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Open chest cardiac massage offers no benefit over closed chest compressions in patients with traumatic cardiac arrest

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Background: Open chest cardiac massage (OCCM) is a commonly performed procedure after traumatic cardiac arrest (TCA). OCCM has been reported to be superior to closed chest compressions (CCC) in animal models and in non-traumatic cardiac arrest. The purpose of this study is to prospectively compare OCCM versus CCC in traumatic cardiac arrest using end-tidal carbon dioxide (ETCO2), the gold standard for determining the effectiveness of chest compressions and detection of return of spontaneous circulation (ROSC), as the surrogate for cardiac output and marker for adequacy of resuscitation.

Methods: This prospective observational study enrolled patients over a nine-month period directly presenting to a Level 1 trauma center after TCA. Continuous high-resolution ETCO2 measurements were collected every six seconds for periods of CCC and OCCM, respectively. Patients receiving CCC-only were compared to patients receiving CCC followed by OCCM. Student t-tests were used to compare ETCO2 within and between groups.

Results: Thirty-three patients were enrolled (16 OCCM, 17 CCC-only). Mean time of CCC prior to OCCM was sixty-six seconds. Within the OCCM group, final, peak, mean, and median ETCO2 levels significantly increased when comparing the initial CCC period to the OCCM interval. Using a time-matched comparison, significant increases were observed in final and peak but not mean and median values when comparing the first minute of CCC to the remaining time in the CCC-only group. However, when periods of OCCM were compared to equivalent periods of CCC-only, there were no differences in the initial, final, peak, mean, or median ETCO2 values. Correspondingly, no difference in rates of ROSC was observed between groups (OCCM 23.5% vs CCC 38.9%, p=0.53).

Conclusions: Although we could not control for confounders, we found no significant improvement in ETCO2 or ROSC with OCCM. With newer endovascular techniques for aortic occlusion, thoracotomy solely for performing OCCM provides no benefit over CCC.

Level of Evidence: Level III, therapeutic

Key Words: Traumatic cardiac arrest; open chest cardiac massage; closed chest compressions; End-tidal carbon dioxide

Background:

Emergency department thoracotomy (EDT) is a commonly performed procedure after traumatic cardiac arrest. For the highest probability of survival, the general indications for an EDT are 1. blunt trauma arriving to the hospital with less than ten minutes of pre-hospital cardiopulmonary resuscitation (CPR) with closed chest compressions (CCC) 2. penetrating torso trauma with less than fifteen minutes of pre-hospital CPR and CCC and 3. penetrating neck and extremity trauma with less than five minutes of pre-hospital CPR and CCC.(1-3) However, mortality remains extremity high despite this invasive procedure especially in patients sustaining blunt trauma.(4-6)

The primary purpose of EDT is to provide direct access to injuries amenable to repair in the ED such as a cardiac wound or temporizing control of a major thoracic vascular injury. Additionally, EDT gives access to the heart to perform open chest cardiac massage (OCCM), and depending on the injury pattern, the ability to occlude the proximal thoracic aorta for temporary hemostasis. Aortic cross clamping for massive hemoperitoneum was described forty years ago as a means for proximal vascular control prior to laparotomy for definitive control.(7, 8)

OCCM allows for direct hand-assisted cardiac compression. This is thought to simulate cardiac contraction, augment cardiac perfusion, increase direct filling of the heart and improve perfusion to distant organs. This technique has been reported to be superior to CCC in improving outcomes following cardiac arrest in non-traumatic cardiac arrest.(9, 10) This, of course, is vastly different in trauma where patients with cardiac arrest are usually profoundly hypovolemic. Various physiologic variables, such as

arterial pressure differences, cardiac indices, and acid-base status, have been used to demonstrate that OCCM is superior to CCC in improving blood pressure and end organ perfusion.(11-15) However, most of these studies have been published over ten years ago prior to current modern management and have been performed in animal models or in the setting of out-of-hospital, non-traumatic cardiac arrest.(16)

End-tidal carbon dioxide (EtCO2) monitoring is now the gold-standard method to determine CPR quality, adequacy of chest compressions, detection of return of spontaneous circulation (ROSC) and to determine when to cease CPR efforts.(17, 18) No study to date has used EtCO2 as the means of efficacy of chest compression when comparing open versus closed CPR in the patient with traumatic cardiac arrest. With the recent advent of endovascular aortic occlusive techniques for temporary proximal hemorrhage control, the need to perform EDT solely for OCCM has come into question.

The purpose of this study was to prospectively compare OCCM versus CCC in our patients with both penetrating and blunt trauma presenting in extremis using EtCO2 as the surrogate for cardiac output (CO) and marker for adequacy of resuscitation. Our hypothesis was that OCCM is more effective than CCC in raising EtCO2 values and thus CO.

Methods:

This prospective observational study over a nine-month period (April 2014 to December 2014) was approved by the University of Maryland School of Medicine Institutional Review Board (IRB). Patients presenting directly to our Level 1 trauma center with traumatic cardiac arrest and had CCC and/or OCCM were enrolled. Patients determined

to be dead on arrival were excluded from analysis, as were patients that received a REBOA device. The R Adams Cowley Shock Trauma center captures real-time video recordings in all trauma bays in the Trauma Resuscitation Unit (TRU). Thus the video recordings of the resuscitation period were analyzed to determine the exact time intervals for CCC and/or OCCM. In addition, using our vital sign data recorder (VSDR) continuous high-resolution EtCO2 measurements were collected every six seconds for periods of CCC and OCCM after EDT, respectively. Patients receiving CCC-only were compared to patients receiving CCC followed by OCCM, using equivalent periods in the CCC-only group to compare to the OCCM interval. Pearson's chi-square or Fisher's exact test was used to compare proportions. Paired one-sample t-tests and two-sample t-tests were used to compare EtCO2 within and between groups, respectively. A p value below 0.05 was considered statistically significant. All statistical analysis was done in Matlab (2014b, MathWorks, Natick, MA).

Results:

Thirty-three patients were enrolled; 16 in the OCCM group and 17 in the CCC-only group. Patients were predominately male (94%) and those who underwent OCCM were younger (31.4 ± 12.7 vs. 42.5 ± 18.8 years, p=0.08) with a higher percentage of penetrating trauma (81% vs. 47%, p=0.04). Gunshot wound was the primary mechanism of injury in both groups and mortality was near universal (Table 1). Mean time of CCC prior to OCCM was 66 seconds. Within the OCCM group, initial, final, and peak EtCO2 levels significantly increased when comparing the initial CCC period to the OCCM interval (Table 2). Figure 1a shows the changing trend of each case. The blunt trauma subgroup (gray lines) presented a generally increasing pattern. The penetrating trauma

subgroup (black lines) showed an ambivalent trend, which might be because of the limited number of cases (n=2). Using a time-matched comparison, significant increases in final, peak, mean and median values were observed when comparing the first minute of CCC to the remaining time in the CCC-only group (Table 2). The trend of each case in the CCC-only group is displayed in Figure 1b. When periods of OCCM were compared to equivalent periods in the CCC-only group, there was no sufficient evidence showing a difference in the initial, final, peak, mean, or median EtCO2 values (Table 2). Correspondingly, no difference in rates of ROSC was observed between groups (OCCM 23.5% vs. CCC 38.9%, p=0.53). Total time of active resuscitation for all non-survivors was 637 ± 67 seconds and there was no difference in resuscitation times when comparing the two groups (OCCM 720 ± 96 vs. CCC 556 ± 98, p = 0.21).

In the subset of penetrating trauma, initial, peak, and mean EtCO2 levels in the OCCM group were found to be significantly elevated in the OCCM phase as compared to the initial CCC period. However, there was no sufficient evidence demonstrating a difference in comparable values within the first minute of CCC versus the remaining time in the CCC-only group. Likewise, there were no significant differences in EtCO2 values when comparing OCCM periods with commensurate periods of CCC-only (Table 3).

In the subset of blunt trauma patients, no differences were identified in the OCCM group when comparing the initial CCC time period to the OCCM interval. Likewise, no difference in EtCO2 levels were noted when comparing the first minute to the remaining time in the CCC-only group. Finally, there was no sufficient evidence showing differences in EtCO2 values when periods of OCCM were compared to equivalent periods in the CCC-only group (Table 4).

It is worth pointing out that in the penetrating trauma subgroup, OCCM increased EtCO2 from averagely lower levels to averagely higher levels, compared to CCC in the same group (Table 3). Meanwhile, CCC increased EtCO2 from averagely lower levels to averagely higher levels, compared to OCCM in the blunt trauma subgroup (Table 4). Although through two-sample t-test, there still lacks evidence to show the significant difference (all p-values > 0.05), the injury type could be a factor in comparing the clinical effect of OCCM and CCC.

Discussion:

To our knowledge this study is the first to compare the use of CCC versus OCCM in the trauma population. In both blunt and penetrating trauma we found no sufficient evidence showing that OCCM could significantly increase EtCO2, and by proxy CO, as compared to CCC. Likewise, there was no improvement in ROSC with the institution of OCCM following CCC. Our findings are in contrast to the available literature on this topic, which is mostly comprised of animal studies or in patients with non-traumatic cardiac arrest. However, our 'n' for each group was low, therefore, we cannot definitely conclude that there was no difference.

Studies involving traumatic cardiac arrest are intrinsically difficult to perform, as there is little time to initiate data collection and few modalities in place with which to record data. EtCO2 recorded during the period of resuscitation, with time-stamped video to delineate the exact timing of CCC vs OCCM, allowed us to study the effectiveness of both methods.

Data from both basic science and clinical research have shown a tight correlation between EtCO2 and CO.(19, 20) This has translated in to the use of EtCO2 as an indicator for adequacy of CCC and ROSC.(21, 22) A positive correlation between higher EtCO2 and both ROSC and survival has been demonstrated in a number of studies.(18, 23-26) However, specific cut-off values for EtCO2 to determine futility of CPR or chance of survival remain unknown. Several authors have suggested that an EtCO2 level > 10mmHg predicts ROSC with a sensitivity of 100%.(18, 23, 24) Other studies found EtCO2 to be less predictive with a sensitivity below 90% to as low as 40%(27-29)

A systematic review by Touma and Davies evaluating the prognostic value of EtCO2 in cardiac arrest determined that no set EtCO2 value could be used as a predictor of mortality.(30) In addition, it is unclear whether initial, end, maximum, or average EtCO2 level is the best predictor. The position of several resuscitation organizations including the American Heart Association (AHA), while supporting the use of EtCO2 to assess adequacy of CPR, have not agreed upon a specific value of EtCO2 to predict ROSC or mortality(31) However, the AHA guidelines support the futility in CCC to achieve ROSC when EtCO2 levels are consistently below 10mmHg.(32) The initial mean EtCO2 values during the first minute in our groups were both below 10mmHg. However, during the subsequent time period mean, peak, and final EtCO2 values remained above 10mmHg suggesting care rendered should not be considered futile, based on AHA guidelines.

With the utility of EDT in question, several alternative devices have been developed to improve outcomes after cardiac arrest including a hand-held suction device (Cardiopump, Ambu International, Glostrup, Denmark) allowing for active compression and

decompression CPR (ACD-CPR), an inspiratory impedance threshold valve (ResusciValve, CPRxLLC, Minneapolis, MN), and a minimally invasive direct cardiac massage device (MIDCM, TheraCardia, San Clemente, CA), which provides internal cardiac massage without the need for an anterolateral thoracotomy.(33) All have shown some promise in improving perfusion pressures but this benefit has only been shown in animal models and small non-traumatic cardiac arrest patient populations. When comparing EtCO2, only the combination of ACD-CPR with an inspiratory impedance device has been shown to significantly increase EtCO2 levels as compared to CCC.(34, 35) Again this comparison was in non-traumatic cardiac arrest patients and long-term outcomes were not assessed.

The REBOA (Resuscitative Endovascular Balloon Occlusion of the Aorta) device is another less-invasive alternative or adjunct to EDT for temporary hemorrhage control. Renewed interest in its use has shown promising results when compared to EDT alone. An initial report by Brenner and colleagues described the application of the REBOA in profound traumatic shock.(36) In this report, the authors demonstrated the feasibility and success of hemorrhage control and maintenance of proximal arterial pressure using this device. In a subsequent study comparing REBOA to aortic cross clamping for proximal hemorrhage control, Moore et al found a mortality benefit of the REBOA over EDT with aortic clamping.(37) While this study did not focus on cardiac arrest patients, these data have brought into question the utility of EDT with aortic cross clamping for proximal vascular control. With the evolution of this device including a smaller profile and non-fluoroscopic insertion techniques, its use in trauma centers is expected to increase. While our study did not address aortic cross clamping, the combination of CCC with the

REBOA may be a safer and equally efficacious way of obtaining ROSC and hemorrhage control as compared to performing an EDT with OCCM and aortic cross clamping. Thus, an EDT could be limited to addressing intrathoracic injuries.

There are several significant limitations of our study that deserve mention. This was a relatively small cohort of patients, especially in the subgroup analysis, so definitive, meaningful comparisons in a larger study should be pursued to shed more light on this important topic and to reduce the possibility of a Type II error. This study was not randomized; therefore, the exact reason for EDT versus continuing with CCC could not be determined and we could not control for all confounders. Also, pre-hospital CPR time was not taken into account. In addition, medications and blood products administered, and the use of internal cardiac defibrillation were not taken into consideration. Due to the nature of our study design we were unable to determine the physician's decision for length of time for the resuscitative event, their indication for performing an EDT if performed, or the physician's reason for ending resuscitation. This further limited our study. Similarly, we were unable to specifically calculate injury scores or identify exact injuries as the cause of death; however, based on our survival rates all patients had high injury severity scores. Finally, we excluded all patients that received a REBOA device to prevent confounding of our data. However, with increased interest in and increased deployment of the REBOA, further studies investigating its utilization in conjunction with or as an alternative to EDT are warranted.

Conclusions:

While thoracotomy is necessary for the emergent surgical repair of thoracic injury, we found no sufficient evidence showing significant improvement in ROSC with OCCM. So far it lacks evidence showing that OCCM could provide a physiological advantage in improving cardiac output as measured by EtCO2 when compared to equivalent time periods of CCC. With newer endovascular techniques for aortic occlusion, thoracotomy solely for performing OCCM may not provide any benefit to the patient over CCC.

Authors Contributions:

MJB, BWB, LC literature search; MJB, MLB, TSM, DMS, study design; BWB, SY, PH, HL CLL data collection; LC, SY, PH, HL data analysis; MJB, BWB, SY, PH, HL, DSM data interpretation; MJB, BWB, MLB, DMS writing; TSM, DMS critical revision

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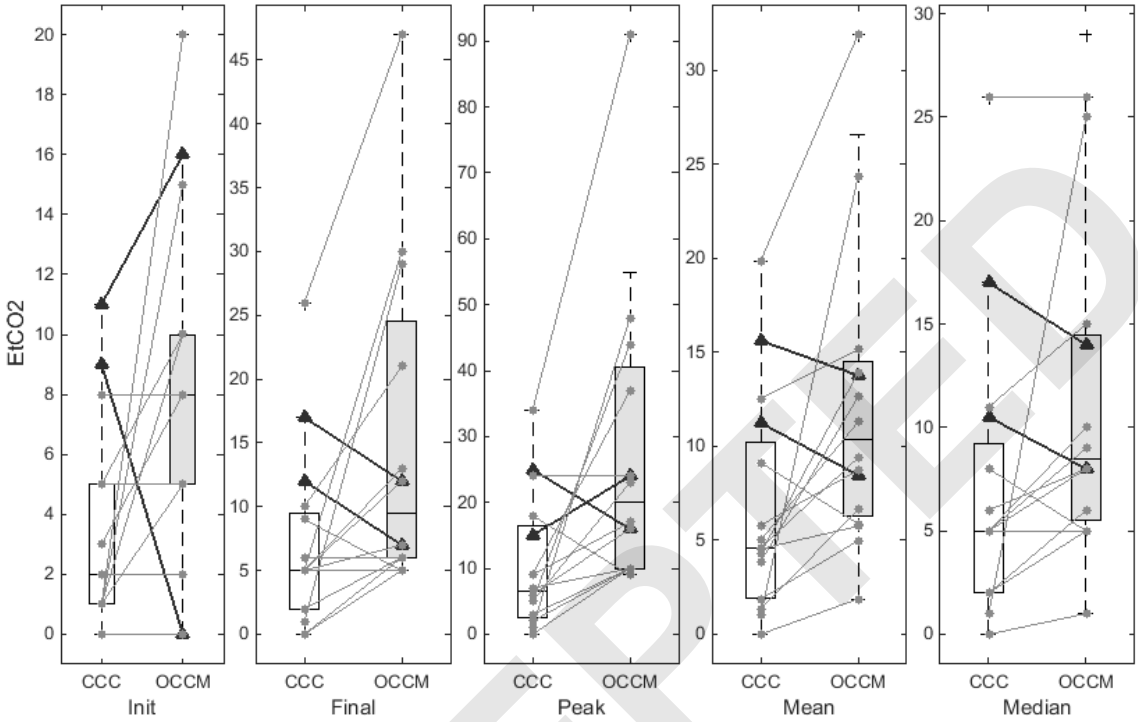

Figure 1a. Boxplots for the initial, final, peak, mean, and median EtCO2 before (CCC) and after (OCCM) opening chest, showing their minimum, 1st, 2nd, 3rd quartiles, maximum. Gray lines with round dots were penetrating trauma subgroup. Black lines with triangles were blunt trauma subgroup.

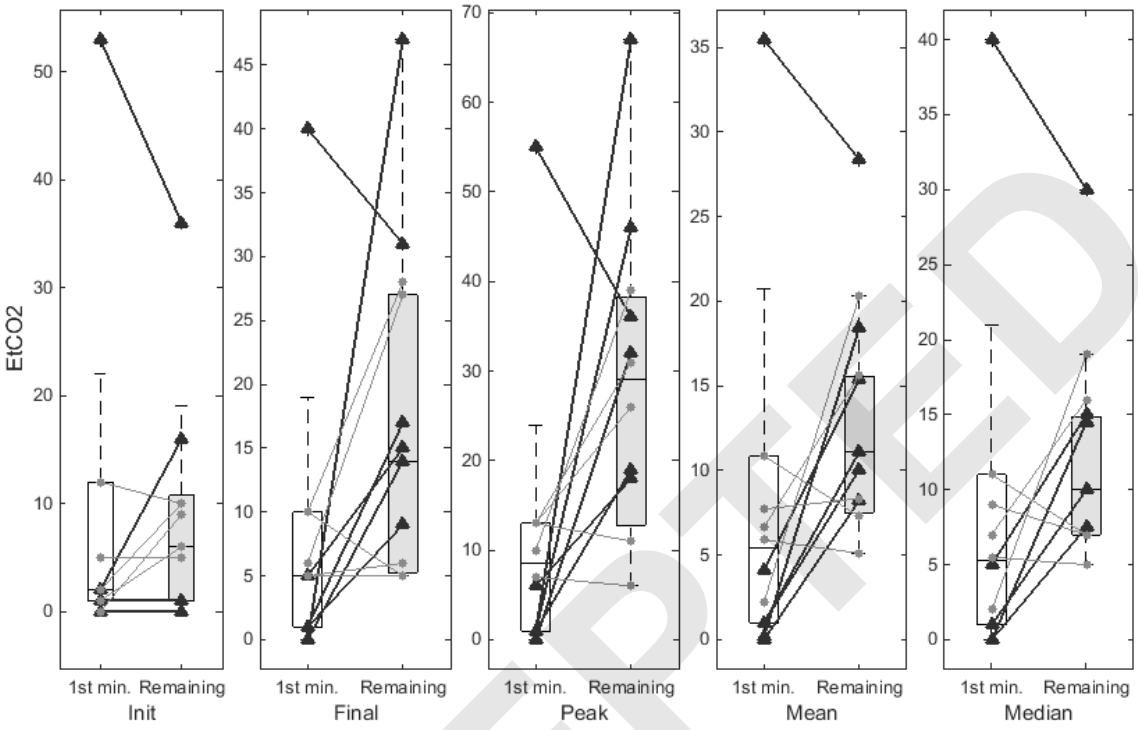

Figure 1b. Boxplots for the initial, final, peak, mean, and median EtCO2 in the first one minute and the remaining time in the CCC-only subgroup, showing their minimum, 1st, 2nd, 3rd quartiles, maximum. Gray lines with round dots were penetrating trauma subgroup. Black lines with triangles were blunt trauma subgroup.

Table 1. Patient Demographics for the Study Population

	CCC Only (n=17)	OCCM (n=16)	p-value
Gender (M/F)	15 / 2	16 / 0	0.49
Age (years) ± SD	42.5 ± 18.8	31.4 ± 12.7	0.08
Witnessed arrest (yes/no/unspecified)	9 / 8 / 0	7 / 8 / 1	0.54
Mortality (percent)	15 (88.2%)	16 (100%)	0.49
Mechanism of Injury			
Penetrating	8 (47.1%)	13 (81.3%)	0.05
Stab	1	3	
Gunshot Wound	7	10	
Blunt	8 (47.1%)	2 (12.5%)	0.04
Crush	1	0	
Fall	0	1	
Found Down	1	0	
Motor Vehicle Collision	4	0	
Pedestrian Struck	2	1	
Unknown	1 (5.8%)	1 (6.2%)	

CCC = closed chest compressions; OCCM = open-chest cardiac massage; SD = standard deviation

Table 2. ETCO2 Values for CCC-only and OCCM groups

	CCC Only* (n=17)			OCCM (n=16)			**CCC vs. OCCM**
	First Minute	Remaining Time	p-value	CCC†	OCCM	p-value	p-value
Initial (Mean± SD)	8.6 ± 14.4	8.6 ± 9.5	0.31	3.4 ± 3.4	7.9 ± 5.6	0.01	0.81
Final (mean± SD)	8.8 ± 10.6	17.0 ± 12.5	0.03	6.8 ± 7.0	14.9 ± 12.5	0.01	0.65
Peak (Mean± SD)	12.2 ± 14.5	29.0 ± 17.4	0.01	10.4 ± 10.4	27.8 ± 22.4	0.005	0.86
Mean (Mean ± SD)	8.4 ± 10.0	12.3 ± 6.4	0.01	6.8 ± 6.8	12.6 ± 8.4	0.09	0.94
Median (Mean ± SD)	8.8 ± 10.9	11.5 ± 6.7	0.04	6.8 ± 7.0	11.4 ± 8.3	0.05	0.97

*CCC-only data separated into first minute and the remainder of CCC period for comparison to OCCM

†Mean CCC period duration prior to OCCM = 66.9 ± 37.6 seconds

ETCO2=end-tidal carbon dioxide; CCC=closed chest compressions; OCCM=open chest cardiac massage; SD=standard deviation

p-value within CCC-only group = comparison between the first minute and the remaining time of CCC; p-value within OCCM group = comparison between the CCC period prior to OCCM and the OCCM period; p-value under CCC vs. OCCM = comparison of the CCC-only remaining time and the OCCM period.

Table 3. ETCO2 Values for CCC-only and OCCM for the Penetrating Trauma Subgroup

	CCC Only* (n=8)			OCCM (n=13)			**CCC vs. OCCM**
	First Minute	Remaining Time	p-value	CCC†	OCCM	p-value	p-value
Initial (Mean± SD)	5.8 ± 6.2	6.3 ± 3.5	0.14	2.4 ± 2.4	7.9 ± 5.3	0.005	0.48
Final (mean± SD)	8.7 ± 4.3	14.7 ± 11.8	0.25	5.9 ± 7.0	14.8 ± 13.2	0.09	0.99
Peak (Mean± SD)	13.0 ± 5.0	25.4 ± 17.4	0.12	9.5 ± 10.5	26.8 ± 23.5	0.002	0.89
Mean (Mean ± SD)	8.5 ± 5.2	10.8 ± 5.7	0.30	6.1 ± 6.9	11.7 ± 8.3	0.006	0.81
Median (Mean ± SD)	8.4 ± 4.8	10.0 ± 5.6	0.38	6.0 ± 7.0	10.2 ± 7.6	0.05	0.96

*CCC-only data separated into first minute and the remainder of CCC period for comparison to OCCM

†Mean CCC period duration prior to OCCM = 71.7 ± 39.1 seconds

ETCO2=end-tidal carbon dioxide; CCC=closed chest compressions; OCCM=open chest cardiac massage; SD=standard deviation

p-value within CCC-only group = comparison between the first minute and the remaining time of CCC; p-value within OCCM group = comparison between the CCC period prior to OCCM and the OCCM period; p-value under CCC vs. OCCM = comparison of the CCC-only remaining time and the OCCM period.

Table 4. ETCO2 Values for CCC-only and OCCM groups for the Blunt Trauma Subgroup

	CCC Only* (n=8)			OCCM (n=2)			**CCC vs. OCCM**
	First Minute	Remaining Time	p-value	CCC†	OCCM	p-value	p-value
Initial (Mean± SD)	11.4 ± 19.9	9.4 ± 13.3	0.91	10.0 ± 1.4	8.0 ± 11.3	0.83	0.89
Final (mean± SD)	9.4 ± 15.1	19.7 ± 14.5	0.11	14.5 ± 3.5	9.5 ± 3.5	0.15	0.38
Peak (Mean± SD)	12.6 ± 20.5	32.7 ± 19.3	0.08	20.0 ± 7.1	20.0 ± 5.7	0.55	0.41
Mean (Mean ± SD)	8.9 ± 13.8	14.0 ± 7.5	0.06	13.4 ± 3.1	11.1 ± 3.8	0.13	0.63
Median (Mean ± SD)	9.7 ± 15.3	13.1 ± 8.2	0.11	13.8 ± 4.6	11.0 ± 4.2	0.06	0.74

*CCC-only data separated into first minute and the remainder of CCC period for comparison to OCCM

†Mean CCC period duration prior to OCCM = 57.5 ± 21.9 seconds

ETCO2=end-tidal carbon dioxide; CCC=closed chest compressions; OCCM=open chest cardiac massage; SD=standard deviation

p-value within CCC-only group = comparison between the first minute and the remaining time of CCC; p-value within OCCM group = comparison between the CCC period prior to OCCM and the OCCM period; p-value under CCC vs. OCCM = comparison of the CCC-only remaining time and the OCCM period.