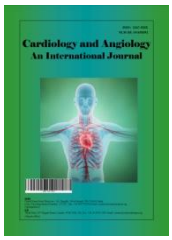

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Time to Resuscitate Cardiopulmonary Resuscitation: The 3R/CPR Refill-Recoil-Rebound

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Author's contribution

The sole author designed, analysed, interpreted and prepared the manuscript.

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ABSTRACT

Introduction: Sudden cardiac arrest (SCA) remains a major healthcare issue worldwide with gloomy outcomes due to poor perfusion of cardiopulmonary resuscitation (CPR), deemed unsuitable for hemostatic conditions, cardiotorsal anatomy, electrophysiology, and thoracic biomechanics. Alternatively, we propose a new management, implementing rational exploitation of the stagnant blood masses: *manually* with a novel technique of cardiac massage and *mechanically* with a circulatory flow restoration (CFR) device.
Methods: Simulated chest compressions were performed through the 5th intercostal space in professional Lifeguards volunteers, placed in the left lateral decubitus position with raised legs and abdominal compression.
Results: Bypassing the sternal barrier, *refilling* the heart, and then compressing the chest with a *recoil-rebound* maneuver (3R / CPR) can significantly promote return of spontaneous circulation (ROSC). The effectiveness of the CFR device versus CPR has previously been demonstrated in the literature.
Conclusion: Unlike current CPR, the 3R/CPR adapts human morphology and provides adequate myocardial perfusion promoting ROSC safely, under all circumstances. Preclinical computational models can confirm the effectiveness of the technique.

Keywords: Sudden cardiac arrest; cardiopulmonary resuscitation (CPR); circulatory flow resuscitation (CFR); 3R/CPR; thoracic biomechanics.

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1. INTRODUCTION

"Science is nothing but trained and organized common sense" Thomas Huxley [1].

Clinically, sudden cardiac arrest (SCA) can be identified by the abrupt discontinuity of organs perfusion following sudden *asystole* of the systemic ventricle, whether fibrillated due to *physiopathological* events [2-4], *pathophysiological* cardiac-extracardiac disorders [5,6], or intentionally *induced* knocked-out [7,8], In-hospitals (IHCA) or Out-of-hospitals (OHCA) [9].

Despite imprecise statistics, more than 1,250,000 OHCAs occur each year in North America and Europe, most often due to cardiac arrhythmia in victims with unknown history of cardiovascular diseases (CVD) [10-13].

Conceivably, current cardiopulmonary resuscitation (CPR) may combine *four* therapeutic modalities, namely: mid-sternal chest compressions, whether manually or mechanically, mouth-to-mouth ventilation, direct current (DC) shocks and invasive-CPR which includes injection of epinephrine, mechanical ventilation, extracorporeal membrane oxygenation (ECMO) known as E-CPR, implantable cardioverter defibrillators (IDC) and direct cardiac massage with cardiopulmonary bypass (CPB) under certain circumstances [14-21].

Despite progress and medical advances, the therapeutic impacts of CPR remain quite poor since the 30-day survival rate of postarrest victims with free brain damage is around 2% [22,23]. Most postarrest survivors succumb shortly after return of spontaneous circulation (ROSC), because of multiple organ failure due to inadequate organ perfusion during CPR [24-26].

On the other hand, induced cardiac arrest has become a safe procedure in open-heart surgery since in nearly 100% of cases the heart defibrillates after being knocked out for a significant length of time. This makes CPR one of the most controversial therapies in modern medical history that requires an entire overhaul of the concept through extensive scientific research.

Previously, we have demonstrated the benefits of prioritizing immediate restoration of circulatory flow dynamics over exhorting return of heartbeat with a noninvasive low-pressure extracorporeal pulsatile device, applicable in refractory OHCA and postarrest [27].

The goal of this study is to present a new technique of chest compressions to be used in the early onsets of cardiac arrest, adaptable to the hemostatic condition and thoracic biomechanics, promoting potential improvements in current CPR outcomes. A safer, less traumatic and an effective procedure that a rescuer can use with less effort, both outside and inside hospital environments.

1.1 Insufficiency of Current CPR

Foremost, it worth remembering that CPR was randomly adopted in the early 1960s following the experiments of a pioneering electrical Engineer while proving the concept of external defibrillators in canine models [28]. However, morphologically, as shown in Fig. 1, dogs are sorely different from humans, which compromises the clinical application of CPR. For example, because of a nearly triangular thoracic cage with a prominent sternal protrusion, chest compressions should be performed on the left chest while the dog is placed in a right lateral recumbency. Likewise, the anterolateral placement of the AED pads is more convenient for delivering effective DC shocks in dogs. Besides, unlike humans, dogs have a well-developed coronary network promoting frequent ROSCs.

Such clinical discrepancy of CPR raises a therapeutic dilemma that needs to be meticulously analyzed and resolved. As illustrated in Schema1, *four* therapeutic modalities of CPR intended to manage throughout *four* phases of cardiac arrest, collide with *four* concomitant pathophysiological barriers that must be overcome.

As, the primary goal of cardiac arrest management is assumed to be rapid ROSC while ensuring adequate perfusion of vital organs during the procedure. In other words, we must create an action potential at the conducting system, while hypothetically compressing the left ventricle (LV) to deliver adequate stroke volume through the aorta, which is practically impossible with current CPR for several reasons.

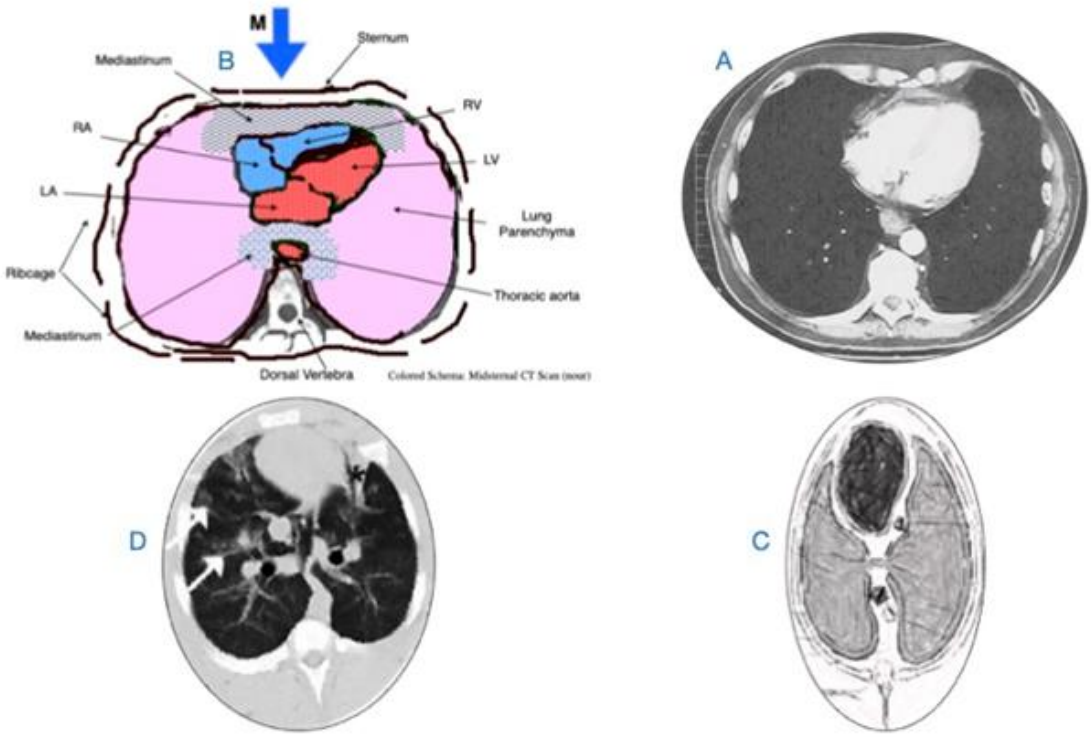
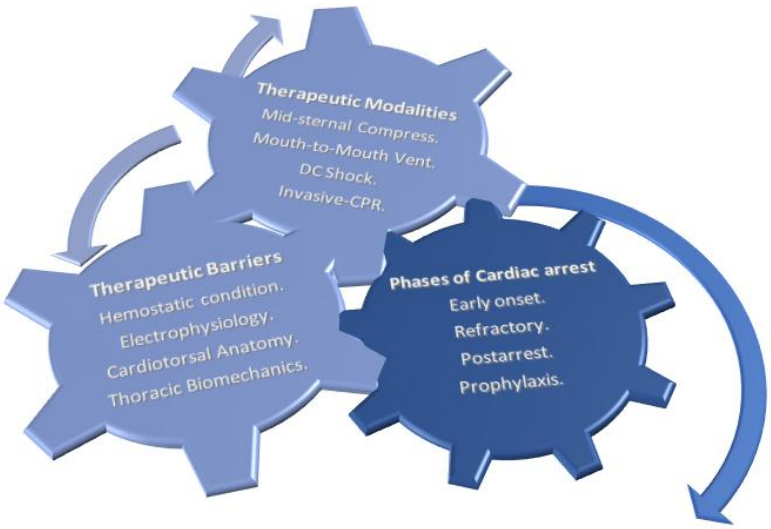


Fig. 1. Morphological discrepancy between species

- **Upper right panel (A): Midsternal chest CT scan showing the cardiotorsal anatomy of the shell-shaped cylindrical thoracic cage of a living adult person. Upper left panel (B), a colored schema of (A), showing: Mediastinum (dashed color); Right atrioventricular cavity (blue color); Left atrioventricular cavity (red color); Pulmonary parenchyma (pink color); descending thoracic aorta (red color); M (blue arrow): simulated CPR chest compressions**
- **Lower panel: Midsternal CT scans showing the variation in cardiotorsal anatomy between pig (left panel; D) and canine (right panel; C)**



Schema 1. Quaternary therapeutic dilemma of CPR (nour)

First, as is known, a heartbeat starts from within the heart with the action potential creating the polarization-depolarization of pacemaker cells, mainly at the Sinoatrial Node (SAN) inside the right atrium (RA), then spreading through the conducting system inside the intracardiac walls and septum [29]. *Electrophysiological* processing depends primarily on blood flow dynamics that start and control heartbeats from the 21st day of gestation, biochemically with the combinations of neurohumoral factors, and mechanically via the pulsatile impacts of shear stress and wall stress. The superiority of blood flow dynamics in controlling heartbeats over the autonomic nervous system is demonstrated with the denervated hearts transplant patients [30]. Likewise, serious arrhythmias can occur due to loss of RA wall stress, as in post-Mustard operations [31].

Second, due to the *hemostatic* state, the adult heart which roughly contains ≤ 400 mL of blood volume (BV), becomes nearly empty within 30 seconds after cardiac arrest, as part of the intracardiac blood moves backward–forward through the low-pressure valveless vena cavae and the pulmonary artery [32]. Consequently, the left-heart side which normally contains ≤10% of BV becomes almost empty with an aortic pressure (AP) = 0 mm Hg, which increases stagnant venous capacitance as well as venous pressure from ≤ 0mm Hg to ≥20 mm Hg.

Pathophysiological redistributions of stagnant BV that will *hemorheologically* disturb the flow dynamics-electrophysiological processes interdependency, which severely affects ROSC.

Third, *anatomically*, as shown in Fig. 1, several centimeters separate the sternum from the free wall of the right ventricle (RV), which is separated from the left ventricle (LV) by the interventricular septum. Also, the heart becomes further distant from the sternum in postarrest, pushed backward by the mediastinum once the victim the victim is placed in the supine position during CPR, as simulated in Fig. 2. A well-known *cardiotorsal* gravitational effect usually employed by cardiac surgeons accustomed to turning off the ventilator during a sternotomy, even while the heart is still beating.

Finally, we must take into account the *Biomechanics* of the cylindrical shell-shape thoracic cage, in particular the ribs' orientations and axis of their movements, controlled by the sternocostal, costochondral, costovertebral, and costotransverse joints [33].

Fig. 2. Simulation of current CPR mid-sternal chest compressions

1.2 Drawbacks of Current CPR

"The heart is anchored in the body by the great vessels" Dr. Claude Beck [34].

Despite the diversity of cardiac arrest victims, i.e., gender, age, etiology, etc., they nevertheless all share the same abovementioned pathophysiological barriers, namely hemorheological, electrophysiological, anatomical, biomechanical, causing severe complications of CPR [35].

For example, hemorheologically, to ensure adequate organs perfusions and promote ROSC, we need at least a stroke volume ≥ 140 mL to be delivered by the LV in adequate pulse pressure (syst. BP ≥80 mm Hg) and shear rates (≥ 40 / min) with a coronary perfusion pressure ≥ 15 mm Hg [36]. This may become an impossible task to achieve during cardiac arrest. Accordingly, mid-sternal chest compressions, whether manually or mechanically, are performed vigorously (e.g., ≥ 8-16 bar / in^2) in the hope of compressing the hard sternum, loose mediastinal and cardiopulmonary compartments to deliver stroke to deliver a stroke volume from the far distant near-empty LV, and in total disregard of thoracic biomechanics. Besides, the high frequency of chest compressions (≥100 bpm), restricts recoil of the thorax, venous return and does not adapt the capillary pressure cycle (40 bpm). In addition to the fact that the ribcage becomes more fragile vulnerable to trauma due to the loss of the intercostal muscles tone. As a result, most of CPR mechanical devices are contraindicated in pediatrics and less frequent in females due to their vulnerability to sternal fractures and mammary gland trauma.

Most DC shocks complications arise due to the anterolateral position of the AED electrodes, which is effective in dogs, causing them to deflect the electric field and then must be compensated with more powerful energy (≥ 300 joules). As a result, skin burns occur in nearly 25% of AED cases. In addition, other complications such as tachyarrhythmia, thromboembolic events and pulmonary edema have also been reported after strong DC shocks [37]. Furthermore, the prolonged depolarization period after strong DC shocks promotes myocardial necrosis and electroporation of the rare pacemaker cells which represent about 1% of cardiomyocytes [38].

While mouth-to-mouth ventilation during CPR provides insufficient tidal volume for victims [39], we believe that the entire concept of ventilation during cardiac arrest has no substantial benefit due to the lack of gas exchange at the alveolar level because of the hemostatic condition.

Apart from the employment of ECMO, the benefits of invasive-CPR procedures remain controversial due to the hemostatic condition. For example, attempts to improve cerebral perfusion with intravenous hypertonic saline and/or nitrates, did not change the significant numbers of brain damage in postarrest victims [40]. Yet, ECMO requires skilled squads guided by ultrasounds for its installation via empty flattened arteries, which makes its use in OHCA more difficult [41].

2. METHODS

2.1 Proposed Technique

We modeled the proposed method on clinical observations and outcomes of OHCA and IHCA as experienced by Lifeguards and Cardiac surgeons, respectively.

According to the Utstein style [42], lifeguards have the best results of OHCA. In addition to their rapid intervention, we believe that the Heimlich's maneuver [43], which is constantly practiced by Lifeguards for evacuating the aspired water, mobilizes massive amount of the stagnant hepato-splanchnic blood via the inferior vena cava. This directly stimulates the conducting system promoting ROSC, even without chest compressions in more than 70% of victims [44]. Therefore, we were assisted in this study by one of the most experienced Lifeguards' squads from Greece which has the highest drowning records in Europe [45].

In open-heart surgery, with advances in myocardial reperfusion procedures, ROSCs occur most often in more than 90% of patients without DC shock. Patients most often recover sinus rhythm once myocardial perfusion is established, even whether the heart has been denervated as in transplants or after sacrificing the sinus node artery as occasionally in arterial switching procedures.

Perhaps one of the most impressive demonstrations of ROSC in cardiac surgery is practiced by Sir Magdi Yacoub[*] who used to wean his patients from CPB by the abrupt clamping of venous lines, which instantly refills the RA creating a snap effect at the conducting system with an immediate defibrillation of the heart.

2.2 Procedure

Therefore, to create a similar intracardiac hemorheological effect promoting ROSC, while avoiding the pathophysiological barriers of cardiac arrest, we need to:

- Overrule the sternal barrier and bring the heart closer to the chest wall.
- Refill the near-empty heart.
- Recoil properly the chest wall and the mediastinal-parenchymal structures surrounding the heart within the rules of thoracic biomechanics.
- Induce a sudden *rebound* effect with the intracardiac blood mass to create a snap effect at the conducting system of the heart (e.g., likewise Sir Yacoub's method).

2.3 Steps

1. Avoid panic, seek help, call 911.
2. As simulated in Fig. 3A, place the victim on the left recovery position and clear the airways. Loosen all tight garments (e.g., belt, bras). Place the right palm at the level of the victim's 5th intercostal space and check if there is still a palpable heartbeat as demonstrated in Fig. 3B.

In case of cardiac arrest:

3. *Refill maneuver*: Raise the victim's hip and legs to be kept in a Trendelenburg position, e.g., with pillows. Victims can be easily lifted from their waist as shown in Fig. 4A. Gently wrap the victim's trunk

[*] *Attending Sir Magdi Yacoub's operative sessions at Harefield & Royal Brompton Hospitals (UK).*

(e.g., with a large towel), to compress and mobilize the subdiaphragmatic hepatosplanchnic blood volumes upwards.

4. As demonstrated in Fig. 4B, adjust the victim's position with the right hand and forearm to push his/her chest backward while pushing his/her flank forward with the right thigh.
5. *Recoil maneuver*: as simulated in Fig. 4C, with both hands compress and push the victim's chest backward-upward at the 5th intercostal space, while blocking the victim's back with both thighs and leaning forward compressing the right chest wall with both forearms, to induce a nearly circumferential thoracic recoil.
6. Maintain the *recoil* maneuver for few seconds.
7. *Rebound maneuver*: as simulated in Fig. 4D, the sudden release of both hands and all chest compression sites will induce a water hammer-like mechanism inside the heart with a snap effect on the conducting system.
8. *DC Shock*: after 2 minutes of *recoil-rebound* maneuvers without ROSC, a DC shock of 100J can be delivered after placing the AED pads in the anteroposterior position, e.g., on the sternum and between the scapulae, as simulated in Fig. 5.
9. If unsuccessful, repeat the *recoil-rebound* maneuvers for another 2 minutes then try a 150J DC shock, and then with a final third one within an interval of 3-4min.

- In case of ROSC, keep the victim in the left recovery position while maintaining a slight abdominal compression until transferred to Cardiac Centers.
- In case of non-ROSC, continue the *recoil-rebound* maneuvers until transferred to Cardiac Centers.
- In neonates and infants, as far as we can perform a proper circumferential chest compression we may continue with the present technique as demonstrated in Fig. 6.

Precaution: The *rebound* maneuver should not be attempted in living person as it may provoke ventricular fibrillation like Commotio Cordis syndrome. Therefore, in our study, we used to rebound water pockets, lower intercostal spaces, and abdomen as in appendicitis test.

Fig. 3A. Placing the victim on the left recovery position

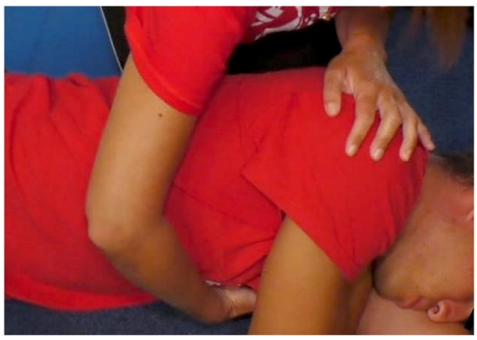


Fig. 3B. Checking the victim's heartbeat via the 5th intercostal space

Fig. 4A. The *refill* maneuver, showing how to lift the victim effortlessly by the waist.

Fig. 4B. Adjustment of the victim's position with both hands and the right thigh in the direction of green arrows

Fig. 4C. The *recoil* maneuver in near-circumferential chest compressions by leaning forward on the victim, compressing the chest with both hands, forearms and both thighs in the direction of the green arrows

Fig. 4D. The *rebound* maneuver: sudden release of both hands and all chest compressions after a few seconds of the *recoil* maneuver

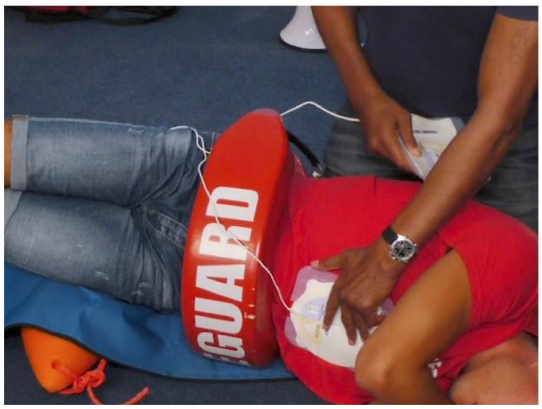


Fig. 5. Installation of AED's electrodes in the anteroposterior position (sternal & intrascapular)

Fig. 6. Demonstration of chest compressions in neonates and infants

3. RESULTS

Bypassing the sternal barrier, refilling the heart, compressing/decompressing the chest within the rules of thoracic biomechanics will significantly improve outcomes of SCA victims, promoting ROSC.

4. DISCUSSION

Compared to current CPR, the proposed 3R/CPR technique can potentially improve outcome of SCA victims for several reasons.

First, instead of vigorously and inconceivably compressing the victim's sternum, the technique provides a more rational hemorheological effect on the conducting system promoting ROSC during the early onset of cardiac arrest. For example, the *refill* maneuver can shift a massive volume of the stagnant infradiaphragmatic venous capacitance to the cardiothoracic compartments to be handled properly with the *recoil-rebound* processes, executed according to the rules of Biomechanics of the thoracic cage [46]. This can enhance chances of ROSC, *directly* by inducing endothelial shear stress (ESS) and atrial wall stress in a water hammer-like mechanism with a snap effect at the conducting system, and *indirectly* by improving the myocardial perfusion due to the increased RV preload. The crucial role of intraventricular ESS enhancement has been demonstrated in our previous studies with pulsatile garments leading to an instantaneous ROSC in ≤ 8min of cardiac arrest model and a significant improvement of hemodynamics in acute RV failure model[*] [47].

Second, inducing chest compressions through the 5th intercostal space while leaning on the

[*]*Operative movies available upon request.*

victim body in the left recovery position, provides near-circumferential (hoop) stress to the cylindrical ribcage in complete harmony with costal axial motion. This makes the technique more effective, less traumatic for victims and less exhausting for rescuers. On the other hand, vigorous mid-sternal chest compressions as schematized in Fig. 1, induce deviated longitudinal stress causing the well-known serious traumatic incidents of CPR [48-50].

Third, the technique provides additional circumferential compression on the heart from the surroundings chest compartments like the mediastinum, parenchyma and diaphragm due to the wrapped trunk. This can literally explain studies claiming the quality of some peculiar CPR trends e.g., prone position or with retained lungs inflating.

Fourth, the left recovery position, which secures the victim's airway, helps the rescuer not to confuse cardiac arrest with similar clinical presentations. For example, as simulated in Fig. 3B, a bystander can easily feel the victim's heartbeats in situations most commonly mistaken for cardiac arrest such as syncope or cardiogenic shock.

In correlation with the other CPR modalities, insofar as ventilation is pointless due to the hemostatic state, it is preferable to continue the 3R/CPR without interruption until arrival of emergency squads to transfer the victim to Cardiac Centers.

Also, since controversies between chest compression first or DC shock remain unsettled, we favor the first choice for several reasons. First, because there is still a lack of studies showing an immediate ROSC following a first DC shock.

Second, since the sensitivities of conventional 12-lead ECG systems remain questionable [51], it is therefore evident that data obtained with two anterolateral chest electrodes of AEDs might not be reliable. Thus, it is more convenient to perfuse and predispose the cardiac tissue with the 3R/CPR first before any trial of DC shock to avoid unnecessary damage by electroporation of the rare pacemaker cells.

Overall, regardless of the ROSC, we believe that 3R/CPR allows more rational exploitation of stagnant hemorheological zones, which will ultimately contribute to effective management of victims during the refractory and postarrest phases.

4.1 Refractory and Postarrest Phases

As is done in cardiac surgery, ensuring organs' perfusion with CAD is a top priority during the refractory phase of cardiac arrest. Also, as it is unwise to flog a tired horse, CAD must be continued in postarrest until full recovery with the restoration of endothelial functions. This has been clearly demonstrated in the literature proving that brain damage can be significantly improved by maintaining adequate perfusion during cardiac arrest, either with CPB as we previously demonstrated [27], or ECMO [52,53].

However, the employment of E-CPR remains a work in progress due to the difficulties of installing it through near-empty flattened arteries in outdoor environments. Moreover, as occurs in postcardiotomy syndrome [54,55], E-CPR provides a steady-flow mode of perfusion that further aggravates endothelial dysfunction. This may be aggravated by vasopressors which increase myocardial oxygen consumption and mechanical ventilation which suppresses the important role of the respiratory pump as a potential generator of ESS, most likely to end in multiple organ failure [56].

As is known, to maintain metabolic processes in a multicellular organism like a human being, we need to restore ESS that controls organs' microcirculation via a range of endothelial mediators. We previously demonstrated the undeniable crucial role of endogenous pulmonary endothelial mediators in controlling hemodynamics, microcirculation, and metabolism, regardless of cardiac conditions, e.g., healthy, dysfunctional, or arrested [57].

Therefore, the employment of CFR, which is a non-invasive device unlike ECPR, can mobilize a massive amount of stagnant blood volume (≥4 liters) in a pulsatile mode promoting ESS with improvement of microcirculation and cellular metabolism during refractory and postarrest phases [27]. In addition, the CFR device's vest, associated with a passive oxygen insufflation device serves as a noninvasive mechanical ventilator with a nasogastric tube could be useful during refractory cardiac arrest and in postarrest to prevent excessive ventilation in maintaining the pulmonary ESS [56].

4.2 Prophylaxis

Although the prediction and prevention of SCA events can be successfully managed in certain pathophysiological conditions such as patients with heart failure [58], or Brugada syndrome [59], it nevertheless remains one of the supreme goals of scientific research. Especially since many physiopathological incidents of SCA often occur in healthy populations such as during physical exercise, due to transient disturbances in the transmission of impulses through the conduction system. A similar phenomenon that perhaps worth further investigation, occasionally occurs in open-heart surgery following hasty manipulations causing ventricular fibrillation even without cardiomyopathy like in-vivo experiments with porcine models. Therefore, we intend to devote our next work to the prophylaxis of SCA in sports, especially since our pulsatile suit patent [47], has been infringed and is currently used by sports elites, but in an unscientific manner that almost does worse than good.

4.3 Limitations

The study was limited to meticulous analysis of clinical observations and fundamentals of medical science as well as rationally organized common senses (Th. Huxley), rather than controversial animal models for several reasons. First, as shown in Fig. 1, the huge discrepancy in cardiotorsal anatomy between species could compromise *recoil-rebound* maneuvers according to the rules of thoracic biomechanics, for example, chest compressions must be induced circumferentially in humans. Second, hemorheologically speaking, differences in volumes and surfaces of stagnant blood zones e.g., splanchnic, that need to be compressed and shifted to the thorax, will significantly affect the *refill*-process. This is in addition to the high mortality of cardiac arrest models, which we considered in our previous study [27], conducted with several prototypes to accommodate the porcine-canine morphological difference and a limited number of animals to avoid unnecessary losses. On the other hand, the advantages and credibility of 3R/CPR over CPR can be proven with angiographic technology on cadavers [60], or via computational models, which are increasingly applied nowadays [61].

4.4 Perspectives

According to recent recommendations from the United States Food and Drug Administration [62], we are planning to demonstrate the benefits of the 3R/CPR versus CPR via computational models [63]. According to the FINER criteria for a good research question and the phases of evaluation of new therapies, we believe that the 3R/CPR technique is feasible, interesting, novel, ethical and relevant [64,65].

5. CONCLUSIONS

Outcome of SCA victims remain poor due to insufficiency of current CPR. Unlike conventional chest compression, the 3R/CPR can adapt to the hemostatic-hemorheological conditions and thoracic biomechanics, providing rational exploitation of the stagnant blood, promoting ROSC across all ages, genders and environments, in a less traumatic and exhausting manner.

DISCLAIMER

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COMPETING INTERESTS

Author has declared that no competing interests exist.

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