hemorrhage in a swine model [15]. ROSC, survival, and carotid flow were all improved in animals with AAJT compared to those without the tourniquet.

The aim of this study was to investigate potential changes in hemodynamic parameters in response to the use of the AAJT to augment chest compressions in a swine model of Vfib. We hypothesized that the addition of the AAJT during BLS would allow for more effective chest compressions leading to ROSC following Vfib. Outcomes of this study included ROSC, survival, and other indicators of effective compressions such as diastolic blood pressure, carotid flow, and coronary perfusion pressure.

2 Methods

We conducted a prospective, randomized, large animal model study. It was approved by the United States Air Force 59th Medical Wing Institutional Animal Care and Use Committee, San Antonio, TX. Animals were housed in an AAALAC-accredited facility and treated according to "The Guide for the Care and Use of Laboratory Animals"[16]. All experiments were carried out during the months of February and March. An overview of the experimental procedures is given in Figure 1.

2.1 Surgical preparation

Twelve Yorkshire-landrace swine (45 to 55 kg) were individually housed for at least seven days to allow for acclimation. Food was withheld the night prior to undergoing surgical procedures. Sedation was performed using an intramuscular injection of 4.4 mg/kg tiletamine-zolazepam and 2.2 mg/kg ketamine. Buprenorphine was also given by intramuscular injection at 0.01 mg/kg for alleviation of pain. Anesthesia was induced with 2-4% isoflurane, but was adjusted to a minimum alveolar concentration of 1.2 or higher following intubation. Ventilation was managed using a volume-controlled setting of 7-10 mL/kg at 40% oxygen while surgical preparation was in progress.

Vascular access was obtained in the neck via a midline cut-down. Catheterization was achieved using an 8.5 Fr catheter introducer (Teleflex, Morrisville, NC) as follows: 1) right external jugular vein – fluid and drug administration, 2) right internal jugular vein – right atrium pressure, 3) right carotid artery – invasive arterial pressure and arterial blood sampling. The left carotid was instrumented with a flow probe (Transonic Systems, Ithaca, NY). Finally, the left femoral artery was accessed by ultrasound-guided percutaneous approach to measure blood pressure below the AAJT. Following line placement, the animal was moved to a custom-built, wedge-shaped table to limit animal movement during compressions. The AAJT was placed under the animal but not buckled for rapid inflation if selected to the AAJT group. Lactated Ringer's solution (10 mL/kg) was delivered during surgical preparation to replace fluid deficits caused by overnight fasting. Following surgical manipulation, FiO_2 was adjusted to 0.21, 100 u/kg heparin was administered, and a 10-minute stabilization period was observed.

2.2 Injury and Intervention

Ventricular fibrillation (Vfib) was induced with a 3-second, 60 Hz, 100 mA electric current delivered across two spinal needles (22 G x 3.50 in; BD Medical, Franklin Lakes, NJ) placed in contact with the left ventricular pericardium identified by ultrasound. Vfib was confirmed by ECG and sudden loss of blood pressure. Immediately following Vfib induction, defibrillation pads were placed over the left and right lateral chest along the midclavicular line as previously described [17]. Initiation of Vfib was identified as T_0.

BLS was initiated following eight minutes of Vfib using an automatic mechanical compression device (AMCD; Thumper 407CC, Michigan Instruments, Grand Rapids, MI). The AMCD was positioned in the center of the chest, 12 cm from the manubrium. Compressions were performed at a rate of 100 repetitions / min, with a compression depth of 5 cm and a compression-ventilation ratio of 30:2. After the start of compressions, the animals were assigned to the control or experimental group via random selection of sealed envelope containing the group allocation. A total of six pigs were selected to each

group. If assigned to the AAJT group, the AAJT was positioned and fastened. After four minutes of compressions, the windlass was tightened and the AAJT inflated until it reached an internal pressure of 300 mmHg.

Following a total of 10 minutes of BLS, ACLS was initiated with a biphasic 120 Joule defibrillation attempt using a ZOLL M-Series defibrillator (ZOLL Resuscitation Products, Chelmsford, MA). The animal was then immediately put on mechanical ventilation at 100% oxygen and chest compressions resumed. Defibrillation attempts were made every two minutes if no organized rhythm returned during the ACLS phase. Epinephrine (0.01 mg/kg) was administered two minutes and six minutes into the ACLS period (T20 and T24). Amiodarone (5mg/kg) was administered at four and eight minutes following the initiation of ACLS (T22 and T26).

2.3 Termination of Protocol

Animals were considered expired if they did not achieve a ROSC by the end of the 10-minute ALS period. Following a ROSC, the animals were considered expired if their MAP fell below 20 mmHg and their $EtCO_2$ fell below 15 mmHg for two minutes.

2.4 Outcomes and Analysis

The primary outcome of the study was ROSC. Secondary outcomes included carotid diastolic pressure, carotid flow, end tidal CO_2, coronary perfusion pressure (CPP), and arterial blood gas (ABG) values. CPP was manually calculated using the end diastolic method [Otlewski MP 2009 Methods for calculating]. ABGs were evaluated by a blood gas analyzer (ABL800; Radiometer, Brea, CA) and sampling occurred at baseline, at the end of the VF period, at the end of BLS, and at the end of ACLS.

Sample size was calculated to be able to demonstrate a 70% delta in survival rate. Power analysis was performed using SAS version 9.4 for Windows (SAS Institute, Cary, NC). A sample size of seven per group was calculated to achieve 80% power of a test of difference in the proportion of survival for a 2-sided test with a significance level of 0.05.

Student's t-test was used when comparing single continuous variables between groups. Two-way repeated measure ANOVA with Student-Newman-Kuels post-hoc analysis was used when comparing variables over time. Data is presented as mean ± standard deviation, except in graphs where data is mean ± standard error of the mean. Excel 2016 (Microsoft, Redmond, WA) was used for data management, and statistical tests were performed with Sigmaplot 12 (Systat Software, Chicago, IL).

3 Results

3.1 Baseline characteristics

A total of 17 animals were entered into the study: three animals were utilized for technical refinement and six animals per experimental group. Two animals used were excluded from analysis: one due to difficulty with oxygenation and one due to iatrogenic bleeding. There were no significant differences between groups (Table 1) at baseline with regards to hemodynamics or laboratory values. No animals in this study survived until the end of the experiment. Furthermore, no animals attained a ROSC during the BLS or ACLS phases of the experiment.

3.2 Hemodynamic and pulmonary parameters

The systolic blood pressure (SBP) (Figure 2) was similar between groups with no statistically significant differences. Mean arterial pressure showed similar results. However, diastolic blood pressure (DBP), was significantly different between groups. Post-hoc analysis revealed that this difference occurred from T8 to T11 and from T14 until the end of experimentation. Interestingly, this difference between groups in DBP was observed after compressions started, but before the AAJT was inflated. Peaks of DBP were observed shortly following epinephrine administration (T21 and T25), but were not observed in the SBP.

Similar to carotid pressure, systolic and right atrium MAP were not significantly different between groups ($p = 0.761$ and $p= 0.505$, respectively). However, right atrium diastolic pressure was significantly different between groups. This difference began shortly after inflation of the AAJT and remained until the end of chest compressions. No substantial transitory increase in diastolic blood pressure was observed following epinephrine administration in contrast to the increases seen in the carotid.

Table 2 shows the values of CPP during chest compressions at critical points in the experiment: before the AAJT was applied, at the end of the last round of BLS, and at the end of the last round of ACLS. The only significant difference was prior to AAJT inflation while the AAJT was being positioned.

Carotid arterial blood flow was significantly different (Figure 3) following inflation of the AAJT. However, these differences disappeared following the onset of ACLS treatment. Both groups had a dip in carotid blood flow following the administration of epinephrine. Differences between groups returned towards the end of the ACLS period.

3.3 Lab Values

No differences were observed between groups at any timepoint with respect to potassium, lactate, or base excess (Table 3). Both groups displayed an increase in extracellular potassium and lactate. However, pH was significantly lower in the AAJT group at the end of BLS and ACLS. The AAJT group had a lower pO_2 and higher pCO_2.

4 Discussion

This animal model was used to assess the effectiveness of the AAJT device during CPR. It demonstrated that, although there was no improvement in the ability to achieve ROSC, there was improvement of the hemodynamic measures of CPR efficacy. Variations of this swine model have been used in the past to examine position of chest compressions, rate of compressions, defibrillation waveform, and various adjuncts due to the basic similarity between swine and human vasculature and anatomy. We theorized that the addition of the AAJT would concentrate the circulating volume and effectively increase both preload and afterload during compressions. Optimally, these effects would increase diastolic arterial pressure and coronary perfusion pressure, which are both thought to increase the rates of ROSC [18,19].

The addition of the AAJT provided some benefit for chest compression efficacy. The diastolic blood pressure, an indicator of CPR efficacy, was significantly higher with the addition of the AAJT. Carotid blood flow was also significantly higher in the AAJT group than the control group indicating improved brain perfusion. Increased brain perfusion is likely an important finding here, as ROSC and survival from cardiac arrest does not always correlate with good neurologic recovery.

The results presented here are contrasted with a similar study that showed a clear benefit to utilizing the AAJT during CPR [15]. In that study, subjects underwent a controlled hemorrhage to create a traumatic cardiac arrest as defined by a systolic blood pressure less than 10 mmHg. With the AAJT applied, ROSC was observed in 83% of animals with the tourniquet compared to only 17% without the tourniquet. Increases in blood pressure and carotid blood flow were also observed. The lack of clear benefit to treat Vfib in this study could be that the AAJT does not provide an appreciable benefit in normovolemic patients and the AAJT has no effect on defibrillation. Alternatively, the current study utilized smaller subjects (45 to 55 kg) compared to the previous study (70 to 90 kg).

Blood gas analysis showed that the subjects with the AAJT had a lower pH, lower pO_2, and higher pCO_2. These differences appeared at the end of BLS and continued through to the end of ACLS. This data suggests that the tourniquet was affecting the ventilation of the subjects and alterations in ventilation may be necessary while the tourniquet is applied.

Future investigations looking to further this research would need to assess the neurologic effect, if any, of including the AAJT during chest compressions. A shorter duration of Vfib will likely lead to increased ROSC and survival allowing for behavioral analysis after recovery to determine neurologic status. Defibrillation technique should also be optimized to determine the appropriate power and waveform to rescue a rhythm from Vfib.

There are some limitations with this study. No survival or ROSC was observed in the current study. There was no observed improvement in coronary perfusion pressure while the AAJT was applied. As no animal in either group was observed to convert from Vfib to a sinus rhythm following a defibrillation attempt, the model may not have been optimal. It has been observed that with increased time in Vfib, there is a decrease in odds of obtaining ROSC[20]. In the current experiment, animals were allowed to undergo eight minutes of Vfib before chest compressions were initiated. Additionally, the biphasic waveform at 120J may not have been the proper waveform or power for use in this model of Vfib with

large swine. The physical differences between quadrupeds and bipeds results in less occlusion as a percentage of body mass, and therefore a lower blood volume, in the swine model as compared to humans by the AAJT device. Furthermore, the position of the AAJT could have different effects on diaphragm movement during compressions in humans compared to pigs. The mobility of the diaphragm during CPR influences intrathoracic pressure, thereby heart compression. These differences could limit the ability to translate our findings directly into a human patient model. Lastly, the detrimental effects of prolonged Vfib may have been too physiologically catastrophic and decreased our potential for ROSC in both groups.

5 Conclusion

In conclusion, our study did not demonstrate an improvement in ROSC or coronary perfusion pressures in a swine model of Vfib. The AAJT did improve cerebral blood flow but this did not translate into improved survival for this model.

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Table 1. Baseline Values

	-AAJT	**+AAJT**	**p-value**
n	6	6	
Weight	47.2 ± 2.6	47.8 ± 1.4	0.635
Sex (M)	4/6 (67%)	1/6 (17%)	0.242
Waist (in)	31.9 ± 2.1	32.8 ± 1.0	0.399
Systolic (mmHg)	84.2 ± 10.4	90.8 ± 8.4	0.250
Diastolic (mmHg)	56.3 ± 5.6	62.3 ± 9.1	0.200
MAP (mmHg)	65.2 ± 6.5	72.0 ± 8.1	0.142
pH	7.38 ± 0.04	7.43 ± 0.07	0.161
Potassium (mmol/L)	3.68 ± 0.25	3.6 ± 0.15	0.682
Lactate (mmol/L)	0.85 ± 0.16	1.1 ± 0.46	0.235
Base excess (mmol/L)	5.95 ± 1.7	6.7 ± 2.7	0.571

Table 2. Coronary Perfusion Pressure

	-AAJT	+AAJT	p-value
Post 1st Rd BLS	8.4 ± 5.9	20.4 ± 3.2	0.0014**
Post AAJT Inflation	6.9 ± 5.8	10.8 ± 3.0	0.170
End BLS/Start ACLS	3.9 ± 8.5	8.0 ± 3.9	0.307
End ACLS	3.7 ± 6.9	7.7 ± 7.9	0.371

**, $p < 0.01$

Table 3. Lab Values

	-AAJT	+AAJT	p-value
Arrest (t=0)			
pH	7.52 ± 0.03	7.51 ± 0.04	0.936
pO_2	76 ± 14	69 ± 6	0.297
pCO_2	36 ± 2	37 ± 4	0.561
Potassium (mmol/L)	4.4 ± 0.8	4.0 ± 0.4	0.346
Lactate (mmol/L)	1.7 ± 0.5	1.3 ± 0.3	0.104
Base excess (mmol/L)	7.2 ± 2.1	7.0 ± 1.9	0.900
End of BLS			
pH	7.15 ± 0.04	7.09 ± 0.04	0.0496*
pO_2	70 ± 22	50 ± 8	0.063
pCO_2	72 ± 11	84 ± 9	0.052
Potassium (mmol/L)	5.7 ± 0.7	6.4 ± 1.4	0.305
Lactate (mmol/L)	6.5 ± 0.9	6.9 ± 0.7	0.459
Base excess (mmol/L)	-4.2 ± 1.9	-4.2 ± 1.0	0.941
End of ACLS			
pH	7.15 ± 0.06	7.02 ± 0.05	0.0026**
pO_2	54 ± 11	39 ± 5	0.009**
pCO_2	57 ± 9	80 ± 15	0.009**
Potassium (mmol/L)	5.7 ± 2.0	6.4 ± 2.0	0.447
Lactate (mmol/L)	9.3 ± 1.1	9.2 ± 2.7	0.946
Base excess (mmol/L)	-8.2 ± 1.5	-9.7 ± 3.8	0.391

*, $p < 0.05$; **, $p < 0.01$

Figures

Figure 1. Experimental Schematic. Times are shown in minutes. Six animals were allocated to each group. Epi: Epinephrine; Amio – Amiodarone;

Figure 2. Hemodynamic Parameters. Ventricular Fibrillation initiated at T_0. Arrow indicates time of AAJT inflation * - $p < 0.05$;

Figure 3. Carotid Flow. Ventricular Fibrillation initiated at T_0. Arrow indicates time of AAJT inflation * - $p < 0.05$;

Figure 1. Experimental Overview

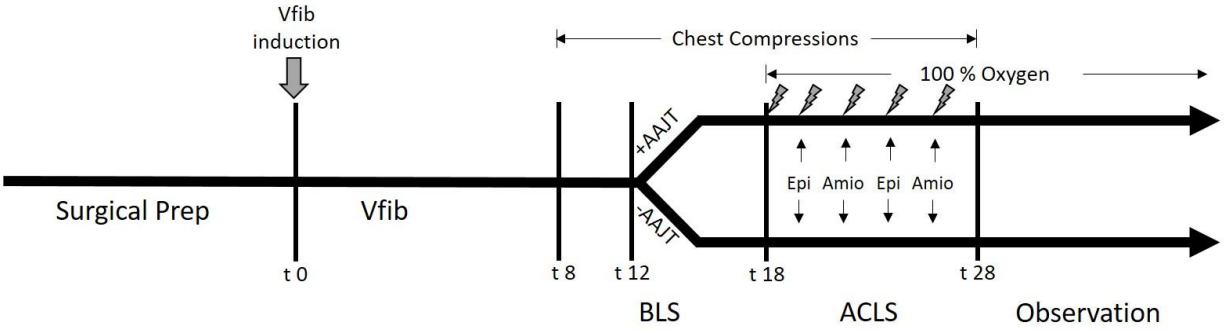


Figure 2. Hemodynamic Parameters

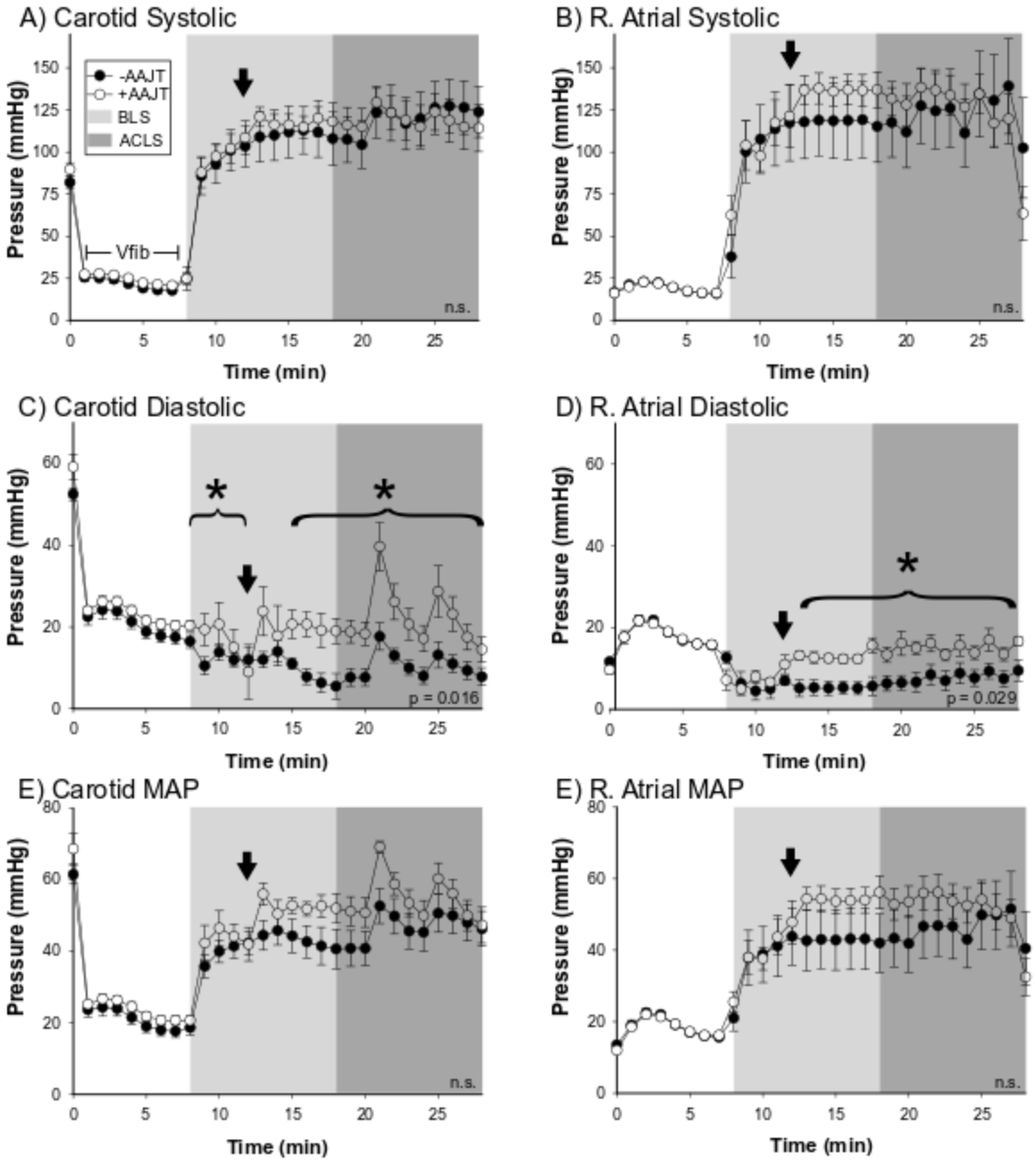
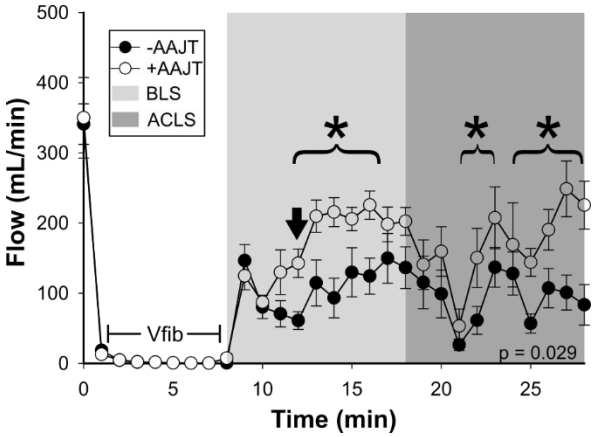


Fig 3. Flow


500
400
300
200
100
0
Flow (mL/min)
-AAJT
+AAJT
BLS
ACLS
Vfib
*
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p = 0.029
0
5
10
15
20
25
Time (min)