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Research Article

Chest Strikes, Back Slams, and Coughing to Promote Early Reflow in STEMI

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Abstract

This paper presents a new first-line emergency treatment hypothesis for confirmed or suspected ST-Elevation Myocardial Infarction (STEMI), at home or in the field, in the hopes of providing early restoration of blood flow to the presumed, acutely thrombosed epicardial coronary artery. The therapy, transthoracic Thrombo-Agitative Percussion (tTAP), consists of robust, patient self-inflicted "fist strikes" to the left and right of the sternum, preferably with deep forceful coughing, and optionally with "upper back slams" (self-administered or delivered by a bystander), in the hopes of promoting early clot disruption and recanalization of the culprit vessel. ST elevation is optimally confirmed by a surveillance wearable, whereafter thrombo-agitative maneuvers begin immediately, taking advantage of an underappreciated vulnerable period while clots (seconds old) are still loosely bound, weakly adherent, platelet aggregates. New penetration studies are submitted demonstrating that tTAP maneuvers, all when delivered with enough force, can cause substantial displacements to the epicardial surfaces of the heart where the coronaries are located, which supports the thrombo-agitative mechanism of the therapy. Moreover, benchtop pilot work is presented showing how gentle, extra-luminally applied serial compressions applied upon a clotted, flexible tube system (sized to resemble a coronary artery), substantially disaggregate and fragment fresh, newly formed clots. There are, of course, many possible added risks for administering tTAP to a heart attack patient, the discussion of which is a primary focus of this paper. Indeed, at this, the "concept phase", tTAP raises many more questions than answers, and to many will be seen as mad, potentially dangerous science, but the theory and preliminary data are herein provided to get the discussion flowing.

Keywords

STEMI, Percussion, Reperfusion, Thrombolysis, Vibro-Acoustic therapy.

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Introduction

Thrombo-occlusive cardiovascular diseases are the leading cause of death in the developed world, and among these, ST-Elevation Myocardial Infarction (STEMI) is the most serious, carrying a high morbidity and mortality. With that, it is well established that rapid and complete restoration of blood flow of the thrombosed artery is the main determinant of positive clinical outcome [1].

It is well known that in a heart attack, "time is muscle" and that there is a clinical advantage to treating STEMI to resolve the ST segment as soon as possible, which implies reflow [2,3], and downgrades the event to NSTEMI.

STEMI cases are typically referred to the Cardiac Cath Lab for Primary Percutaneous Coronary Intervention (PPCI); however, there are sometimes lengthy delays, leaving the thrombosed vessel occluded or suboptimally recanalized, which is known to increase mortality and morbidity [4]. IV thrombolytic therapy, therefore, remains a common alternative therapy worldwide, particularly in rural areas [5], however, IV thrombolysis has a relatively low complete reperfusion rate and has worrisome potential hemorrhagic complications, including stroke [6].

Importantly, all STEMI therapies (including PPCI and IV thrombolysis) suffer from incomplete microvascular reflow, where

small thrombotic fragments after clearing the epicardial infarct vessel, clog the distal coronary vasculature [7].

Recognizing the need for earlier, faster, and more complete reperfusion, the Author and his associates have been studying the method of administering chest wall directed transthoracic Diastolic Timed, Vibration (tDTV), by use of a palpable oscillation massager, operable at ~ 50 impacts / second, ~ 4 mm stroke amplitude [8-12], to promote early reflow in STEMI.

In background, palpable, low-frequency oscillative mechanical percussions in the infrasonic to sonic range (i.e., < 100 Hz) have proven abilities, when directly applied or delivered across a tissue or luminal barrier, to break apart blood clots, with or without a clot-thinning or thrombolytic drug agent [13-18]. Moreover, it is well documented that low-frequency percussive massage (by hand, or by a vibrating instrument) when applied to the body is generally known to stimulate blood flow, primarily by adding cyclic stress and strain to the endothelium, which causes a localized, endogenous liberation of nitric oxide, a powerful vasodilator [19-25].

In particular, our research team has shown that low-frequency vibration (50 Hz, 4 mm stroke) applied across a four-centimeter chest wall-sized tissue barrier, enhanced thrombolytic action to an underlying clotted tube system; this effect likely in part caused by adding turbulence and convection currents, which enhanced lytic mixing and erosion at the clot/ liquid barrier [15]. This supports the concept that tDTV could prospectively be of use to enhance systemically administered thrombolytic drug delivery in STEMI prehospital thrombolysis. See Figure 1, showing a diagrammatic representation of a STEMI victim being treated by a prophesized hand-engaged tDTV device to improve pre-hospital thrombolysis.

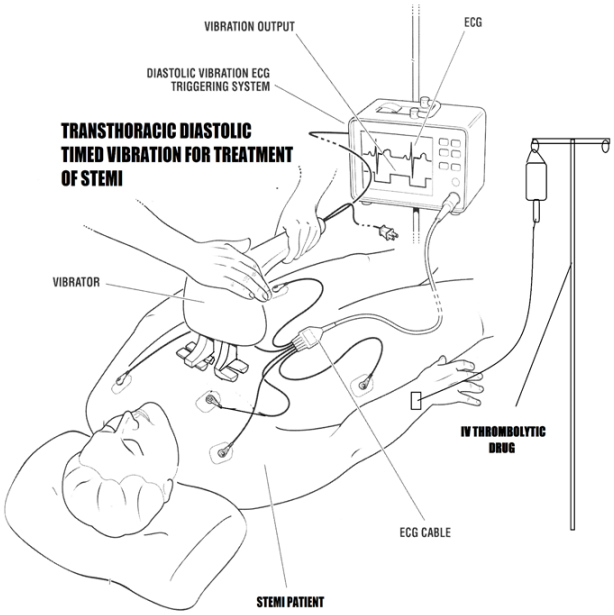

Figure 1: A diagrammatic representation of a proposed tDTV massage system as a means to assist pre-hospital thrombolytic drug delivery to assist STEMI reperfusion.

Notably, chest wall delivered tDTV was extensively studied by a Japanese group led by research Cardiologist Dr. Y Koiwa in the 1990s, where they envisioned the therapy, not for STEMI, but instead as a non-invasive left ventricular assist treatment for ischemic heart failure. Indeed, in a landmark series of pre-clinical and clinical discoveries, Koiwa found not only that chest wall delivered vibration can penetrate to reach the heart, but also that when timed exclusively during the diastolic phase of the cardiac cycle, the therapy improved LV diastolic function [26], enhanced coronary flow [27-29], and improved cardiac output presumably by the Frank Starling's mechanism [30,31].

Hence, with regards to tDTV for STEMI, we inferred that by massaging and assisting relaxation of the ischemic myocardium during diastole (which would in theory lower trans myocardial vascular resistance), the therapy might be useful, beyond promoting initial clearance of the clot in the epicardial vessel, to continue on to tackle the "no" and "slow" reflow phenomenon by assisting distal capillary reflow [32,33]. Importantly, from a practical application standpoint, low sonic frequency vibration has been experimentally shown to have unique internal transmission characteristics through body tissue, including along arteries and the epicardium [34-37]. Hence, in theory, a tDTV device or percussive massager could be routinely placed by a paramedic or nurse upon the anterior chest wall of a STEMI victim, without need for specific imaging guidance or targeting.

See Figure 2, showing the general anatomic location of the coronary arteries relative to the chest wall, with preferred contact points for a tDTV procedure situated to generally overlie the vessels.

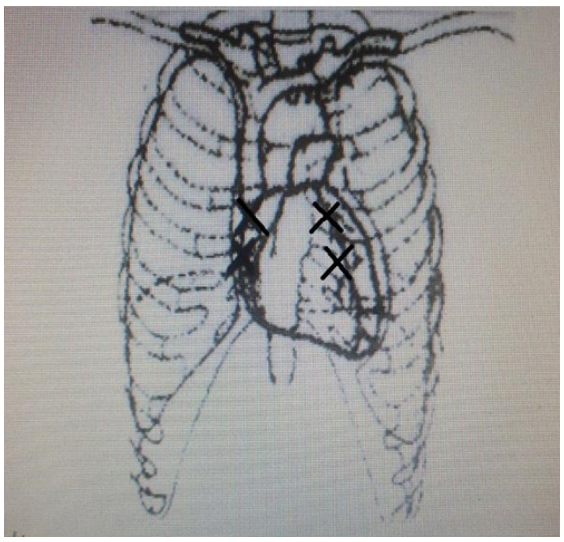

Figure 2: Contact node placement sites for a proposed tDTV device in STEMI care are shown by "X", to generally overlie the RCA and LAD.

Fast forward to 2025, with tDTV never having the opportunity to be studied, it occurred to the Author that perhaps it might be helpful, particularly in STEMI, to forgo equipment and consider

making the delivery of the transthoracic delivered percussions manual, and hence immediately available and deliverable by the patients themselves.

Indeed, could STEMI reperfusion be achieved by simply "beating one's chest" in a King Kong-like maneuver?!

With this, one could also imagine a multifaceted, multidirectional percussive approach, by further adding upper back claps to stimulate moreso the posterior aspect of the heart, and deep coughing, which is known to posterior laterally and medially compress the heart's surfaces, thereby providing therapeutic agitation and serial compressions to virtually the entire coronary tree.

Indeed, the administration of such patient self inflicted thrombo agitative mechanical maneuvers at the very first instant of anginal like symptoms, a therapy herein called transthoracic Thrombo-Agitative Percussion (tTAP), would theoretically take advantage of what may be shown to be an unappreciated early vulnerable period which we are calling the "platinum seconds of reperfusion", when newly formed hyperacute clots (literally seconds old), as loosely bound, weakly adherent, platelet aggregates, are relatively small (hyperacute, just big enough to block the vessel), and particularly unstable prior to any deposition of fibrin.

This therapy hopes to provide percussive forces across and through the thoracic cavity that would transmit sufficiently to cause resultant gentle but persuasive serial compressions and decompressions, percussions, and shaking of the epicardium, including the coronary arteries, in the hopes of safely dislodging, disaggregating, and clearing a new-onset platelet aggregate.

See Figure 3, showing the proposed triad therapy with the Author thumping his chest while deeply coughing, and slamming his upper back against a wall. A movie short showing tTAP can be seen by clicking on the following link: https://youtu.be/yMJao7FUSZw.

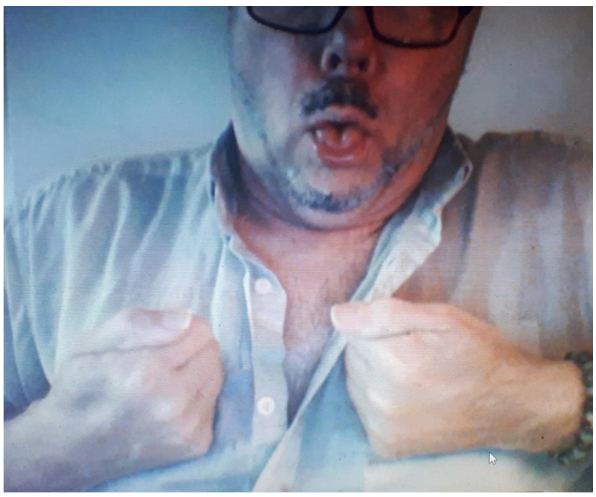

Figure 3: A model (the Author) is shown administering self-administered transthoracic Thrombo-Agitative Percussion (tTAP) via rapid, forceful bilateral fist strikes to the left and right of the sternum, deep aggressive coughing, and upper back slams (not seen in the figure), as an emergency technique to promote clearance of acute coronary thrombosis.

A patient would most preferably be equipped with an ST segment monitoring wearable, with ECG leads strategically positioned or placed subcutaneously to enable real time monitoring of a 12-lead, which would upon showing a sudden elevation of a patient's ST segment, alert the patient to potential STEMI, suggest or automatically make a 911 call, and consider tTAP to assist in clearing the clot.

See Figure 4, image (left), showing a heart attack victim looking at his EKG-enabled smart watch, with image (right) showing the screen on the watch with ST elevation, and a message stating to call 911 and initiate tTAP.

Figure 4: Shown (left) is a model pretending to have a heart attack as he looks at his smartwatch. Image (right) shows the watch displaying ST elevation, with a message to call 911 and consider initiating tTAP. Image downloaded having a valid subscription from OpenAI.

Transthoracic Thrombo-Agitative Percussion (tTAP)

In more detail, herein is introduced a new hypothesis in first line STEMI care to promote initial reflow before arrival of EHS, that "transthoracic Thrombo-Agitative Percussion" (tTAP), by delivery of rapid, forceful, bilateral fist strikes to the left and right of the sternum (~ 3 strikes / second), preferably along with deep forceful coughing (~1 cough per three seconds), and with the option of adding upper back slams (~ 2 to 3 slams / second), initiated by the patient themselves, or with help from a bystander, at the very first suspicion of anginal-like symptoms, may facilitate initial clearance of hyperacute, new onset coronary thrombosis.

The percussions and coughing are to be delivered as forcefully as safely tolerable to the patient, to ensure the delivery of therapeutically efficacious levels of forces to the epicardial surface of the heart.

As it is impossible to know the exact location of a coronary thrombus, an advantage of tTAP is its multidirectional approach.

Fist strikes applied anatomically rightward to the sternum generally provide agitative forces to the Right Ventricular (RV) free wall, which underlies and supports the proximal to mid-RCA and RV marginal branch, while leftward the sternum sends percussive stimuli to the anterior LV free wall, which underlies and supports the Left Main (LM), LAD, diagonals and Ramus vessels. Upper back slams would promote serial compression more so to the posterior aspect of the LV which underlies the Posterior Descending Artery (PDA), and repetitive coughing (as coughing posterior laterally and medially compresses the heart) would serve to provide serially compressive therapy to the PDA, mid-RCA, Left Circumflex (LCx), and Obtuse Marginal (OM) vessels, as well as to re-enforce compression throughout the coronary vasculature.

It should be noted that it is the Author's experience that the techniques employed in tTAP, at least in men, are not overly painful, and as performed while otherwise comfortably resting while standing or sitting, take surprisingly little effort to perform. Moreover, the idea would be "just to try" the technique, the idea being that if it should work, it should work immediately (within seconds), so a reasonable time limit could be set for an attempt of no greater than 2 to 3 minutes.

It is recognized, of course, that the chest strikes of tTAP, would likely be poorly received in females due to the sensitivity of the breast area, however, to this, notably, approximately seventy to seventy-five percent of STEMI victims are men [38,39], and perhaps females could be advised to limit tTAP to upper back claps and deep coughing.

But all this said, how can we be certain that blunt mechanical forces (such as percussions or compressions) applied to the external surface of a hyper-acute thrombosed coronary artery would lead to reflow?

Supportive Data

The "Folts Effect".

The most relevant data to support that mechanical impacts or forces delivered upon the external surface of a hyper-acute, new onset thrombosed coronary artery would lead to near immediate to immediate re-canalization and reflow, comes from experience gleaned from the "Folts' open canine coronary thrombosis model.

Dr. Folts, the Director of the Coronary Artery Thrombosis Research Laboratory at the University of Wisconsin Medical School in the 1980s, demonstrated in an open canine model that gentle "poking" or "shaking", of the external surface of a hyper- acutely thrombosed coronary artery, at the first noted moment of thrombotic occlusion (within seconds after zero coronary flow was registered by a flow meter), reliably led to instant and complete reflow within the vessel [40,41].

See Figure 5, which diagrammatically shows the set up of the Folts' open canine coronary thrombosis flow model.

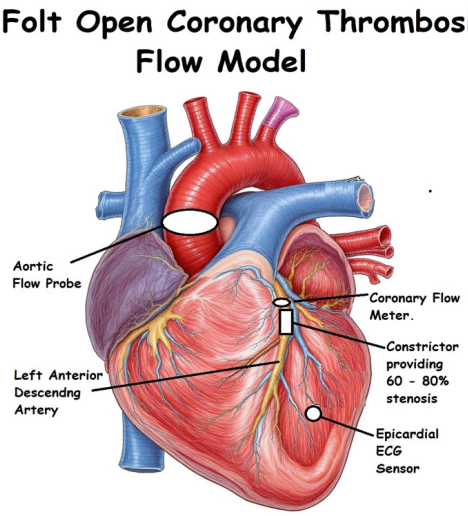

Figure 5: A diagrammatic rendering of the setup of the Folts' open, coronary thrombosis flow model. A constrictor is placed in this case on the proximal LAD, which causes a site of endocardial injury, which causes platelet aggregations to form, and spontaneously release, where the blood flow rate is continuously monitored by a flow meter.

Referring to Figure 5, a plastic, 2.5 cm long cylindrical "constrictor" (shown here deployed upon the proximal LAD) was reportedly used to produce a "60–80%" stenotic site of endothelial injury (an experimental equivalent to an ulcerative plaque), which led to a cyclic aggregation and disaggregation of platelets at the injury site, where coronary flow was monitored. The system was designed to analyze the frequency of development of partially obstructive platelet aggregates in the assessment of various antiplatelet medications.

Dr. Folts reported that at times the studied coronary artery in his model would become untowardly thrombotically occluded (with zero flow being registered on a flow meter), whereby, a surgical instrument would gently "shake" or "poke" at the artery or the constrictor [40,41], which reportedly led to reliable and immediate reflow of the vessel (herein dubbed the "Folts' Effect").

It is unknown whether such an occlusive clot would have simply detached and moved forward intact downstream, or perhaps disaggregated (or fragmented), or both; however, generally complete occlusions were rare, and once an artery was recanalized, blood flow rate was generally fully restored to baseline levels.

An example of a Folts flow meter readout is shown in Figure 6, where zero flow occurred (in this case, the left circumflex artery had become thrombotically occluded), whereafter the experimenter would "shake loose" the constrictor, which led to immediate flow restoration. Noteably, Folts showed that given a site of endocardial injury on a coronary artery, his model generally caused platelet aggregations (which slowed flow), followed almost always by "spontaneous release" (shown by "y"), and once a vessel became occluded (causing a STEMI), this did not mean, necessarily, another occlusion would take place.

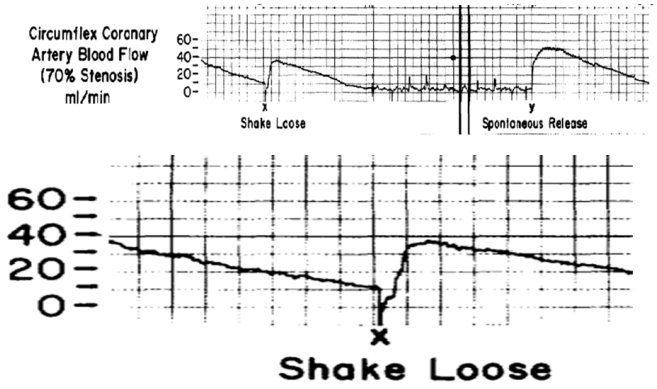

Figure 6: (Top) The "Folts Effect". A flowmeter readout from the Folts coronary thrombosis model indicating that the left circumflex artery had become thrombotically occluded with zero flow - shown at "X", whereby the Experimenter simply "shook loose" the constrictor, which immediately led to normalized flow. (Bottom) Zoomed in view of the flow meter, showing zero flow at the time of "Shake Loose," which quickly led to restoration of baseline flow. Taken from Folts J et al.: Blood Flow Reductions in Stenosed Canine Coronary Arteries: Vasospasm or Platelet Aggregation? Circulation 65, No. 2, 1982 pp 248 – 254. Image downloaded, August 5th, 2025, from http://ahajournals.org (open Access).

The Folts' Effect, of course, begs the question of whether tTAP maneuvers could sufficiently penetrate to cause an adequate comparable mechano-agitative stimulus, similar to direct tapping or shaking, upon a newly thrombosed human coronary artery to promote reflow?

Chest Wall Fist Strikes

To prove that powerful chest wall fist strikes just leftward the sternum can penetrate to cause an impact upon the anterior surface of the LV (home of the LAD), the Author, took an M-mode image of his LV while aggressively striking the left sternal margin of his chest (strikes delivered upon the left 3rd intercostal space, echo transducer held pointing upwards from the left 4th intercostal space). See Figure 7, to see what mechanical effects to the LV myocardium could be observed.

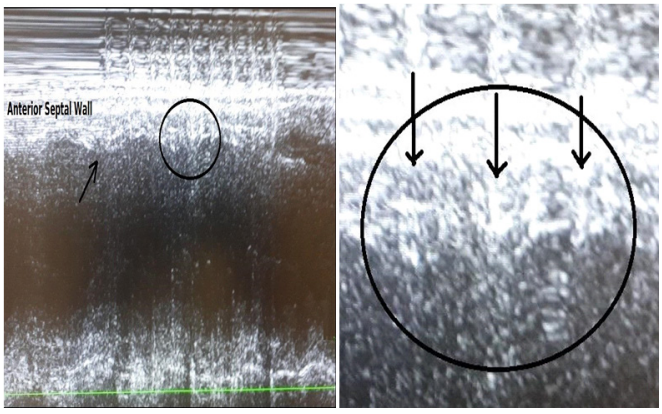

Figure 7: Effect of Chest Wall Fist Strikes Just Leftward of the Sternum. M-mode figure (Left) showing the LV during forceful fist strikes to the left sternal margin of the chest wall (starting at the arrow). Figure (Right) shows a zoomed-in view of the anterior-septal wall to better show the resultant compressive displacements. Image taken from the Echo-lab, False Creek Healthcare Center.

Referring to Figure 7, the commencement of fist strikes to the left sternal margin of the chest (shown by arrow) is consistent with marked vertical echoic displacements of the anterior septal wall of the LV (circled), which is known anatomically to underly, support, and be perfused by the LAD. It is hypothesized, therefore, that chest strikes leftward of the sternum could plausibly provide the required agitative and compressive forces to accelerate and ensure first-line early recanalization and initial reflow in Anterior-Septal STEMI.

On the other hand, to show how chest-wall fist strikes just rightward the sternum penetrate to reach the Right Ventricle (RV - home of the proximal to mid-RCA and RV marginal branch), see Figure 8, which shows an M-mode of the RV free wall, along with chest wall fist strikes delivered just rightward the sternum.

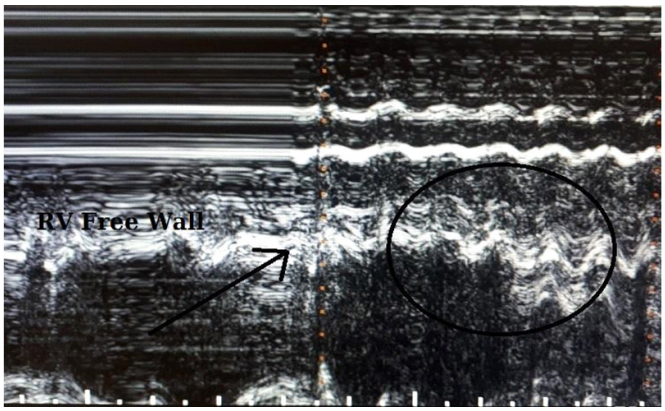

Figure 8: Effect of Fist Strikes just Rightward the Sternum on the RV free wall. M mode showing the RV before and during fist strikes (starting at the arrow). Image taken from the Echo-lab, False Creek Healthcare Center.

Referring to Figure 8, the commencement of fist strikes along the right sternal margin of the chest (shown by the arrow) is accompanied by marked vertical echoic compressions of the RV free wall (circled). As the RV free wall is known anatomically to underly, support, and is perfused by the proximal to mid-RCA and RV marginal branch, it is hypothesized that fist strikes just rightward the sternum may be beneficial to promote early recanalization and initial reflow in Inferior wall STEMI, with or without RV involvement.

Forceful Coughing

Now, turning our attention to forceful coughing, The Author also took an M-mode image of his LV to assess how deep coughing may affect the heart (see Figure 9).

Referring to Figure 9, three consecutive deep coughs (starting at the arrow), caused substantial compressive displacements, most evident to the posterior wall (near the bottom of the image), but

also seen to a lesser, but still significant degree to the anterior-septal wall.

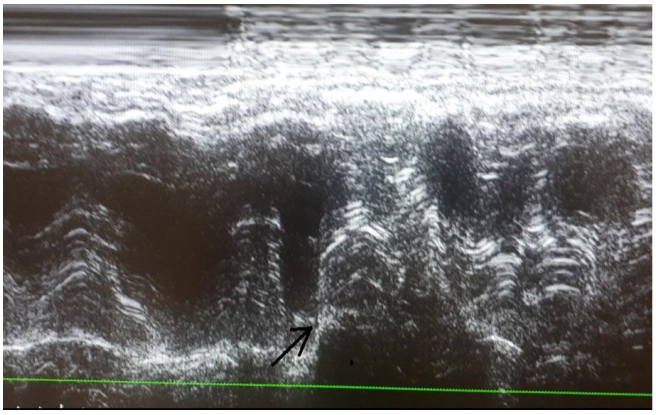

Figure 9: Effect of Deep Coughing Upon the Heart. M mode showing the LV (anterior septal wall - top, posterior wall - bottom) with three consecutive deep coughs (starting at the arrow). Image taken from the Echo-lab, False Creek Healthcare Center.

It must be stressed that if coughs were to be utilized in tTAP for STEMI, they must be strong and vigorous. A deep breath should be taken, and the cough must be strong and prolonged as when producing sputum from deep inside the chest. The cough should be repeated every two to three seconds, (thereby allowing the patient to get into a comfortable rhythm and provide good deep, respirations, to avoid getting tired or short of breath) and should be continued, with intermittent pauses for re-assessment, until there is an abatement of anginal discomfort (and/or a normalization of the ST-segment) which signifies reperfusion has occurred.

Upper Back Slams

Finally, to show how forceful self-inflicted upper back slams can penetrate to mechanically affect the heart, the Author again, took an M-mode image of his LV, this time while aggressively slamming his upper back, back and forth, against a wall (see Figure 10).

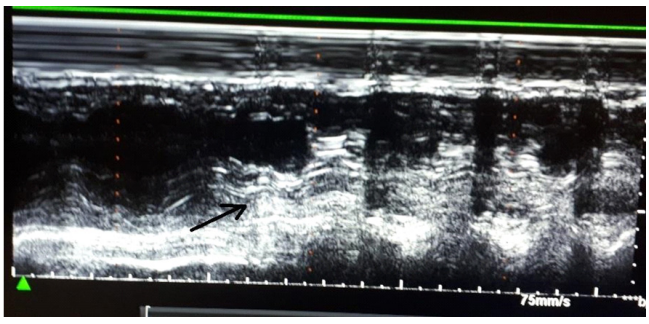

Figure 10: Effect of Upper Back Slams Upon the Heart. M mode showing the LV (anterior septal wall - top, posterior wall - bottom). The arrow indicates the beginning of self-administered upper back slams. Image acquired from the Echo-lab, False Creek Healthcare Center.

Referring to Figure 10, marked vertical echoic displacement lines into and deforming the posterior wall (and to a lesser degree the anterior septal wall) can be seen, consistent with the initiation of upper-back slams (marked by the arrow). It is thereby hypothesized that upper back slams could provide serial, repetitive compressive stimuli to the posterior epicardial surfaces of the heart, including the PDA, and could thereby potentially assist STEMI reperfusion with posterior involvement.

It should be added that upper back slams are the most comfortable (in the Author's experience, they "don't hurt at all, like receiving a back massage") and take very little energy to perform.

Benchtop Data: The Effect of Extraluminal Serial Compressions Upon Newly Formed Clots

Introduction: The following experiment was designed to gain some preliminary data and experience to understand how newly formed clots respond to external transluminal serial compressions. Experimental Hypothesis: Newly formed venous clots dispensed within a deformable rubber tubing (4 mm vessel diameter), are expected to significantly disaggregate and fragment following two minutes of gentle serial fingertip compressions (~ 3 compressions per second) applied to the external surface of the tubing overlying the clot.

Methods: Venous blood (n=10) was self-collected with a 26-gauge needle from the Author (left arm, antecubital space) into 5 cc syringes, then allowed to sit for one minute while a band-aid was applied to the venipuncture site. Then 1 to 2 mL of blood was dispensed from the syringes into the open end of a section of deformable rubber tubing (4 mm lumen diameter), being sized to resemble a coronary artery.

Prior analysis of venous clots collected from the same test subject and timed for clotting by the "Lee -White" method showed that clotting occurred at room temperature usually within 7 minutes and in all cases within 8 minutes, so to ensure clotting all blood dispensed within the rubber tubing sat for a 10-minute "incubation phase", whereafter the tubing with clot within was photographed.

Once the tubing was deemed clotted, samples entered an "evaluation period", where they were randomized to sit quiescently for an additional 2 minutes (Control samples) or instead receive gentle fingertip compressions along the tubing overlying the clot for 2 minutes (Compressed samples).

After the evaluation period expired, water was gently dispensed into the opposite end of each tubing to flush the clot out, and the resulting clot was photographed.

Results

Control Samples: Control samples, which sat quiescently following the two-minute evaluation period, demonstrated, once the tubing was flushed, a substantially intact clot, with varying, only very minor degrees of pinkish serum bleeding therefrom.

See Figure 11, showing two typical control samples (left to right: End of the 10-minute clotting incubation phase, to the expulsion of the clot following two minutes of quiescent sitting).

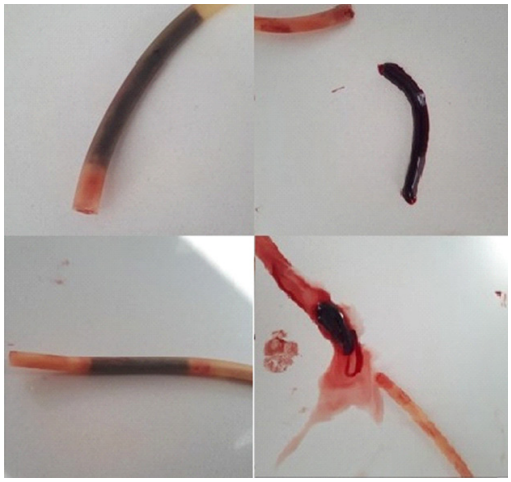

Figure 11: Control Samples. Images (Left) show a pair of rubber tubes, each containing a blood clot following a 10-minute incubation phase. Images (Right) show the resulting clots having been flushed from their tubes after sitting quiescently for an additional two minutes. As can be seen, the clots appear substantially intact.

As can be seen in Figure 12, the control samples exhibited minimal to no evidence of clot disaggregation following two minutes of quiescent sitting within their respective rubber tubes.

Compressed Samples

In comparison, all five "Serially Compressed" samples, following two minutes of compressions over the clot, when flushed, appeared as several tiny, clotted fragments, indicating that a great deal of clot disaggregation, including fragmentation of the clot into tiny pieces, had occurred.

See Figure 12, showing two examples of samples treated by serial compressions (left to right -> end of the clotting incubation phase, to expulsion of clot following 2 minutes of compression treatment).

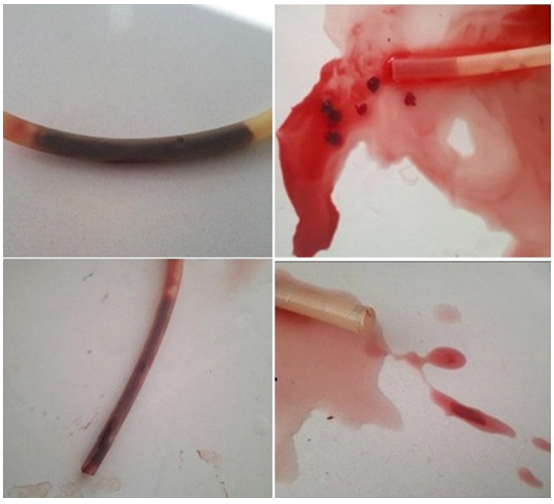

Figure 12: Compressed Samples. Images (Left) show a pair of 10-minute-old venous blood clots having formed within a rubber tubing sized to resemble a coronary artery. Images (Right) show the resultant clots flushed from their tubes after having received two minutes' worth of fingertip compressions of their rubber tubing overlying the clot. As can be seen, the clot has been crushed into tiny multiple fragments.

Discussion

To many cardiovascular researchers, the concept of, in a frenzy, using one's vital energy to promote initial recanalization of acutely thrombosed coronary artery by beating one's chest, literally like King Kong, will be considered as mad, dangerous science, that is best forgotten and sequestered from the public.

Indeed, at this early "concept stage," it may be considered unethical to broadly deliver information on tTAP to the public. Indeed, heart attack victims, who would be frightened and desperate to "do something", would undoubtedly try tTAP well before the concept may be proven as safe and effective.

However, it has been established by review of the literature that in the Folts open canine model, gentle shaking or poking of the external surface of a hyper-acutely thrombotically occluded coronary artery, whereby a platelet aggregate had formed atop a site of endocardial injury, leads to immediate and reliable reflow of the vessel (refer to Figures 5 and 6) [40,41].

With that, we have shown that tTAP maneuvers, all, when delivered with enough force, can prospectively transmit their percussive/compressive forces to cause observable displacements to the epicardial surfaces of the heart where the coronary arteries are located.

These facts alone, without further inquiry, support the hypothesis that tTAP could possibly render immediate to near-immediate (perhaps within seconds!) initial recanalization and reflow to a new onset thrombotically occluded coronary artery, the chief mechanism of STEMI. But would that, even if it "works" in initially clearing the vessel, be a good thing?

It has been counter-intuivitivey sugested in peer review that, tTAP, by clearing and leaving exposed the site of thrombosis development (the ulcerated plaque), may simply lead to increased clotting (with further re-occlusions), which, even if cleared again and again by subsequent tTAP, may in turn untowardly increase overall clot burden (with sticky clots, and platelets moving downstream), making matters worse.

However, on the other hand, perhaps tTAP recanalization would be helpful in decreasing overall clot burden? After all, platelet aggregations are dealt with incredibly early (literally at the moment the clot becomes occlusive), hence are not given the opportunity to grow and consolidate upstream or downstream from the stenosis site. Furthermore, once reflow is established, it is presently unknown whether a hyperacute platelet clot (seconds old) would substantially disaggregate and wash innocently through the circulation or begin to occlude downstream (indeed, blood flow rates always returned to baseline in the Folts Model), nor do we know for certain that a

re-exposed ulcerated plaque would necessarily re-occlude, at least prior to arrival of EHS or upon reaching the cath lab. To answer these questions, we need clinical trials.

Prospective Mechanisms for tTAP

In light of our experimental result, see Figure 13, which diagrammatically prophesies an ideal, best-case situation, how a new platelet clot within an acutely occluded epicardial coronary artery may be anticipated to disaggregate and fragment into smaller pieces, as a consequence of coronary compressions delivered by tTAP.

That extra-luminal percussion or compressions can lead to clot detachment, migration, and clearance in a fluid-filled luminal system is not surprising, as our research group has shown previously that external percussion/compressions overlying clotted, pressurized, flow tubes have caused mobilization/clearance of clots, even past significant stenosis sites [13].

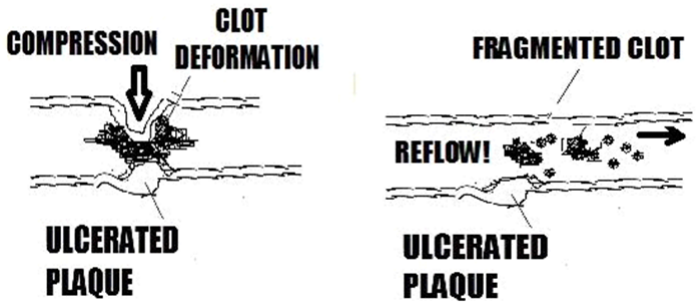

Figure 13: Diagrammatic rendering of (Left) shows an acutely thrombosed coronary artery being deformed by an extra-luminal percussive stimulus rendered by tTAP, thereby vis-à-vis "squishing" or "smashing" the clot. Image (Right) shows how the clot, following multiple compressions, has broken up into smaller fragments, thereby rendering initial recanalization of the vessel.

Less ambitiously, it is also possible that the delivered forces of tTAP may not be of enough strength to deform or compress the culprit vessel (which is needed to squish the clot), or may not hit the artery at the level of the clot, or may miss the artery completely and only stimulate the epimyocardium.

However, according to the Folts Data, all that was needed to effect recanalization of a hyperacutely thrombosed artery was to gently "shake" or "poke" at the vessel or the constrictor (which was solid, so would not have squished the artery), so actual compressions of the artery, which actually squish the clot may not be necessary to effect reflow.

Notably, as mentioned earlier, experimental studies have shown that low-frequency vibration stimulus to the epimyocardium (somewhat analogous to serially applied percussions) transmits, presumably by a tethering effect, both basally and apically from the tissue's impact arrival point [34-36], and that this transmission effect has also been proven to occur along the length of arteries [37]. Hence, from this, even a percussive stimulus delivered to the heart or coronary artery "off mark", may still likely provide at least some degree of a shaking effect to the coronary vessels.

There is also some mechanistic experimental evidence that suggests that extra-luminal compression of a thrombosed artery, even rendered far remote from the clot, may be helpful to render reflow.

Indeed, in 2013, the Engineering Department of Simon Fraser University performed a pioneering in-vitro flow model study [18] which showed that rapid partial compression/decompression (~ 24 compressions per second) of a pressurized, clotted, stenosed, multi-branch "deformable" flow tube system, INCLUDING WITH COMPRESSIONS APPLIED TO THE FLOW TUBE FAR REMOTE FROM THE CLOTTED SEGMENT (E.G. 60 CM UPSTREAM FROM THE CLOT!), caused significantly improved recanalization rates to the flow system vs. controls having received no pulsations, plus an observed fracturing and debulking of the clots!

So, in view of the results of the SFU flow model study, see Figure 14, which diagrammatically shows how, in vivo, a new platelet clot could theoretically be "pushed", deformed, and fragmented from its site of attachment by an upstream compression of its connecting artery, thereby rendering reflow.

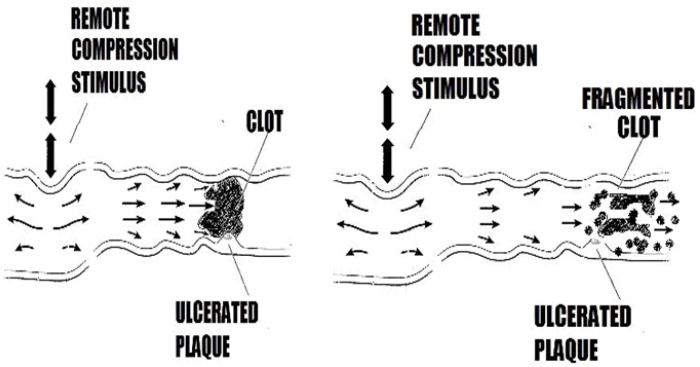

Figure 14: A diagrammatic image, Figure (Left), of a coronary thrombosis upon a ruptured plaque whereby a compression stimulus is applied to the thrombosed coronary far upstream from the clot, which thereby sends pressure waves (arrows) downstream to bombard and deform the clot. Figure (Right) shows how the pressure flow waves eventually fragment the clot into smaller pieces, thereby rendering reflow.

It must also be re-emphasized that, as cited earlier, low-frequency mechanical stimulation (yielding stress, strain, and increased shear stresses to endothelial cells) is known to carry potent vasodilatory capabilities, particularly evident when applied to arteries in a state of heightened vascular tone or spasm.

This holds relevance because studies from the Cath Lab in STEMI PPCI have reported that at least 50% of presentations retain a degree of associated localized coronary spasm at the site of acute thrombosis, as shown by instant reflow following administration of intra-coronary nitroglycerine to the infarct vessel [42-44]. Hence, it could be theorized that tTAP, by providing some degree

of mechano-stimulation upon the coronary endothelium and extracellular matrix of an acutely thrombosed, spasming vessel, could provide a localized vasodilatory effect which would (like a dose of intra-coronary nitroglycerine), promote initial reflow (see Figure 15).

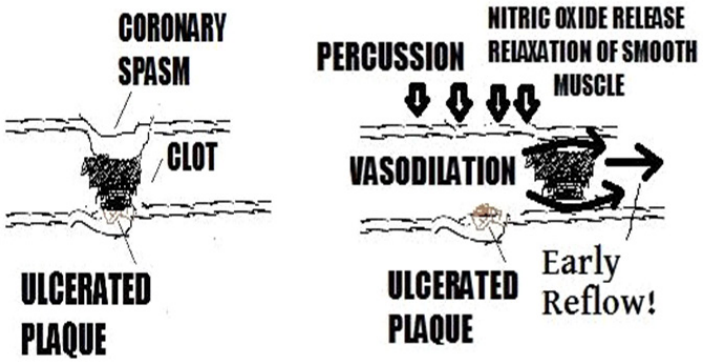

Figure 15: A diagrammatic rendering of (Left) a fresh platelet aggregate accompanied by coronary spasm, a common feature in STEMI, which occludes the vessel. Image (Right) shows how percussive stimuli upon the endothelium from tTAP could promote relaxation of the spasming artery to promote reflow.

Prospective Safety Concerns

Worsening of Aortic Dissection

Probably the most significant safety concern regarding the application of tTAP in the face of new onset symptoms suspicious for heart attack, even with a confirmation of ST elevation, is that it would be unknown for certain whether the patient may be having, instead, or concomitantly, an acute aortic dissection.

Indeed, the sudden surges and spikes in blood pressure that would be produced by Valsalva-like coughing in a tTAP procedure could most certainly greatly increase wall tension and thereby pose some increased risk of worsening an acute dissection. The incidence of aortic dissection in the 60 – 80 age group is 0.009% [45], compared to the risk of STEMI, which is ~ 0.05% [46] (hence the STEMI occurrence is only a factor of 5 greater), so the aortic dissection question in the acute chest discomfort patient cannot be ignored.

However, importantly, according to the literature, once ST elevation is shown in conjunction with acute chest discomfort, the likelihood that the event is due to an acute coronary thrombotic event becomes far greater than that of aortic dissection. Indeed, ST elevation as a consequence of dissection, while it does sometimes occur (most often involving the RCA), is actually an exceedingly rare occurrence expected in less than 1% of dissection cases [47], hence substantially mitigating the concern.

To absolutely negate the dissection issue, it could conceivably be a requirement to pre-screen any prospective user of tTAP to ensure their aorta is of a safe luminal size with therefore low to zero risk for dissection (i.e., perhaps < 45 mm in diameter), which is a highly conservative safety guideline used in pilots [48]).

Possible Increased Risk of Arrhythmia, including Commotio Cordis

While Koiwa and his associates did not, in his many writings, mention any concerns that chest wall administered tDTV may increase the risk of arrhythmia (and this included the study of ischemic LV [26,27]), the risk of potentiating ventricular arrhythmia during a much more powerful and indiscriminately applied tTAP procedure, particularly in a STEMI patient, remains completely unknown. However, the hope of tTAP is that it would work quickly (perhaps within seconds!) and thereby limit ischemic burden to the myocardium, which one would surmise should diminish rather than add to the risk of sudden death.

Nevertheless, ventricular arrhythmia is a dire worry for any heart attack victim, so if tTAP were ever to be tested in a STEMI population, certainly, immediate availability of an AED would be a reasonable safety aspect for any clinical trial.

It is also worth mentioning that while extremely rare, percussion to the chest wall, such as occurring during high-speed projectiles striking the chest of athletes, has caused the incidence of pulseless cardiac arrest in otherwise healthy individuals, an effect called "Commotio Cordis" [49]. Commotio Cordis is again extremely rare, but it is unknown if the effect could be heightened or made more prevalent in the face of severe ventricular ischemia; hence, this is another safety issue that would require vetting.

A Potential Burden to Myocardial Cross-bridge KKinetics

It is also noteworthy that indiscriminately timed perturbations applied to an ischemic myocardium could pose some risks to the hemodynamic stability of a STEMI victim. Indeed, Koiwa showed in an animal study that vibrations (50 Hz) administered directly to ischemic LV, including during the systolic phase of the cardiac cycle, predictably led to a negative inotropic effect in heart function (leading to a decrease in cardiac output and blood pressure), this likely due to interference in the actin-myosin kinetics of the myocardial sarcomere [50]. Hence, monitoring a patient's blood pressure (hemodynamic stability) would also be an important safety measure in any planned clinical trial involving tTAP for treatment of STEMI, with a sudden drop of twenty points or blood pressure below ~90 mm HG systolic, indicating termination of the procedure.

Again, the clinical hope would be that if tTAP would work to restore coronary flow in a STEMI victim, it would work quickly (again, if we believe the Folts data, perhaps within seconds), so any negative inotropic effects, or introduced risks of arrhythmia, would be short-lived and of low significance.

Increased Workload Demand

Of course, another issue could be that if recanalization is not immediate (say within 5 to 30 seconds), a patient may continue administering tTAP, and perhaps with ever-increasing force and effort (including with faster and harder coughing), where after the extra energy and diminished oxygen consumption from the

procedure may start to become a significantly detrimental factor.

With this regard, the Author must express that tTAP, which is performed while in a sedentary position (seated in a chair, or standing still), contrary to what one may imagine, actually takes very little, almost negligible effort. However, it is recognized that further research should include determining a "time limit" where tTAP, should it not work (i.e., ST's stay up), be discontinued, where perhaps 2 to 3 minutes may be a first best guess. Indeed, according to the Folts Effect, the tapping of the vessel should lead to immediate or near-immediate reflow, within seconds.

Overzealous Applications Leading to Blunt Trauma Self-injury

Severe blunt trauma to the chest wall (such as in car accidents or sports) is known to cause adverse events such as coronary arterial dissection [51-53], damage to the heart valves [54,55], and pericardial effusion, possibly with ventricular rupture [56]. While these events are exceedingly rare, with chest wall impacts of significantly greater force than what would be recommended in tTAP, they are still valid safety concerns that would require vetting, and may pose some risks, especially should a patient overzealously apply tTAP, such as when / if symptoms do not immediately subside.

For this reason, there is additional work to be done to establish safety guidelines for what would be deemed as a "safe yet effective" level of force, particularly delivered by fist strikes to the anterior chest wall. Safety studies could be performed in healthy volunteers, where the strength of fist strikes could be monitored by a hand-held accelerometer/force meter, and resultant penetration of forces could be measured, perhaps, again by m-mode echocardiography or via a transesophageal accelerometer.

Notably, the sports of boxing, wrestling, and karate include blows to the chest wall, and for the most part, these athletes leave the ring just fine.

Increased Clotting in view of Clearing the Ulcerative Plaque

It bears repeating that it is presently unknown whether clearing an acute thrombotic lesion, which thereby leaves the site of endocardial injury exposed, would be, despite offering early reflow, an overall good thing. Indeed, perhaps this would lead to re-occlusion after re-occlusion, which may increase overall clot burden to the artery, making the artery more difficult to treat once reaching the cath lab?. These questions could only be answered in pre-clinical to clinical research.

Further Studies

In view of further investigation, the Author suggests that tTAP could be studied in large human-sized pigs, where acute coronary occlusion could be instigated by the electric guide-wire technique suggested by Siegel et al. in their canine model of acute mid-LAD infarction [57,58], with electrocardiographic and CT confirmation of occlusion versus recanalization and TIMI flow.

Coronary angiography may not be appropriate to assess STEMI onset and reperfusion in a study of tTAP, as in the hyperacute case (when a clot is fresh, and still weakly bound to its endothelial surface), even the force of intra-coronary dye injection could plausibly lead to reflow.

A massage therapist (with a force meter, to standardize force delivered) could be used to assess effectiveness for chest wall and upper back percussions, and perhaps stimulation of the spinal cord [59] could be used to invoke coughing in the animal, hence completing the tTAP triad of therapy.

It would be of great value to see firstly if tTAP works (relieves ST elevation and offers initial recanalization of an acutely occluded vessel), and, of equal importance, to investigate what happens immediately afterwards, in view of rates of re-occlusion and establishment of TIMI flow.

Conclusion

The hypothesis of striking one's chest like King Kong while having a STEMI may be dangerous, mad science, and this idea, unless it were to be properly tested as safe and effective therapy, should best be sequestered from the general public.

That said, it may work to offer initial recanalization of a hyperacutely thrombosed epicardial artery prior to arrival of EHS, and could work within seconds, so maybe that would be a good thing.

Hence, the assessment of whether to lift a finger (or in this case, "raise a fist") to investigate this new hypothesis in STEMI therapy remains entirely open for debate.

Affiliation Details

The Author of this manuscript is the President and CEO of Ahof Biophysical Systems Inc., a private cardiovascular research and development corporation, located in Vancouver, B.C., Canada. ABS Inc. provided the funding and resources for this study.

Ethical Statement & Informed Consent

The experiments outlined in this report were developed and performed in a private lab setting (Ahof Biophysical Systems Inc., Vancouver, B.C., Canada).

The test subject (the Author, AH) who self-delivered and received echocardiography in documentation of the penetration studies, and who collected and donated blood for the in vitro experiment, gave informed consent to both / all studies which had been approved by ABS Inc's Institutional Review Committee in accordance with the U.S. Department of Health & Human Services Basic Policy for Protection of Human Research Subjects.

Conflict of Interest

The Author has recently filed a Patent Application regarding the novel features of an ST-segment surveillance wearable, which incorporates the tTAP method for treatment of STEMI.

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