Auto-Transfusion Tourniquet (A-TT) reanimation of cardiac arrest patients – retrospective charts review

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Abstract

The outcome of out-of-hospital witnessed cardiac arrest undergoing CPR is disappointing with only very few of the patients actually being alive and discharged from the hospital with intact neurological status 30 days after the arrest. We hereby describe observations from charts review of 17 patients brought to Eisenhower Medical Center, Emergency Department, Rancho Mirage, CA in cardiac arrest. All patients were in terminal arrest, comatose and with dilated pupils. ***Methods***: The patients were treated in the ED with auto-transfusion tourniquets (A-TT) placed on one or both their legs (HemaShock®, Oneg HaKarmel Ltd. Haifa Israel) in addition to routine CPR protocol. The A-TT is an elastic ring that is tightly rolled up the limb to shift the blood from the limb to the core and prevent its re-entry to the limb. The reported information is based on retrospective chart review of the ED records of patients who were treated with A-TT per ED physician's clinical judgement. The ED charts were critically reviewed for history of arrest event, condition upon ED arrival, technical aspects of A-TT placement, cardiac rhythm prior to and after A-TT placement and patient disposition. ***Results***: All patients were brought to the ED while receiving standard CPR by paramedics 22 to 56 minutes after they collapsed. In seven of the patients the collapse was unwitnessed (2) or was due to non-cardiac etiology (GI bleed (1), traumatic pneumohemothorax (1), drug overdose (2) or when inadequate A-TT was used (1 patient)). The collapse event in the other 10 patients was witnessed and of presumed cardiac cause. Seven of the 10 patient (70%) showed ROSC within 1-5 minutes from the A-TT placement on one or both legs. Five (50%) of the patient had sustained spontaneous circulation long enough to be transferred to the ICU for brain preservation treatment and one of those (Pt #5) recovered and was discharged 30 days after the arrest in good neurological status. ***Discussion***: The A-TT technology is routinely used in orthopedic surgery for up to 120 minutes to create a bloodless surgical field. Physiologically it can be characterized as a "mechanical vasoconstrictor" that shifts 95% of the blood from each leg to the core. As such, it can auto-transfuse over 1000 cc of blood from the two legs to fill up the heart chambers (pre-load) and block the flow to the legs, thereby causing total resistance to flow into the legs, diverting the cardiac output to the essential organs and increasing diastolic pressure and thereby coronary perfusion pressure, which is key to ROSC during CPR. The rate of ROSC in this group of witnessed arrest (70%) is double that observed with pre-hospital witnessed arrest in a recent study when adrenaline was used ((36%), NEJM, July 18, 2018), despite the relatively long time from collapse to A-TT application. ***Conclusion***: These cases indicate that mechanical distal-to-proximal squeezing of the blood by the A-TT can improve the chances of ROSC in witnessed cardiac arrest when applied together with standard CPR. Clearly, additional large scale, pre-hospital studies should be done in order to verify and optimize the use of A-TT in cardiac arrest patients.

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Introduction:

The key to successful cardioversion and return of spontaneous circulation (ROSC) in cardiac arrest is

resumption of coronary blood flow (1,2) by maintaining sufficient coronary perfusion pressure (CPP) (12). Increasing chest compression cardiac output requires higher venous return and filling of the heart chambers (pre-load), while raising peripheral vascular resistance and diastolic pressure (after-load). Both can be achieved by shifting the blood from the legs (auto-transfusion) and blocking its return to the legs (tourniquet). The first mention of expelling blood from the limbs and restricting its re-entry during cardiac arrest was by Dr Woodward in 1952 (7). He described a case of a 4 years old child who had cardiac arrest during orthopedic surgery. Attempt to perform internal cardiac massage failed until Esmarch bandages were applied to the legs from toes to groin, leading to "more than doubling the size of the heart and spontaneous (temporary) return of heartbeat". This is now called in the anesthesia literature "Woodward Maneuver". The Woodward Maneuver was recreated in a recent porcine study by simultaneous placement of Esmarch bandages on all 4 limbs during induced VF cardiac arrest (6). The study showed significantly higher systolic and diastolic blood pressures, Coronary Perfusion Pressure (CPP), Cerebral Blood Flow (CBF), and end-tidal CO2 (ETCO2) during mechanical chest compression of the induced VF pigs treated with the limb binding vs. those who were not. Vasoactive drugs (e.g. adrenaline, vasopressin, norepinephrine etc.) are routinely used during CPR and in fact adrenaline is part of the formal AHA protocol for CPR. In a recent study by Perkins et Al (27) (NEJM July 18, 2018) of 8000 pre-hospital witnessed cardiac arrest patient it was found that the patients who received adrenaline as part of their treatment protocol were 3 times more likely to get to the hospital in ROSC relative to those who received placebo (36% vs. 12%). Unfortunately, the ultimate outcome 30 days after the incident was as disappointing in those who received adrenaline as in those who did not (~3% survival, ~2% in acceptable neurologically condition).

This manuscript reports the information collected from retrospective charts review of the first 17 cardiac arrest patients treated with A-TT in the emergency department of Eisenhour Medical Center in California. The treating physician used A-TT units, the like of which are routinely used in orthopedic surgery as an "exsanguination tourniquet" to shift blood from the limbs to the central circulation and block its re-entry to facilitate a bloodless surgical field (HemaClear®, Oneg HaKarmel Ltd. Tirat Carmel, Haifa, Israel). All patients were brought to the ED in terminal arrest and the A-TT was used as an exsanguination tourniquet in a heroic attempt to revive them following Woodward's initiative. This was not done as part of a clinical study hence the lack of a control group or IRB approval. In this retrospective review, 7 of the patients did not meet the inclusion criterion for "witnessed cardiac arrest" and 10 of them did. 7 of the latter 10 patients had ROSC shortly after the A-TT was placed. In 5 of them ROSC was sustained and they were transferred to the ICU for brain-preservation treatment, care of 4 of them was eventually withdrawn due to severe and irreversible brain damage, while one of these 10 patients successfully recovered to home discharge in good neurological condition.

Methods

Copies of the original ED charts that include doctors' and nurses' entries as well as lab reports were reviewed retrospectively reviewed by the author, who was not the treating physician, for the following items:

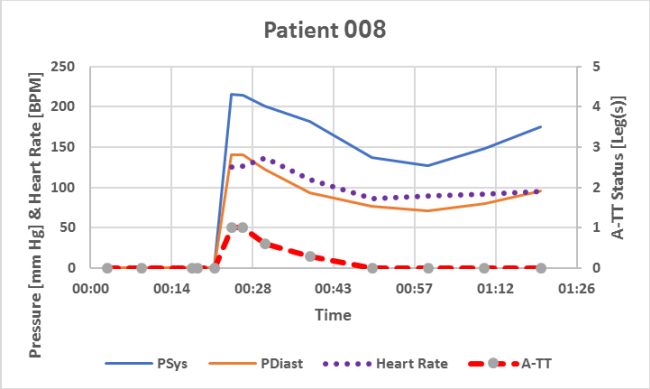
1. Clinical information and History
2. Patient status prior to A-TT placement
3. A-TT Placement timing
4. Patient status immediately after A-TT placement
5. Description of next level of care
6. Patient outcome and disposition
7. Technical comments
8. Discussion of A-TT use in this case and critique
9. Time-line

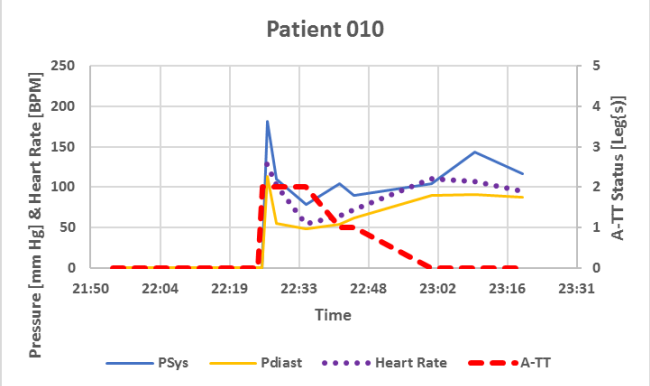
In those patients who had ROSC and measurable blood pressure for more than a few minutes, graphs of systolic and diastolic blood pressures and heart rate were drawn per the nurses' chart with respect to the timing of A-TT placement and removal. The detailed data from all 17 patient's chart are included in an appendix in an internet depository.

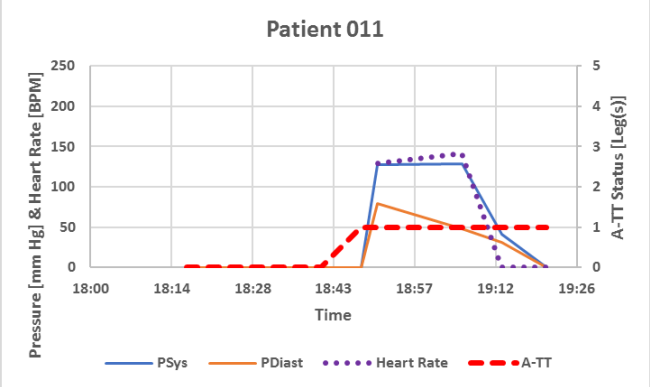
Cases and Results

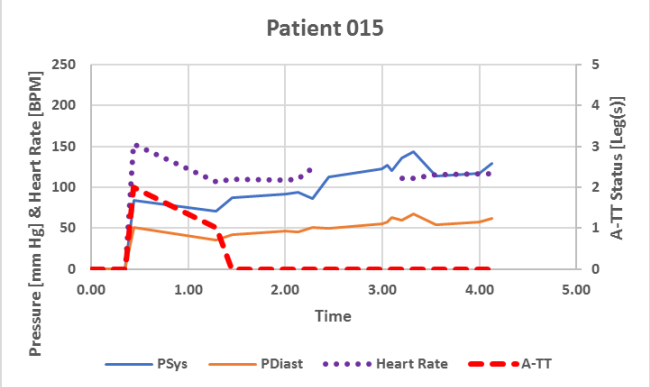
All patients were brought to the ED by ambulance staffed by paramedics, while receiving CPR per AHA protocol. All patients were in terminal arrest, had no measurable blood pressure or palpable pulse and were in coma with dilated, non-responsive pupils. Table 1 concisely lists the 10 patients meeting inclusion criteria, namely, patient treated with A-TT whose primary cause of collapse was witnessed cardiac arrest. Table 2 lists the 7 patients treated with A-TT (one with pediatric A-TT) who were treated with A-TT but did not meet inclusion criteria (unwitnessed cardiac arrest, evidently non-cardiac cause of collapse: overdose, poisoning, hypoglycemia, or hyperthermia). Placement of A-TT had no effect on the patients who

did not meet inclusion criteria and they were all









Figures 1-4

Timeline of blood pressure, heart rate and A-TT status in Patients 8, 10, 11, and 15. The charts start at the time of collapse. Note the coincidence of ROSC with the placement of A-TT in these patients. Patient 011 died in the ED, while the other patients had sustained circulation and were transferred for brain preservation treatment in the ICU.

pronounced dead in the ED. Among the 10 witnessed cardiac arrest patients 7 had ROSC right after A-TT was applied. In 3 of them ROSC was observed after A-TT was applied on only one leg, while in the other 4, A-TT was applied on both legs. In two of the ROSC patients the period of spontaneous circulation was short (5-6 minutes in one and 22 minutes in the other) and they were pronounced dead in the ED. The other five ROSC patients had sustained spontaneous circulation and were transferred to ICU with brain-preservation protocol. Care was terminated after 6, 8 and 12 days in three of these patients due to severe irreversible brain disfunction. Figures 1-5 show the blood pressure, heart rate and A-TT status time line of five of the ROSC patients. One of the sustained-circulation patients was transferred to another hospital and his final disposition is not known. One patient (#005) was discharged home in good neurological status after 30 days in the hospital. This case is described below in detail since the patient had an implanted defibrillator that recorded his EKG and defibrillation activity throughout the time he was being resuscitated.

Case #005 detailed description:

A 78 years old male collapsed in witnessed arrest in a restaurant. The patient had cardiac history of MI, chronic atrial fibrillation, cardiomyopathy, status-post triple bypass surgery, multiple coronary stents, aortic valve replacement, inferior vena cava filter and automatic implantable cardioverter-defibrillator AICD (26). Bystander CPR was started immediately and was followed by paramedics ACLS. The patient was brought to ED by paramedics approximately 33 minutes after collapsing with on-going CPR, multiple IV doses of epinephrine, IV Amiodarone, and multiple AICD shocks and external defibrillation attempts. Upon arrival the patient was in VF, comatose, with dilated pupils. Auto-transfusion tourniquets (A-TT) were placed on both legs immediately upon arrival to the ED. The patient was then defibrillated successfully with achievement of ROSC within 4 minutes of ED arrival, initially with extreme tachycardia and very high blood pressure followed within 8 minutes by sustained and effective cardiac rhythm at 89±14 bpm with blood pressure of 110±12/75±8 mm Hg. At this point gradual removal of the A-TT was started, while monitoring closely the patient's hemodynamic status. Figure 5 shows the timeline of the patient's blood pressure and the A-TT placement status until the patient was transferred to the ICU. The numbers in Figure 5 correspond to the timing of the AICD records shown in Figures 6 and 7.

The AICD (Medtronic Protecta XT CRT-D) record showed persistent VT/VF pattern at 150 to 273 waveforms per minute (Figure 6). The AICD discharged 10 times prior to A-TT placement with no conversion or

pacing capture (Records 26 and 45 in Figure 6) and 4 additional times after A-TT placement with transient paced capture as seen in records 67 and 80. In addition, external cardioversion shocks at 200J were given multiple times before ED arrival with no conversion and additional 4 external shocks were given after the A-TTs were applied as shown in record 95, this time with transient conversion; the first ROSC was 4 minutes after A-TT placement, eventually leading to steady sustained ROSC 12 minutes after A-TT placement.

Hypothermia brain preservation treatment was started within 33 minutes of ROSC. A-TT removal started 44 minutes after they were placed. First A-TT was rolled down to knee level, and then removed followed by the second one being rolled to knee level and then to ankle and then removed completely. The removal process took 37 minutes and total A-TT time was 81 minutes. When the first A-TT was rolled down from the thigh to the knee, supplemental bolus of IV NS was given to avoid sharp drop in blood pressure. The patient was maintained in brain-ischemia protection protocol for 7 days and gradually regained consciousness with meaningful communication and cognition over the subsequent week. Rehabilitation was started and after 30 days in the hospital the patient was discharged and flown to his home-town for continued rehabilitation in good neurological status.

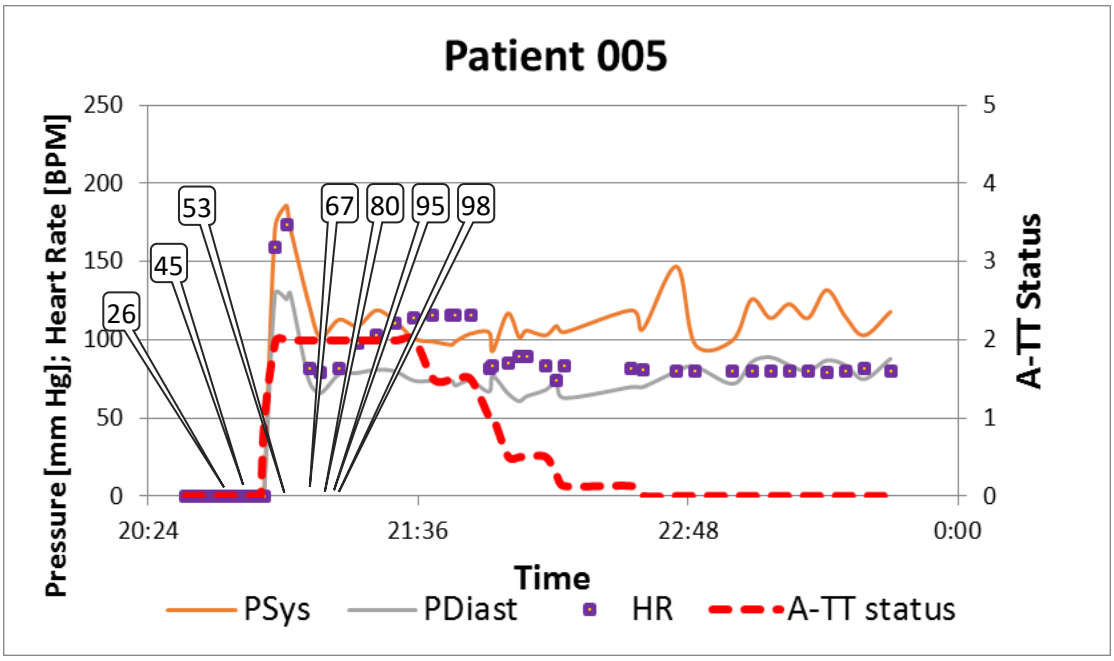


Figure 5 Timeline of Patient 005 blood pressure Heart Rate and the A-TT status from the time of collapse until the patient was transferred to the ICU. The numbers indicate the timing of the AICD records shown in Figure 6 and 7.

Figure 6 AICD records from before the application of the A-TT. The arrows indicate shocks applied by the AICD. Note the lack of conversion or capture.

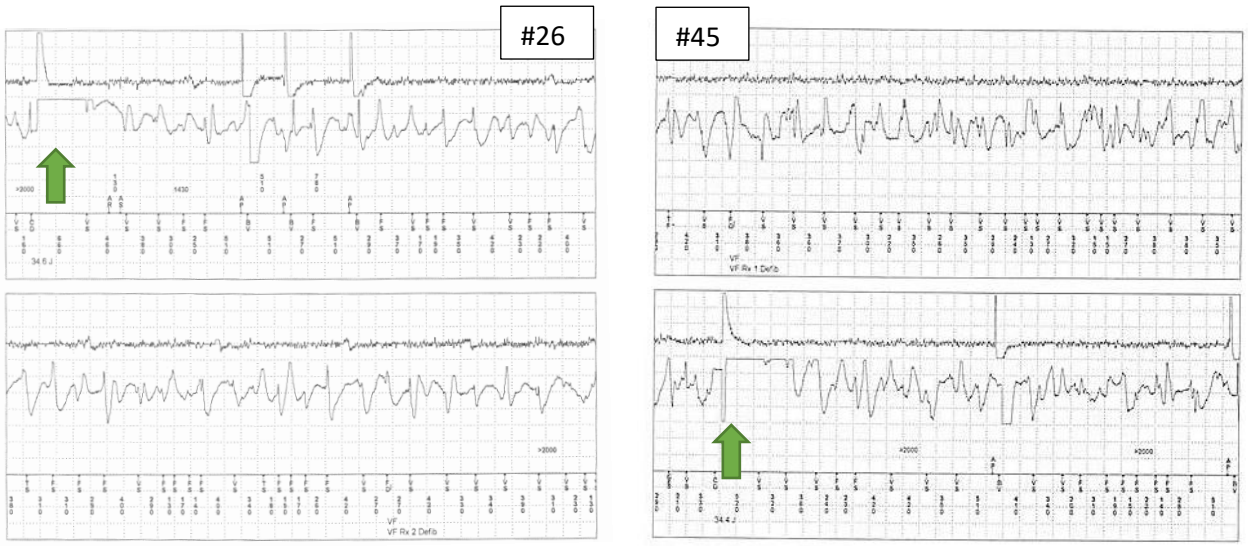
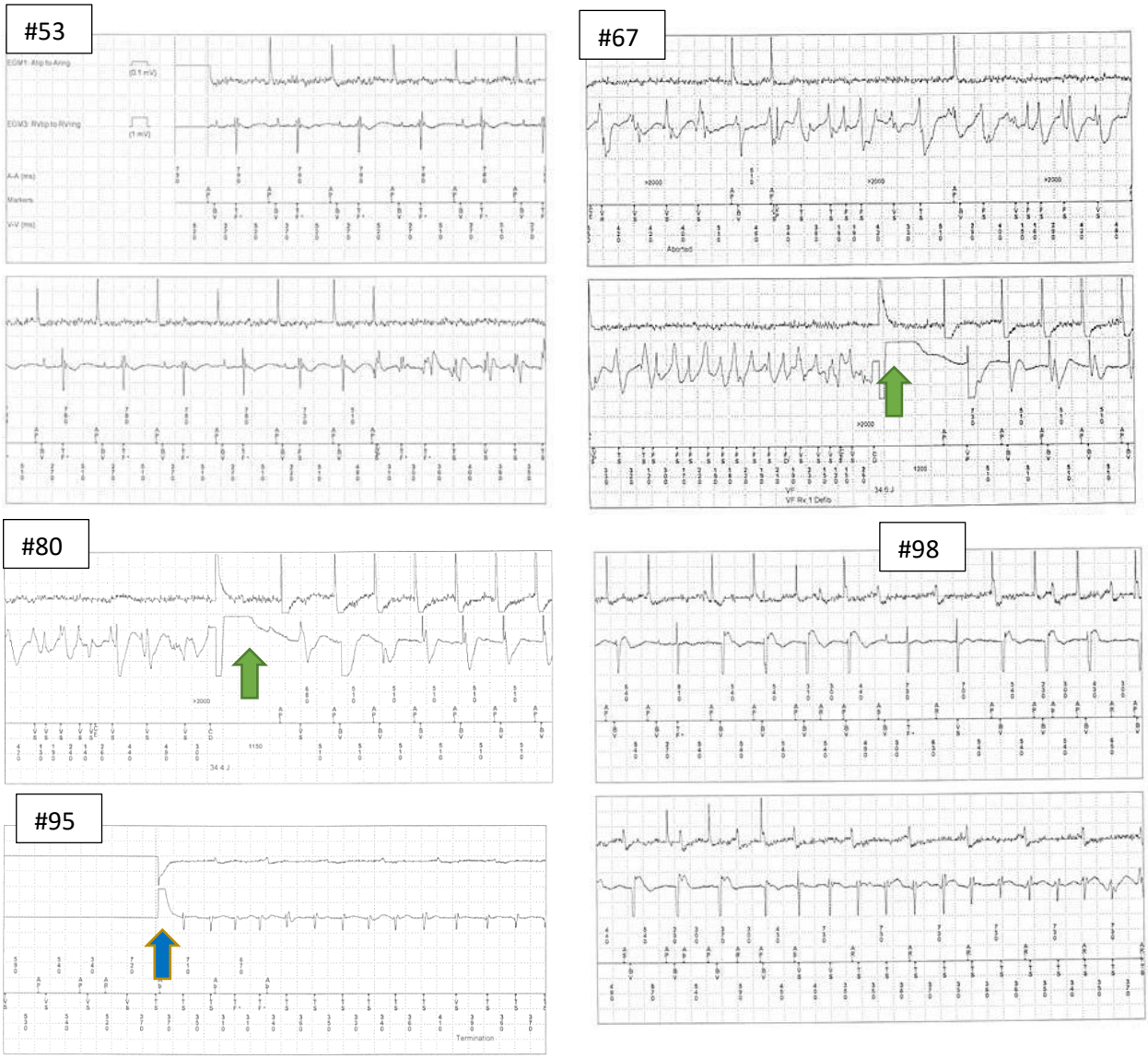


Figure 7 AICD Records from after the A- TT were applied. The numbers indicate the times shown in Figure 5. Brown arrows show the shocks of the AICD. Blue arrow indicates an external defibrillator shock. Note the return to regular (paced and un-paced) electrical activity, which was initially transient and from record 98 on was sustained.



Discussion

Pathophysiological considerations

The exsanguination tourniquet used as a last resort measure in these cases is routinely used in orthopedic limb surgery to effect bloodless surgical field (5). It evacuates all the blood in the limb except the blood inside the bone marrow and blocks arterial flow into the limb. As such it is an ultimate mechanical vasoconstrictor, exclusively focused to the limbs. It has been shown that about 500 cc of blood can be shifted from each leg of a normovolemic adult to the central circulation (3, 4). It is conceivable that during cardiac arrest, when the sympathetic nervous system shuts down and vasodilatation occurs, the volume of blood accumulating in each leg may be even larger than normal. As such, applying the A-TT to a leg can quickly (i.e. in less than 30 seconds) displace more than a pint of whole blood with intact oxygen carrying capacity from each leg. The blood then feeds into the heart and central circulation to enhance venous return, increase end-diastolic volume of the heart chambers and expand stressed volume of the central circulation. A recent porcine study was done where surrogates of the A-TT were used – simultaneous placement of Esmarch bandages on all 4 limbs of induced-VF pigs (6). The study showed significantly higher systolic, diastolic and Coronary Perfusion Pressures (CPP), Cerebral Blood Flow (CBF), and ETCO2 during mechanical chest compression in the pigs treated with the limb-binding vs. those who were not. This study recreated the Woodward Maneuver where Esmarch bandages were used to restore heart volume in a child in cardiac arrest during open chest cardiac massage (7).

The effect of the A-TT can be viewed as a "mechanical vasoconstrictor" whose effect is focused and limited to the extremities. Chemical vasoconstrictors (8-12) are routinely used during CPR to achieve a similar effect. However, the distribution of the chemical vasoconstrictors is throughout the body and depends on the circulation time, which may actually be longer than the half-life of drugs such as adrenaline. Moreover, studies have repeatedly showed that chemical vasoconstrictors reduce cerebral blood flow and neurological outcome. It is important to note that the shifting of blood to the core and increasing of pre-load is not the only effect of the A-TT. Its use also prevents blood from feeding into the treated limbs. This promptly increases peripheral resistance, and redirects the less than optimal cardiac output achieved with CPR to the essential organs. In fact, in the J Resuscitation porcine study (6) it was shown that the diastolic pressure was 14-17 mm Hg higher in the limb-binding group and CPP was 10-14 mmHg higher, despite the relatively small limbs of the pig.

Experience with the use of tourniquets in orthopedic surgery (5) and in prevention of bleeding in penetrating limb trauma facilitated setting the time limit of uninterrupted application of the A-TT to 120 minutes, but it is prudent to start removing the device as soon as possible after ROSC was achieved and do so gradually while monitoring hemodynamic parameters.

History

The first mention of expelling blood from the limbs and restricting its re-entry during cardiac arrest was by Woodward in 1952 (7). He described a case of a 4 years old child who had cardiac arrest during orthopedic surgery. Attempt to perform internal cardiac massage failed until Esmarch bandages were applied to the legs from toes to groin, leading to "more than doubling the size of the heart and spontaneous (temporary) return of heartbeat". This is now called by some "Woodward Maneuver", but it should not be confused with just elevating the legs.

Studies done in the 1980's with MAST anti-shock trousers and Abdominal Binding during CPR did not have an overall beneficial effect on the measured parameters nor on the outcome (13-19). The general perception is that their use restricted respiration by compressing the lower ribs and upwards shifting of the abdominal content. The logistics and time delay for applying the MAST were a likely additional factor in their not becoming a standard of care.

The logic of trying to shift blood from the widely dilated periphery to the core prompted the invention of Compression-Decompression CPR devices (20) as well as the inspiratory impedance threshold valve (21-23). Both methods attempt to maintain negative intra thoracic pressure in order to induce a pressure gradient along the vena cava(s) in order to increase venous return and end diastolic volume. The debatable success of these methods in improving ultimate CPR outcome is most likely due to difficulty to suck fluids through soft-wall collapsible blood vessels (i.e. "Starling resistor").

Vasopressors such as epinephrine, norepinephrine and vasopressin are being used routinely in CPR. Controversies around the use of these drugs continue (24-25), but the current standard of care and AHA ACLS directives are to use them since their use increases the rate of ROSC 2-3 fold. It should be noted that with the reduced cardiac output during CPR circulation time may take as much as 4 minutes for a dose of Epinephrine to reach from a peripheral vein injection site to the arterial and venous vessels of the limbs but less so to the heart, brain gut, kidneys and liver. This may exceed the half-life of adrenaline in the body, so that their constricting effect is greater on the brain and the central organs than on the periphery. In addition, the excitatory effect of these drugs on the heart contractility and its energy consumption are of interest beyond the scope of this discussion (24-25). However, we noted exceedingly high blood pressure immediately after ROSC in two of the patients (Figures 2 and 5) and the initial undulation between effective and ineffective contractions (Figure 7) and we speculate that the multiple doses of Adrenaline given to these patients contributed to these adverse effects.

Summary of Observations

The information gained by systematically reviewing the charts of the 17 patients treated with the A-TT may be summarized as follows:

1. No A-TT effect was seen when un-witnessed arrest with unknown down time
2. No A-TT effect was seen when arrest is due to overdose
3. No effect was seen when time from arrest to A-TT placement was very long
4. A-TT had no effect on a patient with severe hypoglycemia (36 mg/dl) due to Insulin (Lente) OD
5. A-TT had no effect on a patient with suspected pulmonary embolus
6. A-TT cannot be placed on a leg that has an IO port in place. As soon as possible, peripheral IV should be started and IO removed so that second A-TT placement is possible if needed.

Comments and contraindications

The fact that ROSC occurred within a short time from applying the A-TT on patient 005 together with the fortuitous availability of the AICD (26) recording that showed absolutely no effect of the cardioversion shocks prior to A-TT placement and sustained conversion after it was placed is compelling.

The A-TT should not be placed on a leg that has known DVT in it. It is not contraindicated to place it on the other leg or on the arms. Only the use of the A-TT in cardiac arrest is discussed here. In patients with severe shock other instructions apply to the A-TT application which include gradual placement, so as to avoid raising blood pressure too much. The removal of the device in all cases should always be done by rolling it down gradually and in steps while monitoring the patient's hemodynamic status.

In all of the cases described here the A-TT was placed in the ED with a relatively long delay from collapse to placement. It is conceivable that doing so earlier, by field care-givers could improve the outcome. This needs to be evaluated by a dedicated pre-hospital clinical study. As always, the successful care of the near-death patient is the result of combining many elements. As with the AED, some 15 years ago, the addition of the A-TT as an added link in this chain should be critically studied, optimized and if proven useful, meticulously implemented.

References

1. Ditchey RV, Winkler JV, Rhodes CA. Relative lack of coronary blood flow during closed-chest resuscitation in dogs. *Circulation*. 1982; 66: 297-302.
2. Kette F, Weil MH, von Planta M, et al. Buffer agents do not reverse intramyocardial acidosis during cardiac resuscitation. *Circulation*. 1990; 81: 1660-66.
3. Gavriely O, Nave T, Sivan S, Shabtai-Musih Y, Gavriely N. Auto Transfusion and Blood Pressure Elevation by Elastic Leg Compression in Normal Subjects. Rappaport, Technion; Haifa: Israel Institute of Technology; 2000. [Accessed January 7, 2019]. Available at: http://www.emergencyeed.com/uploads/1/9/1/4/19141635/study_-_auto_transfusion_mk0036200_compressed_2.pdf
4. www.emergencyeed.com
5. Demirkale I, Tecimel O, Sesen H, Kilicarslan K, Altay M, and Dogan M. Nondrainage decreases blood transfusion need and infection rate in bilateral total knee arthroplasty. *J Arthroplasty*. 2014; 29:993-7.
6. Yang Z, et al. A tourniquet assisted cardiopulmonary resuscitation augments myocardial perfusion in a porcine model of cardiac arrest. *Resuscitation* (in press, 2014).
7. Woodward WW. Treatment of cardiac arrest: filling the heart by expelling blood from the limbs. *Lancet* 1952; 1:82.
8. Pearson JW, Redding JS. Influence of peripheral vascular tone on cardiac resuscitation. *Anesth Analg* 1965; 44: 746-52.
9. Lindner KH, Prengel AW, Brinkmann A, et al. Vasopressin administration in refractory cardiac arrest. *Ann Intern Med* 1996; 124: 1061-4.
10. Herlitz J, Ekstrom L, Wennerblom B, et al. Adrenaline in out-of-hospital ventricular fibrillation. Does it make any difference? *Resuscitation* 1995; 29: 195-201.
11. Hayakawa et al.: Effects of epinephrine administration in out-of-hospital cardiac arrest based on a propensity analysis. *Journal of Intensive Care* 2013 1:12.
12. Kern KB, Ewy GA, Voorhees WD, et al. Myocardial perfusion pressure: a predictor of 24 h survival during prolonged cardiac arrest in dogs. *Resuscitation* 1988; 16: 241-50.
13. Bircher N, Safar P, Stewart R. A comparison of standard, "MAST" augmented, and open-chest CPR in dogs. A preliminary investigation. *Crit Care Med.* 1980; 8:147-52.
14. Niemann JT, Rosborough JP, Criley JM. Continuous external counterpressure during closed-chest resuscitation: a critical appraisal of the military antishock trouser garment and abdominal binder. *Circulation.* 1986; 74: 102-7.
15. Babbs CF. The evolution of abdominal compression in cardiopulmonary resuscitation. *Acad Emerg Med* 1994; 1: 469-77.
16. Chandra N, Snyder LD, Weisfeldt ML. Abdominal binding during cardiopulmonary resuscitation in man. *JAMA* 1981; 246: 351-3.
17. Redding JS. Abdominal compression in cardiopulmonary resuscitation. *Anesth Analg.* 1971; 50: 668-75.
18. Babbs CF. Interposed abdominal compression CPR: a comprehensive evidence based review. *Resuscitation.* 2003; 59:71-82.
19. Ralston SH, Babbs CF, Niebauer MJ. Cardiopulmonary resuscitation with interposed abdominal compression in dogs. *Anesth Analg.* 1982; 61: 645-51.
20. Aufderheide TP, Frascone RJ, Wayne MA et al. Standard cardiopulmonary resuscitation versus active compression-decompression cardiopulmonary resuscitation with augmentation of negative intrathoracic pressure for out-of-hospital cardiac arrest: a randomized trial. *Lancet* 2011; 377: 301–11.
21. Lurie KG, Mulligan KA, McKnite S, et al. Optimizing standard cardiopulmonary resuscitation with an inspiratory impedance threshold valve. *Chest*. 1998; 113: 1084-90.
22. Lurie KG, Voelckel WG, Plaisance P, et al. Use of an inspiratory impedance threshold valve during cardiopulmonary resuscitation: a progress report. *Resuscitation*. 2000; 44: 219-30.
23. Lurie KG, Zielinski T, McKnite S, et al. Use of an inspiratory impedance threshold valve improves neurologically intact survival in a porcine model of ventricular fibrillation. *Circulation*. 2002; 105: 124-9.
24. Tang W, Weil MH, Sun S, et al. Epinephrine increases the severity of postresuscitation myocardial dysfunction. *Circulation.* 1995; 92: 3089-93.
25. Marwick TH, Case C, Siskind V, et al. Adverse effect of early high-dose adrenaline on outcome of ventricular fibrillation. *Lancet* 1988; 2: 66-8.
26. Volker Kühlkamp. Initial experience with an implantable cardioverter-defibrillator incorporating cardiac resynchronization therapy *J Am Coll Cardiol*. 2002;39(5):790-797
27. Perkins, GD et Al. A Randomized Trial of Epinephrine in Out-of-Hospital Cardiac Arrest. N Engl J Med 2018; 379:711-721.

Table 1. Patients meeting inclusion criteria (witnessed cardiac arrest, no overdose, no hypoglycemia, correct size product)

Pt. #	Duration prior to HS	Immediate HS effect	Outcome	BP graph
002	43 min	Transient ROSC for 5-6 minutes	Deceased	no
005	34 min	ROSC within 4 minutes of A-TT	Discharged in good neurological status 30 days after admission	yes
006	>30 minutes	ROSC within 5 minutes of A-TT on ONE leg	ICU under brain preservation protocol via air evacuation.	No (air evac)
008	22 minutes	Spontaneous ROSC within 1 minute of A-TT placement on ONE leg.	Deceased after 12 days in ICU (treatment withdrawn) with brain protocol	Yes
009	45 minutes	No effect	Deceased in ED	No
010	56 minutes	ROSC upon A-TT placement	ICU with brain protocol. Treatment withdrawn after 6 days	Yes
011	30-35 minutes	Transient ROSC after one A-TT placed, lasting 22 minutes	Deceased in ED	Yes
013	48 minutes	No effect	Deceased in ED	No
014	37 minutes	No effect	Deceased in ED	No
015	36 minutes	Immediate ROSC	ICU with brain protocol; care withdrawn after 8 days	Yes

Table 2. Patients **not** meeting inclusion criteria

Pt #	Time from collapse to ED	Reason for not being included	Outcome
001	>30 min	Pediatric models were applied to knee level	Deceased
003	?	Unknown down time; OD, multiple substance intoxication	Deceased
004	>25 min	Drug OD and severe hypoglycemia	Deceased
007	38 min	Collapse after double dose of Insulin. Severe hypoglycemia	Deceased
012	45 minutes	Pneumothorax/ Hemothorax	Deceased
016	Unknown duration	Hyperthermia at 110F, unwitnessed collapse; no HS effect	Deceased
017	Arrested in ED	GI bleed, hemorrhagic shock	Deceased