Case Report

ISSN 2639-8486

Cardiology & Vascular Research

Chest Strikes to treat STEMI? Exploring the Platinum Seconds of Reperfusion

Andrew K Hoffmann*

Ahof Biophysical Systems Inc., Canada, USA.

***Correspondence:**
Andrew K. Hoffmann, Ahof Biophysical Systems Inc., Canada, USA.

Received: 20 Sep 2025; **Accepted:** 28 Sep 2025; **Published:** 04 Oct 2025

Citation: Hoffmann AK. Chest Strikes to treat STEMI? Exploring the Platinum Seconds of Reperfusion. Cardiol Vasc Res. 2025; 9(2): 1-7.

ABSTRACT

ST Elevation Myocardial Infarction is a deadly disease, and opening the acutely thrombosed epicardial coronary vessel quickly with good reflow, are the main determinants for a positive outcome. This paper outlines a new first line, emergency treatment hypothesis, whereby a patient, once experiencing symptoms of a heart attack, preferably following confirmation of ST Elevation by a surveillance wearable, would, along with a 911 call, immediately begin beating their chest to the left and right of the sternum. With this, the patient could also simultaneously provide deep Valsalva-like coughing, and even more preferably, while comfortably seated or standing fling their backside repeatedly against a solid back rest, or wall. Data is presented showing how these percussive and compressive maneuvers, herein described as "Thrombo-Agitative Percussion" (TAP), when delivered with enough force can transmit and cause substantial mechanical displacements to the epicardium of the heart, where the coronaries are located. Also presented is evidence from an open canine model showing how gentle tapping or shaking of a hyper-acutely thrombosed coronary artery (while the clot is still a loosely bound, weakly adherent platelet aggregate) is known to lead to immediate and complete reflow to the vessel. Hence is hypothesized that TAP, by causing an agitative, percussive, shaking and compressive effect of the coronary vessels, could quite plausibly lead to immediate to near-immediate reflow (within seconds) to the culprit vessel. There are many potential added risks to TAP in a STEMI patient, which are discussed in this paper.

Keywords

STEMI, Thrombosis, Thrombolysis, Vibro-acoustic therapy, Percussion therapy, Diastolic timed vibration.

Introduction

Methods providing faster and more complete reperfusion in ST Elevation Myocardial Infarction is a subject of great interest, with resolution of the ST-segment an indicator for reflow and better clinical outcomes [1].

Our group has worked on applying low-frequency, infrasonic to sonic Diastolic Timed Vibration (DTV) to the chest wall in STEMI, to provide agitation and improve thrombolytic drug mixing to clear the clot, and by relaxing the otherwise stiff, ischemic myocardium, improve trans myocardial blood flow in the prevention of the slow to no-reflow phenomenon [2-8].

Indeed, DTV has been shown clinically to penetrate through the chest wall to improve diastolic relaxation [9], enhance coronary blood flow [10-12], and improve cardiac output by the Frank-Starling mechanism [13,14], all solid factors for a reasonable first line STEMI reperfusion system. Moreover, infrasonic to sonic frequency vibration in general has been shown to be useful to break apart blood clots [15-17], promote a local vasodilatory effect [18-22], and accelerate thrombolysis [23], including across a chest wall-sized tissue barrier [4].

After years of frustration of never being able to fund research to test DTV for STEMI, it occurred to the Author, that a similar but more simplified approach may be in order…

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

patient could do it themselves, ending STEMI once and for all! There are, of course, a myriad of questions and concerns relating to the prospective safety and efficacy of this seemingly aggressive, almost "violent" approach.

Thrombo-Agitative Percussion (TAP)

Herein is introduced a new hypothesis in first-line STEMI care, to promote initial reflow to the patient prior to arrival of Emergency Health Services (EHS). The therapy, called transthoracic "Thrombo-Agitative Percussion" (TAP), consists of patient self inflicted, forceful, 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 (~ 1 slam per two seconds, or 2 to 3 slams / second with the help of a bystander) commencing at the very first suspicion of anginal-like symptoms. The hope of these thrombo-agitative maneuvers is to facilitate initial clearance of hyperacute, new-onset coronary thrombosis.

Most preferably, a prospective TAP patient would be wearing an ST-segment monitoring surveillance system (with ECG leads placed on the skin by a wearable shirt or an undergarment, or alternatively applied subcutaneously), so initiation of the percussive therapy could be considered only following an alert from their device indicating new ST elevation.

The confirmation of ST elevation is an important safety feature for TAP, as it is a highly specific marker for an acute coronary occlusion, in particular versus aortic dissection, where TAP (especially in view of the Valsalva-like coughing which would lead to wild spikes in blood pressure) would be contraindicated. However, notably the incidence of aortic dissection with *ST – elevation* is exceedingly rare, occurring in only about 1% of dissection cases [24], hence substantially negating the concern. Moreover, it should be emphasized that TAP should work almost instantaneously (within seconds), if it is to work at all, and to negate all concerns to the dissection question, prospective TAP patients could be pre-screened for their aortic sizes, to prove they are of "safe" diameter (< 45 mm), which is a conservative safety threshold adopted in pilots.

The therapy works by providing agitative mechanical forces across the thoracic cavity to cause resultant gentle but persuasive serial compressions and decompressions, as well as percussions and shaking of the epicardium of the heart, including the coronary arteries, in view to safely dislodging, disaggregating, and clearing a new-onset platelet aggregate.

The therapy theoretically takes 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.

See Figure 1, 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 TAP can be seen by clicking on the following link: https://youtu.be/yMJao7FUSZw.

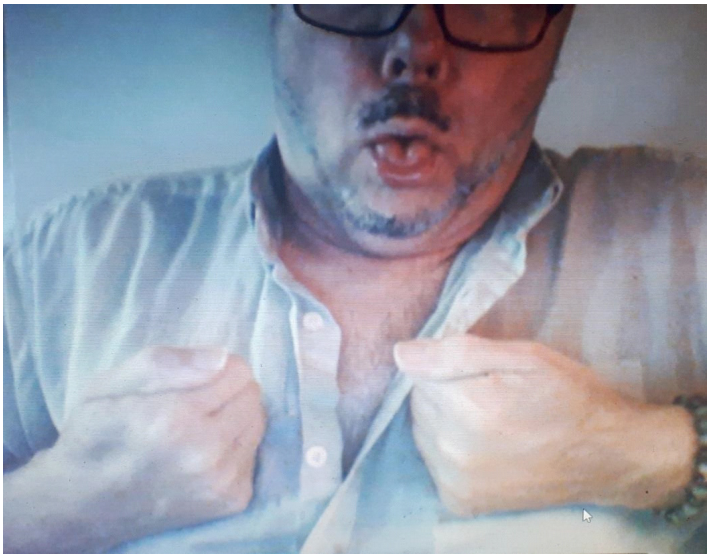

Figure 1: A model (the Author) is shown administering self-administered transthoracic Thrombo-Agitative Percussion (TAP) via rapid, forceful bilateral fist strikes to the left and right of the sternum, deep coughing, and upper back slams (not seen in the figure).

But all this said, how can we be certain that mechanical agitation applied to the external surface of a hyper-acute thrombosed coronary artery, would even lead to reflow?

The "Folts Effect"

The most relevant data to support that TAP would lead to near immediate to immediate re-canalization and reflow in STEMI, 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", "pinching", "tapping" or "shaking", of the external surface of a hyper- acutely thrombosed coronary artery with a surgical instrument, 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 [25,26].

See Figure 2, which diagrammatically shows the set up of the Folts' open canine coronary thrombosis flow model.

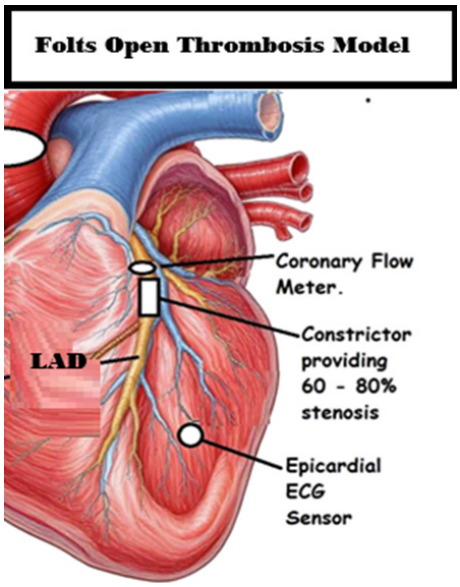

Figure 2: A diagrammatic rendering of the setup of the Folts' open, coronary thrombosis flow model. A constrictor is placed in this case on the proximal LAD, which causes a site of 60 – 80% stenosis with endocardial injury, which causes platelet aggregations to form, and spontaneously release, where the blood flow rate was continuously monitored by a flow meter.

Dr. Folts reported that at times the studied coronary artery in his model would become untowardly thrombotically occluded causing a STEMI, whereby, the experimenter (presumably Dr. Folts) would gently "shake" or "poke" [25,26] at the vessel or constrictor, 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 in the Folts model, and once an artery was recanalized, blood flow rate was generally fully restored to baseline.

See Figure 3, which shows an example of a Folts flow meter readout where zero flow occurred (in this case, the left circumflex artery had become thrombotically occluded), whereafter the experimenter had "shaken loose" the constrictor, which reportedly led, reliably to immediate and complete flow restoration. It is notable that platelet aggregations (which slowed flow), even following an acute occlusion almost always led eventually to "spontaneous release" (shown by "y"), so once a vessel became occluded (causing a STEMI), this did not mean necessarily another occlusion would take place, at least not soon after.

But could TAP maneuvers sufficiently penetrate to cause an adequate comparable mechano-agitative stimulus, similar to direct tapping upon a newly thrombosed human coronary artery to reproduce the Folts' effect in promotion of reflow?

Chest Wall Fist Strikes

To prove that powerful chest wall fist strikes just leftward the sternum penetrate to reach 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 4, to see what mechanical effects to the LV myocardium could be observed.

As can be seen in Figure 4, 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 therefore hypothesized that fist

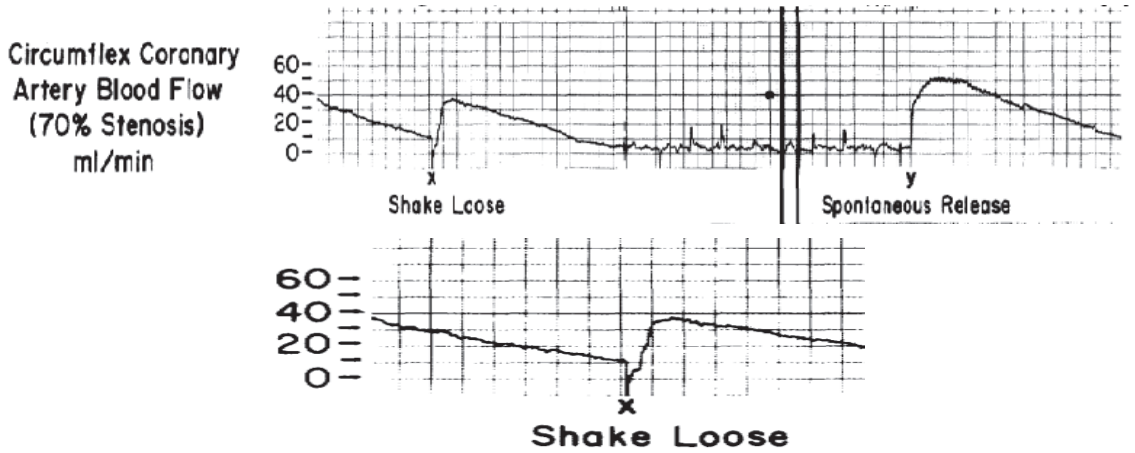

Figure 3: (Top) The "Folts Effect". A flowmeter readout from the Folts coronary thrombosis model indicating that the left circumflex artery had become thrombotically occluded with zero flow - shown at "X", whereby the Experimenter simply "shook loose" the constrictor, which immediately led to normalized flow. 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).

strikes leftward the sternum may assist reperfusion in anterior / anterior septal STEMI.

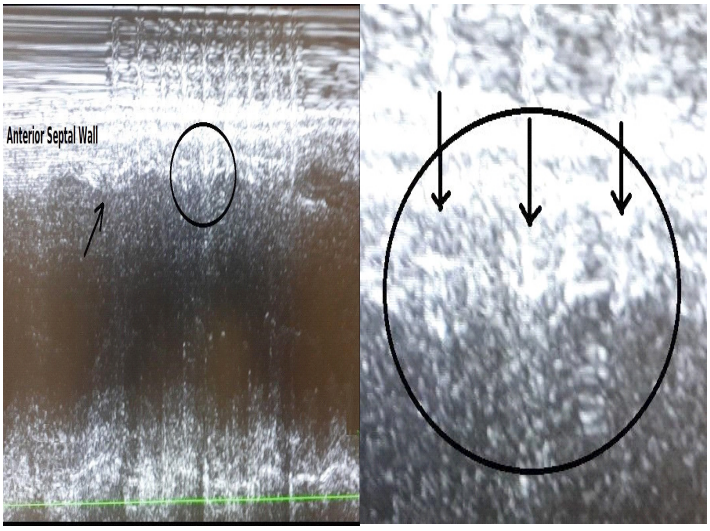

Figure 4: M mode figure (Left) showing the LV during forceful fist strikes to the left sternal margin of the chest wall (starting at the arrow). Figure (Right) shows a zoomed-in view of the anterior-septal wall to better show the resultant compressive displacements. Image taken with permission from the Echo-lab, False Creek Healthcare Center.

On the other hand, to show how chest-wall fist strikes just rightward the sternum penetrates to reach the Right Ventricle (RV - home of the proximal to mid-RCA and RV marginal branch), see Figure 5, which shows an M-mode of the RV free wall, along with chest wall fist strikes delivered just rightward the sternum.

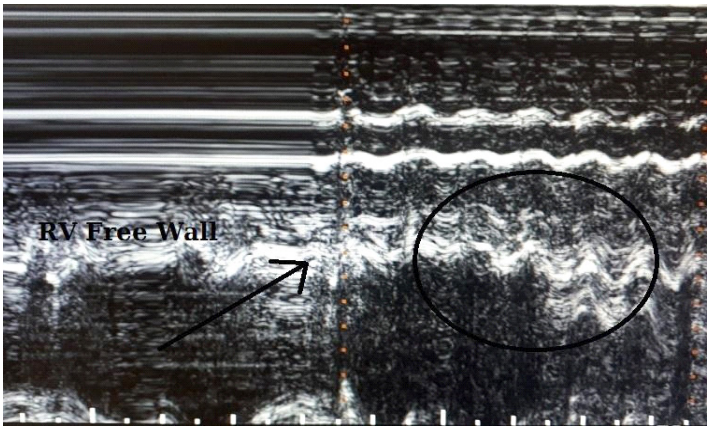

Figure 5: M mode showing the RV before and during fist strikes (starting at the arrow). Image acquired and taken from the Echo-lab, False Creek Healthcare Center.

Referring to Figure 5, the commencement of fist strikes along the right sternal margin of the chest (shown by arrow) is accompanied by marked vertical echoic compressions of the RV free wall (circled), which underlies, supports, and is perfused by the proximal to mid-RCA and RV marginal branch.

It is therefore 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 during deep coughing, to see how it may affect the heart (see Figure 6).

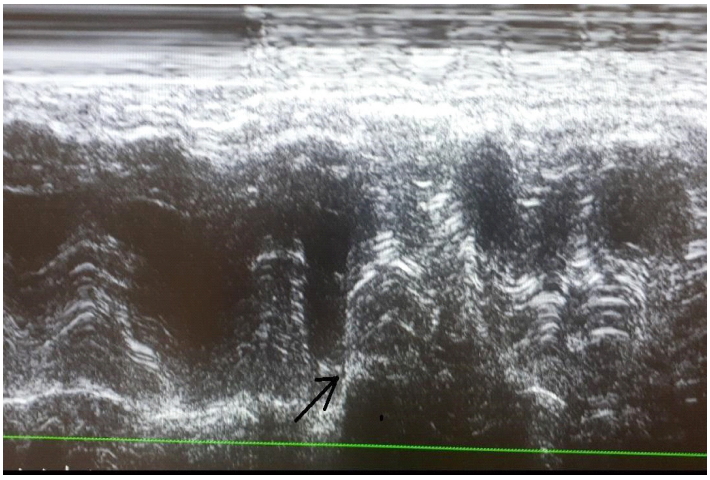

Figure 6: Effect of Deep Coughing Upon the Heart. M mode showing the LV (anterior septal wall - top, posterior wall - bottom) with three consecutive deep coughs (starting at the arrow). Image taken with permission from the Echo-lab, False Creek Healthcare Center.

Referring to Figure 6, 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 degree to the anterior-septal wall.

Indeed, deep coughing causes circumfrential compressions to the heart, which would reinforce stimulation to the entirety of the coronary tree.

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 flinging his upper back, back and forth against a solid wall while comfortably seated on a stool (see Figure 7).

Referring to Figure 7, 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 self-inflicted upper-back slams (marked by the arrow). It is thereby hypothesized that upper back slams could provide serial, repetitive compressive stimuli more-so to the posterior epicardial surfaces of the heart, including the PDA, but also of lesser but still some effect to the anterior aspect of the heart, which could affect the LAD,

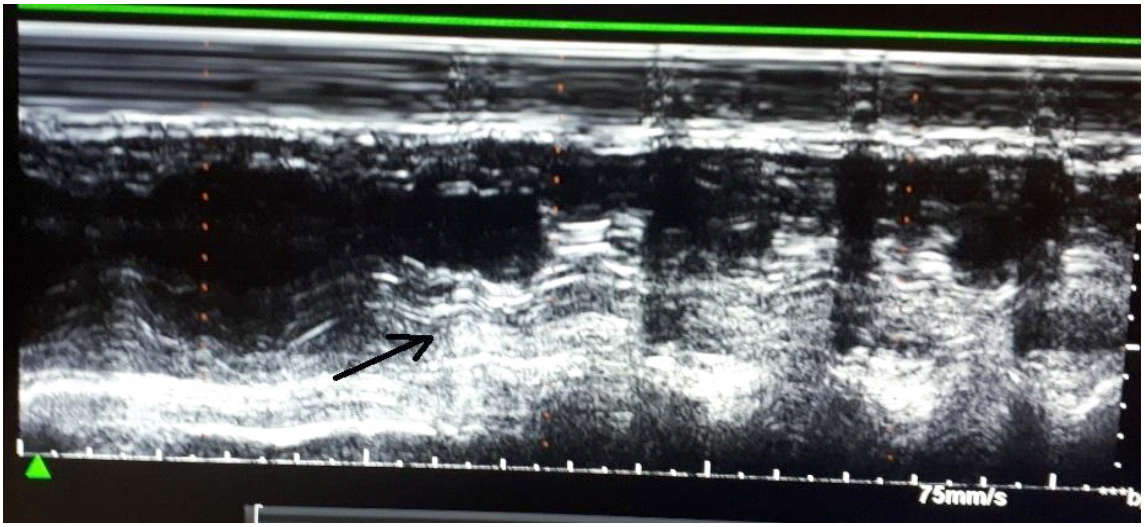

Figure 7: Effect of Upper Back Slams Upon the Heart. M mode showing the LV (anterior septal wall - top, posterior wall - bottom). The arrow indicates the beginning of self-administered upper back slams. Image acquired from the Echo-lab, False Creek Healthcare Center.

Diagonals and Septal perforators.

Discussion

TAP as a first line treatment for STEMI would be seen by many as crazy, dangerous science that is best forgotten and sequestered from the public. Moreover, even if TAP would work to clear the site of thrombosis and thereby initiate early reflow, would that, necessarily be a good thing?

It has been counterintuitively suggested that TAP, by clearing and leaving exposed the presumed ulcerated plaque, would simply cause re-occlusions which would (even if "re-tapped"), increase clotting, which in turn might untowardly increase overall clot burden (with sticky clots, and platelets moving downstream). This could make matters worse, including making it more difficult to treat the artery with PPCI, upon arrival to the Cath lab.

However, on the other hand perhaps TAP recanalization might decrease overall clot burden, as the platelet aggregations are dealt with incredibly early (literally at the moment the clot becomes occlusive). Also, it is expressly unknown whether a freshly cleared platelet aggregate would disaggregate to such a degree to flow innocently through the distal vasculature (indeed, flow following dispersal of an acute thrombotic occlusion in the Folts model generally went back to baseline, and re-occlusions would not necessarily re-occur).

It is recognized of course, that the chest strikes of TAP, would likely be poorly received in females due to the sensitivity of the breast area, however, far less force would be needed in females to achieve useful transthoracic penetration due to a much thinner chest wall, so this remains an open question. Also, notably, approximately seventy to seventy-five percent of STEMI victims are men [27,28], and perhaps females and those who cannot strike their chests could be advised to limit TAP to upper back claps and deep coughing.

Prospective Mechanism of TAP

The prime mechanism of action of TAP is to provide gentle compressive and percussive forces upon the epicardium, including the coronary arteries, which, in the case of an acute thrombosis, would, vis-à-vis squish and disaggregate a newly formed occlusive platelet aggregate.

See Figure 8, which diagrammatically prophesies a best-case scenario how a new platelet clot may be anticipated to disaggregate and fragment into smaller pieces, as a consequence of epicardial compressions delivered by TAP.

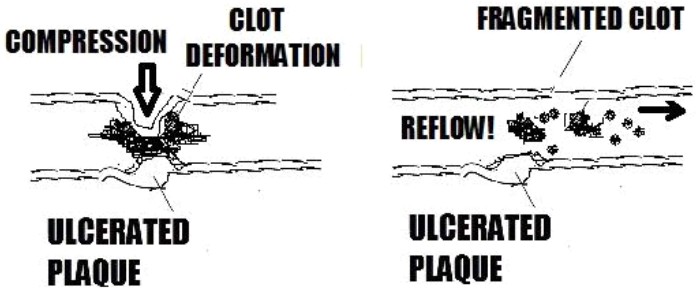

Figure 8: Diagrammatic rendering of (Left) shows how an acutely thrombosed coronary artery may be deformed by an extra-luminal percussive stimulus rendered by tTAP, thereby vis-à-vis "squishing" or "smashing" the clot. Image (Right) shows how the clot, following multiple compressions, has broken up into smaller fragments, thereby rendering initial recanalization of the vessel.

Moreover, according to the Folts data, the mere act of "shaking", or "poking" at a new onset thrombo-occluded coronary artery (without necessarily compressing the vessel) was all that was required to effect immediate and complete reflow. To this, it is notable that infrasonic vibration stimulus to the epi-myocardium (somewhat analogous to serially applied percussive blows) has been shown experimentally to have unique internalized transmission effects, by a tethering effect, along the epimyocardium [29-31] and along

arteries [32].

Hence, even if a TAP-directed percussive force were to reach the heart "off mark" of the thrombus, hitting or compressing a healthy or downstream ischemic aspect of the epicardium or a part of an adjoining artery which is not occluded, the forces could still theoretically transmit to cause a therapeutic shaking response across the epicardium and along the arteries to affect much of the coronary tree.

Also, as referenced earlier, low-frequency impacts to vascular tissue can stimulate an acute vasodilation response, largely due to an endogenous release of nitric oxide. With that, studies from the Cath Lab in STEMI PPCI cases have reported that at least 50% of presentations retain a degree of associated localized coronary spasm at the site of acute thrombosis [33-35]. Hence, it could be theorized that TAP, 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 could also lead to reflow by a separate mechanism.

Prospective Safety Concerns

Here is a brief list of prospective safety concerns regarding a TAP procedure, in view of confirmed ST elevation, all of which would need to be considered and vetted during clinical trials.

- **Worsening of concomitant or misdiagnosed Aortic Dissection.**
  - Pre-screen TAP candidates for normal aortic sizes.
  - Confirm ST elevation prior to TAP
- **Possible increased risk of arrhythmia, including commotio cordis.**
  - Would need a defibrillator or AED handy, in case of any clinical trial.
  - Unknown risk, however, if TAP should work, it could work within seconds. Improving early perfusion in theory should decrease risk of sudden death.
- **A potential burden to myocardial cross-bridge kinetics. May lead to a lowering of systemic blood pressure with decreased cardiac output if TAP is applied for prolonged periods.**
  - Would need to monitor blood pressure for any clinical trial.
  - Again, if TAP should work it would do so within seconds, which should mitigate any significant concerns.
- **Overzealous applications leading to blunt trauma self-injury, or a significant, detrimental and unwarranted increase in workload.**
  - Safety study is needed to assess minimal force required regarding fist strikes to the chest for safe penetration.
  - A smart watch or ring could thereafter house an accelerometer, to provide feedback to the STEMI patient, as to whether to hit "harder" or "softer".

Again, a takeaway point in TAP, is that according to the Folts Data, the therapy in clearing the acutely thrombosed vessel should occur immediately, within seconds, if it is to work at all.

Also, TAP can be performed while sitting comfortably in a chair or stool, and contrary to what one may think, is not overly painful and takes very little energy to perform.

Conclusion

The hypothesis of striking one's chest while having a STEMI may be dangerous, and this idea, unless it were to be properly tested as safe and effective therapy, should be sequestered from the public. That said, it may work to offer initial recanalization of a hyper-occluded epicardial artery within seconds, and few would say that is a bad thing, and could be a game changer for public health. Hence, the assessment of whether to investigate this provocative, new hypothesis in STEMI therapy remains open for debate.

References

1. O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2013; 61: e78-e140.
2. Hoffmann A. Low Frequency Vibration Assisted Blood Perfusion Emergency System. US Patent No. 7,517,328. 2004.
3. Hoffmann A, Yohannes FG. Non-invasive low frequency vibration as a potential adjunctive treatment for heart attack and stroke. An *in-vitro* flow model. J Thromb Thrombolysis. 2008; 25: 251-258.
4. Hoffmann A, Gill H. Diastolic timed Vibro-Percussion at 50 Hz delivered across a chest wall sized meat barrier enhances clot dissolution and remotely administered Streptokinase effectiveness in an in-vitro model of acute coronary thrombosis. Thrombosis Journal. 2012; 10: 23.
5. Khosrow-khavar F, Marzencki M, Tavakolian K, et al. Diastolic Timed Vibrator: Applying Direct Vibration in Diastole to Patients with Acute Coronary Ischemia during the Pre-hospitalization Phase. Autonomous and Intelligent Systems. AIS 2011. Lecture Notes in Computer Science. 2011; 6752: 355-356.
6. Marzencki M, Kajbafzadeh B, Khosrow-khavar F, et al. Low frequency mechanical actuation accelerates reperfusion in-vitro. Biomed Eng Online. 2013; 12: 21.
7. A Uryash H Gill, A Hoffmann. Can Upper Torso Vibro Acoustic Stimulation Treat No Reflow following STEMi-Directed PPCI?. Rationale and Literature Review. 2016; 24: 36-38.
8. A Uryash, H Gill, A Hoffmann. Upper Torso Vibroacoustic Stimulation for Treatment of No-Reflow Following STEMI-Directed PPCI: A Device and Pilot Study Protocol. Shake, Rattle, and Rock n'-Reflow! Cath Lab Digest. 2016; 24: 2-5.
9. Koiwa Y, Honda H, Takagi T, et al. Modification of human left ventricular relaxation by small-amplitude, phase-controlled

mechanical vibration on the chest wall. Circulation. 1997; 95: 156-162.

10. Koiwa Y, Honda H, Hoshi N, et al. The effect of diastolic vibration on the coronary flow rate in the canine heart with ischemia. J Cardiovasc Diagn Procedures. 1994; 12: 110.
11. Koiwa Y. Precordial or epicardial input of phase-controlled minute vibration: effect on the coronary flow rate in regional ischemia. In New Horizons for Failing Heart Syndrome. Edited by Sasayama S. Tokyo; New York. Springer. 1996: 117-130.
12. Koiwa Y, Naya T, Honda H, et al. Diastolic vibration from the precordium increases coronary blood flow in humans. J Cardiovasc Diagn Procedures. 1994; 12: 110.
13. Koiwa Y, Takagi T, Kikuchi J, et al. Diastolic vibration improves systolic function in cases of incomplete relaxation. Circulation. 1992; 86: 1955-1964.
14. Koiwa Y, Takagi T, Kikuchi H, et al. The improvement of systolic function of depressed left ventricle by external vibration at diastole. Tohoku J Exp Med. 1989; 159: 169-170.
15. Hoffmann A, Gill H. Externally Applied Vibration at 50 Hz Facilitates Dissolution of Blood Clots *In-Vitro*. Am J Biomed Sci. 2012; 4: 274-284.
16. Dubrul. Vibrating Catheter, US Patent No. 5,713,848. 1998.
17. Wobser E, Stumpff U. Intragastral disintegration of blood coagula by mechanical vibration. Endoscopy. 1978; 10: 15-19.
18. Lindblad LE, Lorenz RR, Shepherd JT, et al. Effect of vibration on canine cutaneous artery. Heart Circ Physiol. 1986; 19: H519-H523.
19. Ljung B, Silvertsson R. Vibration-induced inhibition of vascular smooth muscle contraction. Blood Vessels. 1975; 12: 38-52.
20. Hudlicka O, Wright A. The effect of vibration on blood flow in skeletal muscle in rabbit. Clin Sci Mol Med. 1978; 55: 471-476.
21. Ichioka S, Yokogawa H, Nakagami G, et al. In vivo analysis of skin microcirculation and the role of nitric oxide during vibration. Ostomy Wound Manage. 2011; 57: 40-47.
22. Maloney-Hinds C, Petrofsky JS, Zimmerman G, et al. The role of nitric oxide in skin blood flow increases due to vibration in healthy adults and adults with type 2 diabetes. Diabetes Technol Ther. 2009; 11: 39-43.
23. Arko F, Davis C, Murphy E, et al. Aggressive Percutaneous Mechanical Thrombectomy of Deep Venous Thrombosis. Arch Surg. 2007; 142: 513-519.
24. Zhu Q, Tai S, Tang L, et al. STEMI could be primarily presentation of acute aortic dissection. Am J Emerg Med. 2017; 35: 1713-1717.
25. Folts J, Gallagher K, Rowe G. Blood Flow Reductions in Stenosed Canine Coronary Arteries: Vasospasm or Platelet Aggregation?. Circulation. 1982; 65: 248-254.
26. Folts J. An *In Vivo* Model of Experimental Arterial Stenosis, Intimal Damage, and Periodic Thrombosis. Circulation. 1991; 83: 3-14.
27. Kuehnemund L, Koeppe J, Feld J, et al. Gender differences in acute myocardial infarction - A nationwide German real-life analysis from 2014 - 2017. Clin Cardiol. 2021; 44: 890-898.
28. Ziwei Xi, Hong Qiu, Tingting Guo, et al. Contemporary sex differences in mortality among patients with ST segment elevation myocardial infarction: a systematic review and meta-analysis. BMJ Open. 2022; 12: e053379.
29. Koiwa Y, Hashiguchi R, Ohyama T, et al. Measurement of instantaneous viscoelastic properties by impedance-frequency curve of the ventricle. Am J Physiol. 1986; 250: H672-H684.
30. Hashiguchi R, Koiwa Y, Ohyama T, et al. Dependence of instantaneous transfer function on regional ischemic myocardial volume. Circ Res. 1988; 63: 1003-1011.
31. Smith D, Ishimitsu T, Craige E. Mechanical vibration transmission characteristics of the left ventricle implication with regard to auscultation and phonocardiography. J Am Coll Cordiol. 1984; 4: 517-521.
32. Farber JJ, Purvis JH. Conduction of cardiovascular sound along arteries. Circ Res. 1963; 12: 308-316.
33. Dalen JE, Ockene IS, Alpert JS. Coronary spasm, coronary thrombosis, and myocardial infarction: a hypothesis concerning the pathophysiology of acute myocardial infarction. Am Heart J. 1982; 104: 1119-1124.
34. Oliva PB, Breckinridge JC. Arteriographic evidence of coronary arterial spasm in acute myocardial infarction. Circulation. 1977; 56: 367-374.
35. Maseri A, L'Abbate A, Baroldi G, et al. Coronary vasospasm as a possible cause of myocardial infarction. N Engl J Med. 1978; 299: 1271-1277.

© 2025 Hoffmann AK. This article is distributed under the terms of the Creative Commons Attribution 4.0 International License