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Negative Sequence Voltages in Spontaneous Ventricular Fibrillation or Tachycardia*

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Abstract*—The patho-physiology of initiation of ventricular fibrillation or tachycardia is not known. Could it be possible that etiology of tachyarrhythmia is hidden in the territory other than medicine? Can an abnormal rotation or an abnormal slow conduction of the T-loop explain the genesis of ventricular fibrillation or tachycardia? Total of 473 emergency Frank vectorcardiographic tracings were recorded by one physician from 148 patients with acute myocardial infarction. All 137 (93 %) out of 148 patients had abnormal rotation of the T-loop. Twenty-three of these patients developed ventricular fibrillation or tachycardia. Twenty (87%) of these 23 patients had abnormal rotation of the T-loop. Out of the total of 148 patients, there were 146 patients (99 %) with abnormal slow conduction of the T-loop. In conclusions, the heart has inherited similar problems as electrical power generators. The abnormal rotation types (figure-of-eight or clockwise) of the T-loop from the ventricles, which are similar to the partial and complete negative sequence in electrical power engineering, are similar to the P-loop from the atria documented in the literature. Based on negative sequence voltages' concept in electrical power engineering , a new theory of ventricular fibrillation, tachycardia, or ventricular premature contraction has been proposed. From findings in diseased hearts and malfunctioning generators, the negative sequence must inevitably be recognized as a new law in physics and medicine. Physicians' routine management of ventricular fibrillation or tachycardia is wrong in bio-electricity and bio-mechanical aspect. Electrical power engineer with vectorcardiographic training should be the choice for arrhythmia research.

***Index Terms*—Negative sequence voltage; Vectorcardiography; Ventricular fibrillation; Ventricular tachycardia..**

"*Truth is stranger than fiction.*"---Lord Byron 1788-1824

***"Without theory, practice is only routine, governed by the force of habit"*---Louis Pasteur 1822-1895**

I. INTRODUCTION

The incidence of ventricular fibrillation (VF) at the time of cardiac arrest was estimated to be 60-70% in all patients and 80-85% in the cases with probable heart disease in 14,065 cases of out-of-hospital cardiac arrest [1]. Ventricular tachycardia (VT), VF, and ventricular premature contraction (VPC) are very frequently encountered by physicians caring for patients with cardiac arrest. However, the patho-physiology of VT or VF initiation is not known. Physicians depend on their diagnostic armamentaria, which are similar to radar systems, to make a diagnosis or to do research. Could it be possible that those armamentaria could miss some phenomena, which are the missing links to explain ventricular tachyarrhythmia? Furthermore, without knowing the true etiology of ventricular tachyarrhythmia, how can we know whether our therapy is adequate in treating our patients under the guidance of biostatistics? As the late Dr. Robert Mendelson wrote ". . . the greatest danger to your health is the doctor who practices Modern Medicine. I believe that Modern Medicine's treatments for disease are seldom effective, and that they're often more dangerous than the diseases they're designed to treat . . ." [2]

During an emergency there are too many obstacles to obtaining human data. However, atrial study has established evidence of abnormal type of figure-of-eight or clockwise rotation of the P loop, which is linked to the partial or complete negative sequence in physics [3]. There are substantial training, research-funding, and career-development deficiencies in patient-oriented, physiological and patho-physiological research [4]. Vectorcardiographic studies of the T-loop rendered thousands of the QT dispersion studies of the electrocardiogram (ECG) since 1990 obsolete [5]. Can a similar history be repeated in the study of VF, VT and VPC?

Except for disciples of Professor Alberto Benchimol [6],[7]in the literature there have been very few serial emergency vectorcardiographic studies of the T loops in patients with acute myocardial infarction. The marks and inscription directions of the T-loop in the vectorcardiogram are equivalent to the footprints and footpath of electrical trails. They may help us to understand the arrhythmogenesis in patients with VT or VF.

II. MATERIALS AND METHODS

One hundred and seventy eight consecutive patients with suspected coronary heart disease within 24 hrs of their admission into the Coronary Care Unit enrolled in the serial emergency vectorcardiographic studies Phase I Study (1979-1983) of acute myocardial infarction. On admission to the Coronary Care Unit through the Emergency Service, each

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patient was given electrocardiographic and vectorcardiographic examinations, which were repeated at intervals of 24hrs if the patient's condition was conducive to doing so. All the ECGs and vector cardiograms were recorded by the first author according to orthodox methods of medical research [8], and read by the other author without any knowledge of the patients. The investigation conforms to the principles outlined in the Declaration of Helsinki, and informed consent was obtained from each patient. Detailed methods are published in the literature [3].

The vectorcardiographic QRS-loop diagnostic criteria for different location of myocardial infarction were according to the literature [9]. The left ventricular enlargement is in the Horizontal plane: (1) The magnitude of the maximum QRS vector is >2.2 mV below the age of 50 years and 1.8 above this age; and (2) the angle of the maximum T vector is over + 70 degree. The diagnosis of right ventricular enlargement is in the Horizontal plane: (1) Sx/Rx value is >0.4; (2) the clockwise inscription or figure-of-eight of the T-loop; (3) the angle of the maximum T vector is less than 0 degree. The diagnosis criteria of complete right bundle branch block are (1) the terminal appendage of the QRS loop is oriented to the right-anterior-superior or the right-anterior-inferior octant. The duration of the terminal appendage is $\geq$ 0.03 second and the appendage inscribes slowly; and (2) the duration of the QRS loop is prolonged to $\geq$ 0.12 second. For diagnosis of incomplete right bundle branch block is the same as complete right bundle branch block but the duration of the QRS loop is prolonged, but it is < 0.12 second. Left anterior hemi block diagnosis is in the Frontal plane: (1) most of the QRS loop is located in the left-superior quadrant due to the fact that spatial QRS loop is in the left-posterior-superior octant; (2) the QRS loop is in anti-clockwise rotation; (3) the initial 0.01 and 0.02 second QRS vectors are pointed to the right-inferior or left-inferior direction; (4) total duration of the QRS loop seldom exceeds 0.10 second. The diagnosis of ventricular repolarization abnormality was determined by an abnormal T-loop morphology [10], sense of inscription and fast or slow conduction of the loop. Non-sustained ventricular tachycardia is defined as $\geq$ 3 or more consecutive beats arising below the atrioventricular node with a rate >120 beats/min and lasting less than 30 s. Vectorcardiographic diagnosis of VPC are the QRS complex in bundle branch block pattern and initial delay ($\geq$ 20 msec) of the QRS loop [11].

In a normal subject the T-loop inscription direction should be anti-clockwise in the three planes of the vectorcardiogram, but it can be clockwise in the Frontal plane. The normal T-loop has efferent limb always inscribed slower than the afferent limb [10].

III. RESULTS

A total of 473 emergency vectorcardiographic tracings were recorded for 148 patients with acute myocardial infarction. Their age was 61$\pm$ 9.8 years, and 129 were male, 19 female. One hundred and thirty six patients had $\geq$ 2 tracings during the acute myocardial infarction. Initially, 127(86%) of the 148 patients had abnormal inscription direction (AID) of the T loop in the first tracing. One patient had linear shape of the T-loop in both Horizontal & Frontal planes. During serial follow-up, 137 (93 %) of the 148 patients had AID of the T loop. Twenty three (16%) (Table I, II) of the 148 patients developed VT or VF. Twenty (87%) among these 23 patients had AID of the T-loop. Twelve of the 148 who died had no VT or VF, but died due to other reasons such as cardiac rupture. Eight patients (No.4,5,9,12,13, 14,21,23) who received emergency vectorcardiographic examination after cardioversion still had a clockwise T-loop in at least one plane of either Horizontal or Left Sagittal plane or both, except two (No. 4, 12), who returned to normal. Two (No. 13, 23) of the 8 later died. Seven of the 148 had a non-sustained ventricular tachycardia episode, and only one patient (No. 1) later developed VT. One hundred and eight (73 %) of the 148 patients had ventricular enlargement (left, right, or both). Of the 23 patients who developed VT or VF, 14 patients (61%) had chamber enlargement. Seven (50%) of these 14 patients who developed sudden death. However, in nine patients without chamber enlargement but who also developed VT or VF, seven (78%) developed sudden death. There were 146 patients (99 %) among the total 148 with abnormal slow conduction (ASC) of the T loop. Only one patient among 23 patients with VT or VF developed Torsade de point, which was abolished by first thump.

A patient with negative sequence in the inscription direction of the T-loop in the Frontal and Left Sagittal planes in the vectorcardiogram is demonstrated in Fig. 1. Figures 2 and 3 show the orthogonal and 12-lead ECG tracings of the patient No. 16; he developed a VT before therapy and cardiac arrest.

Table I. Data of 23 patients with ventricular tachyarrhythmia.

Pt	Age/Sex	Infarction	Enlargement	EVCG before or after event	Note	Killip/outcome
1	52/M	ExtAntLat	LVE	2.5 hrs before	SRVT/VT	I/alive
2	77/F	ExtAntLat	LVE	8 days before	VT	I/dead
3	85/F	ExtAnt	LVE	65.5 hrs before	VF	II/dead
4	53/M	ExtAntLat	LVE	2 hrs after	VF	IV/alive

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5	59/M	ExtAnt	BVE	8 days after	VT	I/alive
6	63/M	InfPost	LVE	16.5 hrs before	VF	III/alive
7	63/F	InfPost	---	90.5 hrs before	VT	IV/dead
8	57/M	LVsubendo	LVE	82.5 hrs before	VT/VF	IV/alive
9	59/F	Inf	LVE	24 hrs after	VT/VF	I/alive
10	68/M	InfAnt	---	26 hrs before	VT/VF	II/dead
11	52/M	AntSep	LVE	6.5 hrs before	VT	I/alive
12	77/M	InfAnt	---	5 days after	VT	I/alive
13	78/F	InfAntLat	---	7.5 hrs after	VF	II/dead
14	47/M	InfAntLat	LVE	24 hrs after	VT/VF	I/alive
15	61/M	ExtAntLat	---	4.5 days before	VT/VF	IV/dead
16	58/M	InfAnt	LVE	15.5 hrs before	VT	IV/dead
17	54/M	ExtAntInf	---	14.5 hrs before	VT/VF	I/alive
18	52/M	Ant	---	47 days before	VT	II/dead
19	54/M	InfAnt	---	5 days before	VF	III/dead
20	60/M	InfAnt	LVE	34.5 hrs before	VT	III/dead
21	64/M	Inf	---	3 hrs after	VT	II/alive
22	55/F	Inf	LVE	6.5 hrs before	VT/VF/Tor	IV/dead
23	72/F	Inf	LVE	15 hrs after	VT	IV/dead

Abbreviations: Ant, anterior; BVE, bi-ventricular enlargement; EVCG, emergency vectorcardiogram; Ext, extensive; F, Female; Inf, inferior; Lat, lateral; LV, left ventricle; LVE, left ventricular enlargement; M, Male; Post, posterior; Pt, patient; Sep, septal; SRVT, short run ventricular tachycardia; Subendo, subendo myocardium; Tor, Torsade de point; VF, ventricular fibrillation; VT, ventricular tachycardia.

Table II. Serial vectorcardiographic findings of the T loop in 23 patients with ventricular tachyarrhythmia.

Pt	SC/H	SC/LS	SC/F	ID/H	ID/LS	ID/F	Ref. to
1	N (Mid&Term)	N (Mid&Term)	N (Mid&Term)	ACW	ACW	CW(8)	1st EVCG
2	Term (N)	Term	Term	CW(ACW)	ACW	CW	last EVCG
3	Term (Mid&Term)	N (Mid&Term)	Mid&Term	ACW	ACW	CW	last EVCG
4	N (Mid&Term)	Mid(Mid&Term)	N(Mid&Term)	ACW	ACW	CW(ACW)	1st EVCG
5	Mid&Term	Mid&Term	Mid&Term	CW	ACW	CW	only EVCG
6	Mid&Term	Mid&Term	Mid&Term(Term)	CW(8)	CW(8)	ACW(8)	1st EVCG
7	Mid&Term	Mid&Term	Mid&Term	CW	CW(8)	ACW	last EVCG
8	Term(Mid&Term)	Term(Mid&Term)	N(Mid&Term)	ACW	CW	CW(ACW)	last EVCG
9	Mid&Term	Mid&Term	Mid&Term	ACW(CW)	ACW(CW)	ACW(CW)	1st EVCG
10	Mid&Term	Mid(Mid&Term)	Mid&Term	CW(8)	CW	ACW(CW)	last EVCG
11	Mid&Term	Mid&Term	Mid&Term	CW	ACW(CW)	ACW	1st EVCG
12	Mid&Term	Mid&Term	Mid&Term	ACW	ACW	CW(ACW)	1st EVCG

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13	N(Mid)	Mid&Term(Term)	N	CW	ACW	ACW	last EVCG
14	Mid&Term	Mid&Term(N)	Mid&Term(Term)	CW	CW	ACW	1st EVCG
15	Mid&Term	Mid&Term	Mid&Term	CW(8)	CW	ACW	last EVCG
16	Mid&term	Term	Term	CW	CW	ACW	only EVCG
17	Mid&Term	Mid&Term	Mid&Term	CW(ACW)	CW	ACW(CW)	1st EVCG
18	Mid&Term	Mid&Term	Mid&Term	CW	ACW	ACW	last EVCG
19	Mid&Term	Mid&Term	Mid&Term	CW(ACW)	CW(8)	ACW(CW)	last EVCG
20	Term(Mid&Term)	Mid(Mid&Term)	Mid&Term	CW	CW	ACW	last EVCG
21	Mid&Term	Mid&Term(N)	Mid&Term	CW	CW	ACW	1st EVCG
22	Mid&Term	Mid&Term	Mid&Term	CW	CW	CW	only EVCG
23	Mid&Term	Mid&Term	Mid&Term	ACW	CW	CW	only EVCG

Note: Words within brackets indicate change appearing during follow-up vectorcardiographic tracing. Ref. to suggest which vectorcardiogram tracing is closest to the ventricular tachyarrhythmia event. For example in patient No. 1, the first EVCG has ACW of the T-loop in the H&LS planes and CW of the T-loop in the F plane. Slow conduction (close marks) of the T-loop is not noticeable because all the three planes have normal conduction.
Abbreviations: ACW, anti-clockwise rotation; CW, clockwise rotation; EVCG, emergency vectorcardiogram; F, Frontal plane; H, Horizontal plane; ID, inscription direction; LS, Left sagittal plane; Mid, middle; N, normal; Pt, patient; Ref, reference; SC, slow conduction; Term, terminal; 8, figure-of-eight rotation.

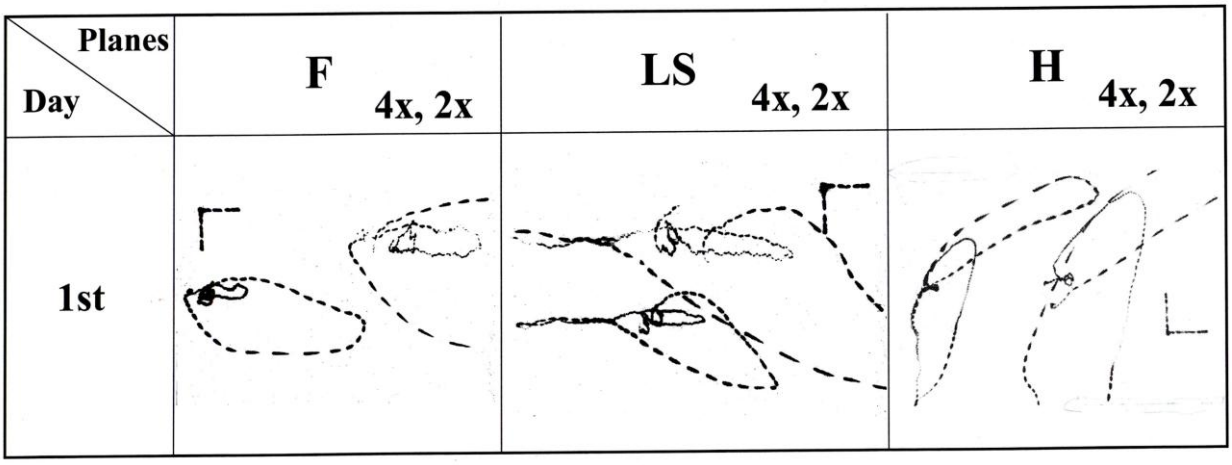

Fig 1. Vectorcardiogram. A patient with abnormal inscription direction of the T-loop. Patient (No.16) has acute anterior-inferior myocardial infarction and complete right bundle branch block with clockwise inscription direction of the T loops in the Left Sagittal(LS), and Frontal (F) planes. This phenomenon is progression toward the complete negative sequence with reverse phase rotation similar to a malfunctioning generator except the Horizontal (H) is not as yet present. The serious slow conduction of the T-loop is noted in the terminal portion of the T-loop in the three planes. Note a short conduction delay (closed marks) in the afferent part of the T loop in the H plane which is also abnormal. This emergency vectorcardiogram was recorded 15.5 hrs before his sudden death. The heads of tear drops indicates the direction of inscription of the T loop.Standard = 1 mV; magnification =x2 for the QRS loops and x4 for the T loops.(Aid in reading Figure 1 please see Supplementary materials)

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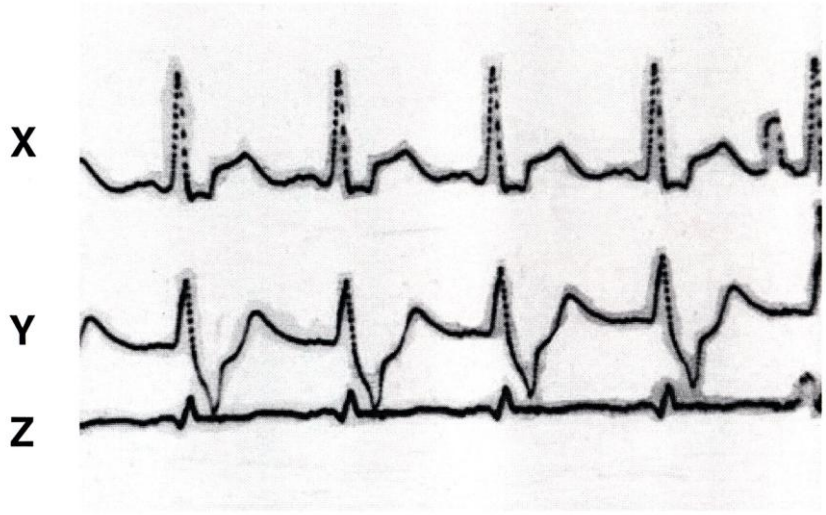

Fig 2. Orthogonal electrocardiogram. X, Y, Z orthogonal electrocardiogram of the same patient (No. 16). X, Y, Z orthogonal electrocardiogram is recorded during normal sinus rhythm with T wave elevation in the X lead tracing and T wave depression in the Y lead tracing. The information about serious abnormal rotation and slow conduction of the T-loop in the vectorcardiogram is not revealed by the X, Y, Z, orthogonal electrocardiogram during the same time. Standard = 1 mV; magnification = x1 for the X, Y, Z, orthogonal electrocardiogram

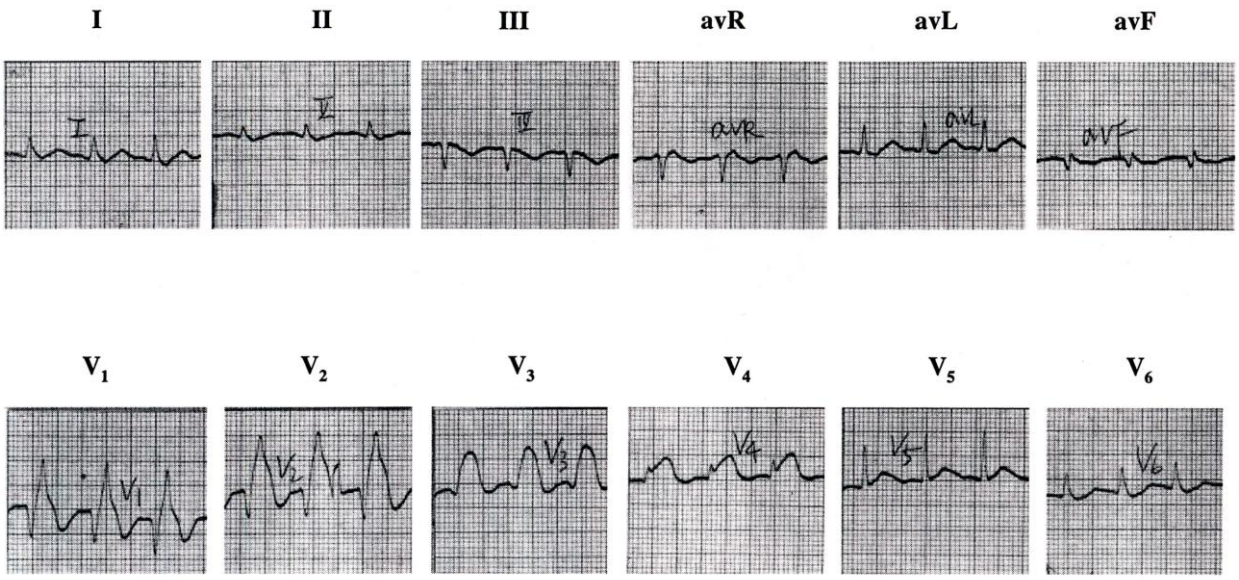

Figure 3. Electrocardiogram. Scalar Electrocardiograms in the same patient (No.16) with acute anterior-inferior myocardial infarction and right bundle branch block. The sequence of the first author recorded the electrocardiogram from bipolar leads I to avF, followed by unipolar leads V1 to V6. The first author recorded this electrocardiogram after vectorcardiogram which presented in figure 1. The information about serious abnormal rotation and slow conduction of the T-loop in the vectorcardiogram is not available from this electrocardiogram.

IV. DISCUSSION

Because a 24-hrs continuously timed vectorcardiographic recording device is not available in the market, therefore, this observational study is like using an old pistol to shoot a moving target during acute myocardial infarction. It is indeed a painstaking process. In the present study, twenty three (16%) among 148 acute myocardial infarction patients developed VT or VF. Twenty (87%) among these 23 patients had AID of the T-loop. A total of 137 of the 148 patients had AID of the T-loop. However, only 127 initially had AID. This can be explained by the fact that a 24-hrs per tracing protocol may not 'catch' the abnormal activation propagation within the ventricle, or it has already been abolished by VPCs. AID could happen in both

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depolarization and repolarization processes in a diseased heart [3], [12]. However, one patient had a huge ST vector that the T-loop is in a linear shape [13]; therefore, it was not possible to identify the inscription direction. Figure 1, 2, 3 of patient No.16 clearly demonstrated that ECG or X, Y, Z, orthogonal ECG could not supply information about AID & ASC of the T-loop. These T-loop abnormalities are also observed in patients with acute aortic dissection and unexpected ventricular tachyarrhythmia [12]. The clockwise rotation of T-loops in the Left Sagittal and Frontal planes but not Horizontal plane in Figure 1 is looks like that negative sequence voltages are already occupied Normandy as in the World War II. It is just a matter of time, in this case 15.5 hrs, eventually it will progressive into reverse-phase rotation of the all three planes, such as performing deep penetration into the Europe (as the heart) in war motion during the World War II.

Due to acute myocardial infarction, almost all of the patients 146 (99 %) among the total 148 had ASC of the T loop. A total of 108 (73 %) of the 148 patients had ventricular enlargement among 148 patients. Fourteen patients (61%) out of 23 patients with chamber enlargement developed VT or VF. Seven (50%) of these 14 patients which developed sudden death. However, in 9 patients without chamber enlargement have also developed VT or VF, 7 (78%) of those 9 have developed sudden death. This tendency indicates that patients with chamber enlargement might have a better chance of survival than patients without chamber enlargement during VT or VF. ASC of the T-loop or ventricular enlargement may help in sustaining VT or VF.

A. Invisible negative sequence voltages

As in the atrial tachyarrhythmia study of the P-loop [3], T-loop also has figure-of-eight (Pt. No. 6 in Table II) or clockwise (Pt. No. 9, 22 in Table II) rotation. Because figure-of-eight rotation is the combination of clockwise and anti-clockwise rotation, the phenomenon is similar to imbalanced partial negative sequence voltages in electrical power engineering. The clockwise rotation of the T-loop is similar to imbalanced complete negative sequence voltages in electrical power engineering.

Synchronous motors, such as ventricles, operate as induction motors while being brought up to speed so that they can be synchronized. However, prolonged load current imbalance, caused by imbalanced supply voltage (partial negative sequence), will form reverse-phase rotation (complete negative sequence). This will cause rotating at synchronous speed in the opposite direction of the rotor; this in turn will create a torque in the negative direction, which represents a more serious hazard for motors. When imbalanced voltages' levels reach 4% [14], a motor can no longer function. Astonishingly, every generator has the potential trouble of negative sequence voltages. Electrical power engineers have for many years known the problems which affect all generators including nuclear generators. The myth of figure-of-eight or clockwise rotation of the abnormal vectorcardiographic loops which is different from normal anti-clockwise rotation of the loops, could only be explained by the abnormal generator imbalanced voltages by mathematical eigenvalue analysis. An abnormal repolarization path in the ventricles is similar to the imbalanced voltages by mathematical eigenvalue analysis in electrical power engineering. Recently, the re-synchronization [15] concept has been appreciated by cardiologists. Furthermore, therapeutic hypothermia [16], [17] improves defibrillation success and resuscitation outcomes from VF. Obviously, when a generator is in reverse-phase rotation status, it will increase the work load and create heat in electrical power engineering. There is more and more evidence to suggest that the heart behaves like a biological generator.

A motor ceases to function when imbalanced negative sequence voltages' levels reach 4%. However, this value is not known in a biological motor: the heart, although the first author believes that the level could also be very low in the human heart. How could such a negligible value cause a devastating result in any motors similar to "the Butterfly Effect" [18]? The first author only recorded "frozen sections" of a dynamic status of the heart during emergency situations. It would be foolish to use these data as a correlation expecting a 95% occurrence rate for patients to develop VT or VF. The heart comes to its own defence because it has an auto-defence mechanism---the VPC. Furthermore, readers should be aware that in medical literature no one has used medical statistics to discover any laws of physics. Negative sequence voltages are indirectly derived from mathematical eigenvalue analysis. When viewing this well-established fact, which exists in the electrical power engineering territory of malfunctioning generators, we can also view the negative sequence in diseased hearts. Based on the study of atria [3] and ventricles of the heart prior to tachyarrhythmia, negative sequence is bound to become recognized as a new law in physics and medicine.

In the statistics involved with the vector cardiogram, there are two new errors, Type IV and Type V. The new Type IV error is one methodology, the QT dispersion studies of the ECG. Although many of these studies agree with each other, there is one slightly different methodology, the vector cardiogram, which can completely wipe out the QT dispersion of ECG [19] in clinical applications. This situation is similar to what happened in 1944 when German stealth jet fighters, Horten 2-29s were invisible to radar, which could not detect them. ECG results, similar to radar systems, showed agreement that there was nothing wrong, but vector cardiogram revealed that there was something serious wrong . The new Type V error is that biostatistics methods are not possible to evaluate a well-established electrical power engineering phenomenon which is based on mathematical eigenvalue analysis. Researchers should be very careful in applying biostatistics methods when

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encountering phenomena which belong to a different territory or discipline.

As disciples of Professor Alberto Benchimol, we are always reminded of the importance of visual inspections of the P, QRS, and T loops [20]. Because most vectorcardiographic researchers have not been able to explain the AID of the loops, they simply ignore it in the literature. The emergency vectorcardiographic Phase I Study was influenced by the Framingham Heart Study. The design was aimed at the Achilles' heel of the Framingham Heart Study: namely serial emergency follow-up study during acute myocardial infarction, vectorcardiogram, and orthodox methods in medical research which characteristically with inter-observers' variation value is zero. Furthermore, the electrical power engineering concept of generators is, in reverse engineering's way, applied in the interpretation of the emergency vectorcardiographic Phase I Study results.

B. VPC

Why will some patients develop VT or VF directly or VF followed by VT after acute myocardial infarction? This may be related to the quantitative level (1 – 4 %) of negative sequence voltages. However, there has no machine available for physician in quantitative analysis of that level. The device must be developed by engineers in near future. When VPCs are ***incompetent*** to stop the abnormal inscription direction in the ventricle, VT or VF will follow. VPCs are ***not*** the cause of VT or VF, as suggested by many electrocardiographic or electro-physiological studies. VF occurs when the ventricles seem to have tried every resource of incompetent VPCs (probably including anti-clockwise but cannot match with the negative sequence) in combating negative sequence voltages. This situation is similar to German boy scouts, bunch of juveniles, defending Berlin (as the heart) against red army during World War II. The ventricles treat death threats from negative sequence voltages seriously during the course of VF. ***The subtle negative sequence voltages, in which bio-electricity and bio-mechanical effect plays a great role similar to electrical power engineering, are formidable***. Therefore, it is for a reason that the heart generates a VPC. Furthermore, in the present study, the VT and non-sustained VT should be viewed as" overdrive suppression" from the heart.

The increase in mortality rates in patients with VPCs treated with anti-arrhythmic drugs in the CAST study [21] is negative proof that, physiologically, VPCs are good for patients. The mortality of 3,000 patients [22] could have been prevented if the research funding had been available for the vectorcardiographic studies. Thus, the VPCs, per se, should not be targeted for therapy, whereas AID and ASC of the T loops should be evaluated seriously. This is especially true for those seemingly "normal" sinus beats before VPC. Furthermore, it is well known that VPCs could appear in normal adults and children, as negative sequence voltages in motors. This could explain how purely electrical heart diseases, defined by the absence of any structural cardiac defects, are responsible for a large number of sudden, unexpected deaths in otherwise healthy, young individuals [23], or patients with emotional stress [24], [25] or so-called "broken heart syndrome".

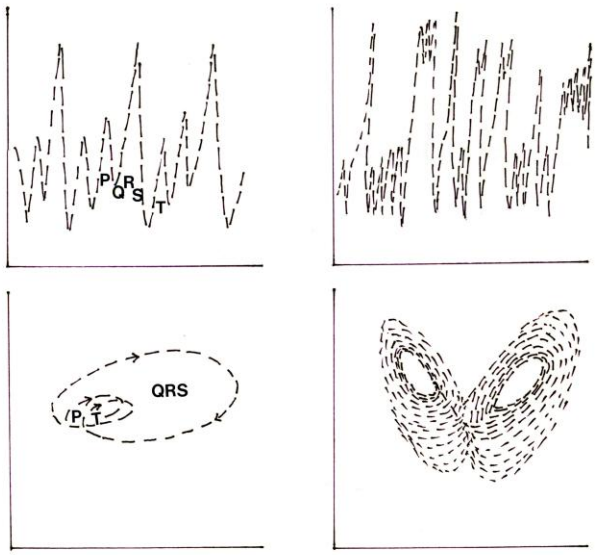

Fig 4. Making portraits in phase space. Traditional time series (above) and trajectories in phase space (below) are two ways of displaying the same data and gaining a picture of a system's long-term behavior. The first system (left upper) repeats itself in a more complex waltz rhythm, a cycle with "period three" (left lower).The interesting thing in here is that the figure from physics is very similar to the P, QRS, T loops in the vectorcardiogram during tachycardia. The second system (right) is chaotic. It's interesting that the figure-of-eight loop (right lower) is similar to abnormal figure-of-eight rotation of the T-loop in the vectorcardiogram, which is similar to the partial negative sequence of malfunctioning generators. [Ref. 18]

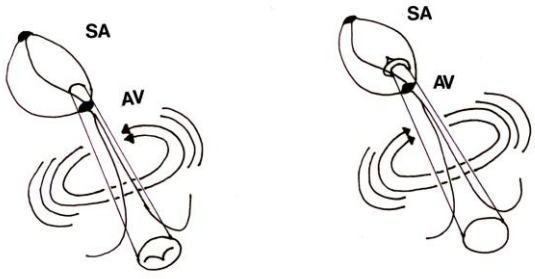

Fig 5. Depolarization vector during normal or DC cardioversion of the heart. Normal depolarization vector from the AV node to ventricles in downward direction (left), which is compatiable with the normal QRS-loop rotation in the vectorcardiogram. An interesting thing in here is that the magnatic field rotation is in anti-clockwise rotation in the heart, whereas, according to the laws of physics, it should be clockwise rotation in physics. DC cardioversion vector from ventricles to the AV node is in an upward direction (right). A

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very interesting thing in here is that the magnatic field rotation is in clockwise rotation in the heart, whereas according to the laws of physics, it should be anti-clockwise rotation in physics.

In a non-linear system, or a non-linear effect, the path might form a loop, going around and around, meaning that the system had settled into a pattern of behavior that would repeat itself periodically as chaotic behavior [18]. The three types (anti-clockwise, figure-of-eight, clockwise) of inscription direction of the T loops mixed with period-doubling and the distance variation of firing of the different VPCs (different T amplitude), similar to the Beluzov-Zhabotinsky reaction in chemical chaos [18], could be applicable in forming a VF rhythm. A small change in one parameter could push an otherwise healthy system across a bifurcation point into a qualitatively new behavior [18] or vice versa. Indeed, the P, QRS, and T loops of vectorcardiogram during tachycardia are similar to a cycle with period three in physics (Fig. 4). Based on the above-mentioned facts in physics**,** if these facts were also true for human hearts, we hypothesized that VT or VF could be terminated easily by forced VPCs in anti-clockwise rotation, which needed less power than the shock delivered from the implantable ventricular defibrillators. The voltage to generate a VPC or several VPCs is certainly much less than a DC cardio version. Cardiac therapy should comply with physiology and be targeted toward preventing heart exhaustion and stony heart formation after tachyarrhythmia. Physicians know that the DC cardio version (Fig. 5) has no foundation in physics. One would expect that the DC cardio version could stop irregular heartbeats at once, and that then the SA node would send in a normal sinus beats. However, the DC cardio version could reverse block the SA node and AV node, and seriously delay these nodes from functioning normally, hence creating the iatrogenic vegetable life of the patient. Furthermore, readers will notice that some patients with post cardio version vectorcardiogram have negative sequence of the T-loop in Table II. This may explain some later mystery deaths of patients in the ward, or at home. This is also similar to how a Taser kills a live human being—Mr. Robert Dziekanski documented on tapes [26]. The question about Tasers should be whether they create iatrogenically imbalanced negative sequence voltages in the heart. Because cardio version is only treating "reactions" of the heart to negative sequence voltages, but not negative sequence voltages per se. Furthermore, there was a high rate of early recurrence of ventricular tachyarrhythmia treated by implantable cardioverter defibrillators [27].

One patient (No. 22) whose torsade de point was terminated by first thump is very interesting. The abnormal rhythm could be due to figure-of-eight rotation of the T-loop similar to chaotic (Fig. 4). The first thump in this case is similarly to "physiological pacing". It is also interesting that low-frequency oscillations hit in a north to south direction will re-create a torsade-like wave in physics, as the erratic ground motion of the magnitude 7.6 Chi-Chi earthquake which struck Taiwan in 1999 [28]. Further study is needed for patients with this type of tachyarrhythmia.

C. Patho-electro-physics-physiological theory of VF, VT and VPC

The duration of VF required to produce pulseless electrical activity was encountered in 100% of nine swine after 180 seconds of VF. It was only possible to eliminate pulseless electrical activity and restore pumping in one animal when untreated VF lasted more than 180 seconds [29]. Syncope of patients due to VT or VF is a "normal" reaction of the body to protect other vital organs' blood supply through the autonomic nerve system. The body needs to work in this way to combat the invisible force of gravity in physics. Therefore, syncope is one of the body natural self-defence mechanisms. It may indicate a fundamental crucial problem in physics aspect of the heart or brain, not just as simple as old explanation caused by vasovagal reflex.

A proposed new theory of VT, VF or VPC. Negative sequence voltages appear as an AID in the repolarization process of the ventricles. These negative sequence voltages could occur in a diseased or electrocardiographically 'normal' heart. The heart will generate a VPC ('physiological' pacing) or VPCs ('physiological' overdrive suppression) to abolish the negative-sequence-voltages-induced abnormal activation propagation. The threshold, when this physiological correction will appear through the autonomic nervous system is variable depending on the individual patient (*i.e.* heart conditions, body weight, age...etc.). If it is not successful, the VT from single foci or chaotic VF from multiple foci will ensue. With the help of chamber enlargement and/or abnormal conduction within the ventricles, the abnormal rhythms will be sustained.

Therefore, in a patient with VPCs, the heart is seriously fighting against invisible negative sequence voltages and the auto-defence mechanism of the heart is activated. In a patient without VPCs, one can not conclude that there are no negative sequence voltages, except if proved otherwise by the vectorcardiogram. Clinicians should be aware that, until now, there has been no therapy of heart diseases which considered negative sequence voltages. At the present time, there is no therapy which can help any heart to fight against negative sequence voltages. In fact, negative sequence voltages have only ***short- or long- term remission,*** until if and when there may ever be any effective therapy proved by future studies. There will be ***no cure*** for negative sequence voltages because it belongs to the territory of bio-electricity and bio-mechanical aspect of electrical power engineering**,** but not to the territory of medicine. It is interesting that in *The Grand Design*, Prof. Stephen Hawking of Cambridge University stated that the universe is created by physics. The present study indicated that human life is also terminated by negligible imbalanced voltages in the heart. Furthermore, a recent publication [30] from the surgical department in this

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institute describing the efficiency of using assisted extracorporeal life-support in detouring and avoiding head-on collision with invisible negative sequence voltages, is rather encouraging in the direction of preserving other vital organs, such as the brain in the human body during ventricular tachyarrhythmia.

D. The inconvenient truth

A very interesting phenomenon occurs whenever physicians go on strike are the death rates go down! This phenomenon has occurred and been recorded in a number of locations. It happened in Canada in 1960. In Columbia in Bogota in 1976 when all the medical doctors, except emergency department, went on strike for 52 days, the death rate dropped by 35%. In the USA, in Los Angeles physicians associated with a USC hospital went on strike in the USA in 1976, and this resulted in an 18% drop in mortality. When the strike was over, the death rate went back to normal. In 1973 in Israel when there was a one-month doctor's strike, a 50% decrease in mortality occurred. The conclusion is that physicians remain very dangerous to the public health. The negative sequence voltage is directly related to cardiac arrest. It indicates that physicians are totally incompetent in bio-electricity and bio-mechanical aspect in the electrical power engineering; especially in mathematical eigenvalue analysis (see Appendix A), and physicians' training in physics is an utter failure.

This independent study has the same age as the Heart Rhythm Society which founded in 1979. However, our results combined with the Hippocratic Oath, indicated that electrical power engineers with vectorcardiographic training should be the choice, when comparing with electro-physiologists or cardiologists, for arrhythmia research.

E. Limitations

Since the "vector" in the vectorcardiography is within the territory of physics, it is not surprising that vectorcardiogram using "physics' language" for communication purpose, are totally indecipherable to those accustomed to using medical or biostatistics methods. Because of that there have been no diagnostic armamentaria, or "radar" systems, designed for directly visualize partial negative sequence voltages or complete negative sequence voltages in the heart. The present study has been forced to utilize electrical trails (like footprints on a footpath), similar to a forensic criminal investigation in a murder case. Certainly, vectorcardiography is a qualitative but not a quantitative method for negative sequence voltages. Traditional vector cardiograph needs two minutes to complete recording Horizontal, Frontal, and Left Sagittal three planes. The negative sequence in the heart could change very quickly during emergency situations. Therefore, modern vector cardiograph should be designed as synchronized recording three planes in any one cardiac cycle.

This study explains the myth of abnormal figure-of-eight and clockwise rotation of the vectorcardiographic loops. Furthermore, as things are at present, medical and biostatistics methods as only "gold standards" are slaughtering without any mercy by the mathematical eigenvalue analysis and tens of thousands of generators running in the world, which including nuclear generators. The domino effect of the collapsing of pharmaceutical [31]-[33], medical [27] and surgical treatments of VT, VF and VPC is inevitable. Whether the "zero sequence" in electrical power engineering will replace the "ventricular asystole" terminology in Medicine, only time will be the vindicator.

In the 30 years since its completion, the emergency vectorcardiographic Phase I data has given rise to very serious questions about different publications concerning VT, VF, or VPC. History is repeated again in the present case of tachyarrhythmia study, as in the Anatomy. The renowned Belgian anatomist Andreas Vesalius disapproved professors not doing their own hands-on empirical studies, and described them as " jackdaws aloft in their high chair" and said that"… everything is wrongly taught,…and days are wasted in ridiculous questions." [34] Is it possible that the etiology of tachyarrhythmia is hidden in the electrical power engineering territory that physician-researchers have no credentials to understand it? Furthermore, problems derived from the Hippocratic Oath [35]: Is it true that physician-researchers betrayed orthodox methods in medical research can have massive publications, so that they think they are smarter? Are they not afraid of their massive arrhythmia publications in these 30 years will hunt them down in return, and eliminate their position (or record) with their pension? Are physician-researchers in teaching hospitals "massaging" their data for promotion purpose, and monopoly for funding of arrhythmia research? Are those physicians not afraid Judge Kenesaw Mountain Landis released statement to the press concerning Black Sox trial in 1921 which is not applicable to MDs? Are families of deceased patients should be legally compensated under therapy guidelines based on these 'massaged-data' publications? These serious conflict-of-interest questions are also applicable to the reviewers of the emergency vectorcardiographic Phase I Study results in these 30 years.

Future enhancement after this research, electrical power engineers should consider:

- Integrate negative sequence voltages into electricity section of general physics textbooks.
- Re-educate physicians in applied physics, and electrical power engineering principle.
- Take over major national research funding, such as the NIH in the USA research funding for arrhythmia research.
- Invent a portable machine can quantitative negative sequence voltages in human hearts, esp. for the Emergency Service or Intensive Care Units.

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- Re-think present hospital policy that only cardiologists or electro-physiologists can investigate patients with cardiac arrhythmia.
- Forbid any big pharmaceutical companies or medical device companies to influence applied physics or engineering journals, such as they did on medical journals [33].

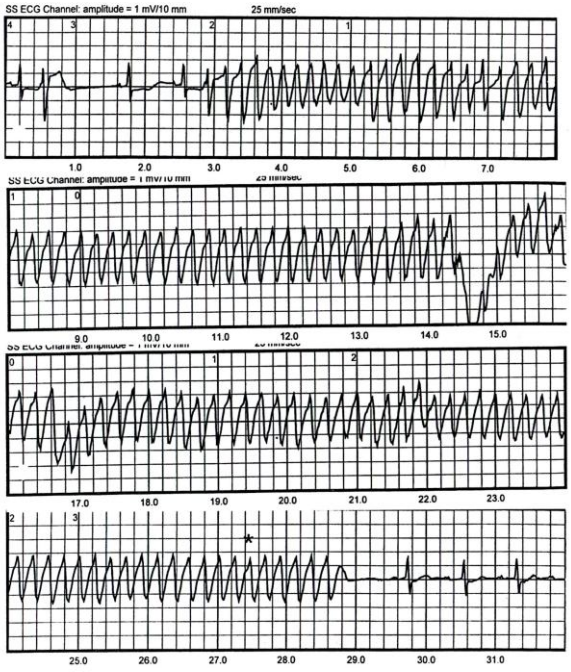

Fig 6. Self-healing ventricular tachycardia. A 56 years old female patient with first ventricular premature contraction (VPC) as a 'physiological pacing' in the second beat, and subsequent VPCs starting from the fifth beat as a continous 'physiological overdrive supression' from the heart. It seems that the heart combating the underlining invisible negative sequence voltages is rather successful before the heart collapse in this patient two months after acute myocardial infarction. The physician was holding the wearable DC cardioversion at the time. No vectorcardiogram was performed for the T-loop of those so-called "normal sinus beats" prior of the first VPC or subsequent VPCs. (Courtesy of Prof. Helmut Klein, Germany).

F. Addendum

On 28th June-1st July; 2006, in Cologne, Germany, the first author noticed a presentation of wearable DC cardioversion device by Prof. Helmut Klein. One of the cases, (Fig. 6), is by far the longest VT with successful conversion back to normal rhythm by the heart itself without deteriorating into VF, or any intervention from physicians.

Prof. Philip Poole-Wilson was one among a number of renowned Cardiologists who declined first author's proposal for further emergency vectorcardiographic studies in 1999. The Professor had a heart attack and died suddenly in 2009. Negative sequence voltages will affect ordinary persons, Cardiologists, or Cardiovascular surgeons, in exactly the same way. The one writing this article is also no exception to this "invisible & invincible Grim Reaper".

DECLARATION

Contribution: Wangden Carson did designing, recording and analysis of data, also preparation of the manuscript. Yung-Zu Tseng read vectorcardiographic diagnosis. Conflict of interest: none declared.

APPENDIX

When there is imbalance in the voltage supply due to any reason, the imbalance in supply can be resolved into three balanced quantities called positive sequence (V1), negative sequence (V2), and zero sequence (V0) voltages. These sequence voltages produce their sequence current as well. Thus negative sequence current is produced. Under normal balanced condition, whatever voltage and current are present are in positive sequence. The negative sequence comes into the picture only when imbalance exists in the supply voltage. With no phase displacement between the three voltages in the zero sequence system, the motor will not rotate at all as there will be no rotating magnetic field.

Regarding the measurement of negative sequence, it is measured by the negative sequence filters within the relays. However, it can always be calculated for a given set of imbalanced voltages. To calculate the negative sequence current, the negative sequence impedance of the machine should be known. The calculation procedure for the sequence voltages and currents can be obtained from any standard textbook of symmetrical components, such as J. Grainger and W. Stevenson, *Power System Analysis*, (McGraw Hill Co.,1994).

Two definitions of the voltage imbalance calculation are the International Electro-technical Commission (IEC) of Europe and the National Electrical Manufactures Association (NEMA) of USA which can each result in different values.
IEC definition: Negative sequence voltage imbalance =

$$\frac{V2}{V1} = \sqrt{\frac{1-\sqrt{3-6\beta}}{1+\sqrt{3-6\beta}}} \text{ where } \beta = \frac{V_{ab}^4 + V_{bc}^4 + V_{ca}^4}{(V_{ab}^2 + V_{bc}^2 + V_{ca}^2)^2}$$

NEMA definition: Voltage imbalance = Maximum deviation from mean of $\{V_{ab}, V_{bc}, V_{ca}\}$/ Mean of $\{V_{ab}, V_{bc}, V_{ca}\}$

The equations to calculate positive and negative sequence voltages are:
$V1 = 1/3 \times (V_a + \square\,\square V_b + \square\,\square^2 V_c)$
$V2 = 1/3 \times (V_a + \square\,\square^2 V_b + \square\,\square V_c)$
Where "□" is an operator equal to $1\angle 120°$. Multiplying a phasor by "□" rotates that phasor 120 degrees anti-clockwise. When squared, the "$\square^2$" operator becomes equal to $1\angle 240°$ and rotates a phasor by 240 degrees anti-clockwise.

Consider a perfectly balanced system.
$V_a = 67\ \angle 0°$ volts

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V_b = 67 ∠-120° volts
V_c = 67 ∠120° volts
V1 = 1/3× (67∠ 0° + 1 ∠ 120° ×67 ∠ -120° + 1 ∠ 240° ×67 ∠120°) = 1/3 × (67∠ 0° + 67∠ 0° + 67∠0°) = 1/3× 3× 67∠0° = 67∠ 0° volts
V2 = 1/3 × (67 ∠ 0° + 1 ∠ 240° ×67 ∠ -120° + 1 ∠ 120° ×67 ∠ 120°) = 1/3 × (67∠ 0° + 67 ∠ 120° + 67 ∠ 240°) = 1/3×0 = 0 volts
For a perfectly balanced system, positive sequence voltage is equal to 67 ∠ 0° volts and negative sequence voltage is zero.

Consider a perfectly imbalanced system or a system that has reverse phase rotation.
V_a = 67 ∠ 0° volts
V_b = 67 ∠ 120° volts
V_c = 67 ∠ -120° volts
V1 = 1/3 × (67 ∠ 0° + 1 ∠120° ×67 ∠ 120° + 1 ∠240° ×67 ∠ -120°) = 1/3 × (67∠0° + 67∠240° + 67 ∠ 120°) = 1/3 ×0 = 0 volts
V2 = 1/3× (67 ∠ 0° + 1 ∠ 240° ×67 ∠120° + 1 ∠ 120° ×67 ∠ -120°) = 1/3 × (67 ∠ 0° + 67∠0° + 67 ∠ 0°) = 1/3×3×67 ∠ 0° = 67∠ 0° volts
For a perfectly imbalanced system, negative sequence voltage is equal to 67∠0° volts and positive sequence voltage is zero.

A forward fault is declared when the negative sequence current leads the negative sequence voltage by 180 degrees minus the characteristic angle of the transmission line. This characteristic can be described by the following:

$$T32Q = |V2| \times |I2| \times \cos(\angle -V2 - (\angle I2 + MTA))$$

Where T32Q is the "torque" produced by the negative sequence directional element and MTA is the characteristic angle of the transmission line. **I**2 is negative sequence current. A positive torque indicates a fault in the forward direction.

Total rotor heat = $\alpha(I_{\pi}^{2} + 3I_{ru}^{2})$

Where I_{π} = rotor current, I_{ru} = negative sequence rotor current

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Supplementary materials

1. 3,000 may have died in U.S. drug test for heart patients. (Ref. 22)

A22/ THE TORONTO STAR, WEDNESDAY, JULY 26, 1989 ★

3,000 may have died in U.S. drug test for heart patients

WASHINGTON (AP) — Up to 3,000 patients may have died prematurely in a nationwide trial of two drugs designed to prevent irregular heartbeats, but which actually caused heart attacks, according to a survey of cardiac specialists.

Dr. Joel Morganroth, director of cardiac research at the University of Pennsylvania, said he arrived at the estimated mortality toll after surveying 1,000 cardiologists to find out who had prescribed the drugs during the trials.

"Let's call that the worst scenario," Morganroth said yesterday of the estimate of 3,000 premature deaths.

Extra heartbeats

The two medicines, encainide and flecainide, were approved in 1987 for preventing a type of cardiac arrhythmia that features extra beats in the heart's two lower chambers, the ventricles.

A national study of the drugs, called the cardiac arrhythmia suppression trial, or CAST, was started in June, 1987. It was halted abruptly last April when data showed sudden death rates increasing among patients taking the drugs.

Since then, Morganroth said he conducted a randomized survey to determine how many patients may have died prematurely from the use of the drugs.

He said half of the 250 cardiologists responding to his survey said they had prescribed the drugs.

Based on these results, Morganroth said he estimated the number of premature deaths.

"Fifteen hundred to 2,000 died, if all the assumptions are appropriate" in the last 12 months of the trials, he said. For the previous year, when the drugs were not as widely used, he estimates the premature deaths at 750 to 1,000.

Physicians' Weekly, which reported on the survey, called the drug trials a "sudden-death debacle."

Dr. Thomas Bigger, a Columbia University heart researcher who was chairman of the arrhythmia studies, said: "We were very shocked because it (the study) seemed very reasonable."

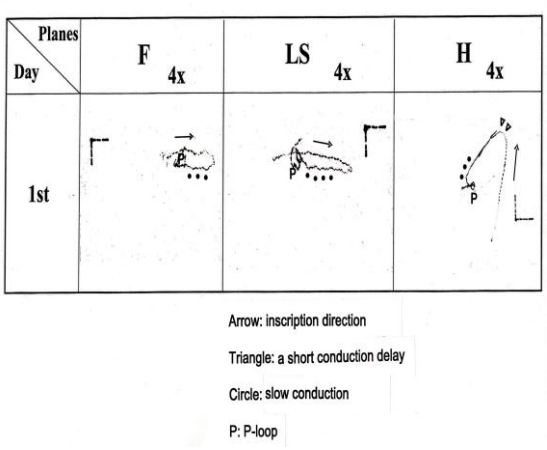
He said the study seemed so logical that at first some doctors were reluctant to have their patients participate because there was a 50 per cent chance that a patient would receive a sugar pill instead of the heart drugs.

He said the doctors felt that those who got the placebo would be the unlucky ones. But the study showed it was the other way around.

Patients in the studies, which were initiated by the National Institutes of Health, had all suffered a type of heart attack and then experienced very mild irregular ventricular heartbeats.

Encainide, marketed as Enkaid by Bristol-Myers, and flecainide, sold as Tambocor by 3M Riker, are still being prescribed, but only for patients who have life-threatening levels of irregular heartbeat, said Dr. Lawrence Friedman, the NIH director of the CAST studies.

2. Aid in reading Figure 1.



AUTHOR BIOGRAPHY

Dr. Wangden Carson has received his Medical Doctor degree in 1981 and then passed National Civil Servant examination and received Government Scholarship for further study in the United Kingdom. Dr. Carson later received his D Phil degree in clinical cardiology from the University of Oxford under Professor Peter Sleight of the John Radcliff Hospital. He then has appointed as a Consultant Cardiologist by the Government to a Taipei Metropolitan Hospital. He is the solo author of *Emergency Vectorcardiography* (1998, UK edition), and the founder of Negative Sequence Voltages in the heart project.

Emeritus Professor Yung-Zu Tseng has received his Medical Doctor degree in 1966 and went to Japan for further study of cardiovascular diseases. He is appointed as a professor of Internal medicine in 1984. He has published hundreds articles and later received his fellowship from the American Chest Physician and the European Society of Cardiology. Professor Tseng is count as an only advocate of Vectorcardiography for the recent four decades in Taiwan. He is a distinguished scholar of the cardiovascular diseases in the Taiwan society of Cardiology. He is also serving as a consultant in the medical legal committee in Taiwan.