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Analysis of Significant Electrocardiogram (ECG/EKG) Variations in Psychological Stress

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ABSTRACT

Purpose: *Mental stress and other psychosomatic disorders have been linked to an increased risk of sudden cardiac death in population studies. There hasn't been a thorough examination as to whether emotional or physical stresses might cause sporadic ventricular arrhythmias in vulnerable patients. An outsized and mounting body of discovery shed light on the fact that internal health is interrelated to risk factors for heart complaint before an opinion of an internal health complaint and through treatment. These things can arise both directly, through natural pathways, and laterally, through parlous health actions. People who are passing through clinical depression, anxiety disorders, common and general stress syndromes, and indeed post-traumatic stress disorder commonly known as PTSD over an absolute period of your occasion may observe definite physiological alterations in the body, similarly, as a highly enlarged cardiac reactivity such as augmented vital signs and elevated blood pressure, deducted or condensed blood inflow directly to the center, and heightened or highly raised amount of an organic chemical compound called cortisol. Over time, these physiologic processes may generate calcium particle depositions or buildup within the normal organic pathways. It may also manifest events of body metabolic dysfunction as well as cardiac conduction problems. Substantiation shows that many physiological and psychiatric/psychological diseases -similar to chronic clinical depression, psychological stress, and PTSD can appear after any cardiac event regardless of its severity.*

Objective: *The focal objective of this article is to identify psychological stress, its influence on cardiac problems such as cardiac arrhythmia, and its symptomatic manifestations on an electrocardiogram. Apart from that, this paper also focuses on the area of how ECG changes due to psychological distress. Another objective of this paper is to provide vivid information about psychological stress and its link with acute and chronic cardiac pathologies. Laymen should know the fact that the mind and the body are linked with each other and if the mind is distressed for a long time will affect the health condition of the physical body also.*

Design/Methodology/Approach: *To build the paper, mostly secondary information has been used from various sources such as clinical literature, journals, websites, etc. Primary information has also been used to increase the accuracy and reliability of the paper. Primary data have been taken from neuropsychologists, cardiologists, clinical psychopathologists, and behavioral medicine specialists. Based on all these databases, systematic and scientific clinical analysis of information has been done to avoid any mistakes and errors.*

Findings/Result: *In people with ICDs, stress and physical exercise might cause ventricular rhythm abnormalities. Future studies into medicines that prevent these stimuli' responses might reduce ventricular arrhythmias and upsets in some of these patients. People with type A personalities should try to control or adjust their innate personality and character to reduce the vulnerability to develop any kind of cardiac arrhythmia or any other cardiac pathologies. All ECG variations and arrhythmias are not related to psychological stress rather it needs differential diagnosis to rule out any other underlying problems. A thorough differential investigation is suggested to rule out whether the cardiac problem is due to psychological stress or not.*

Originality and value: *A comprehensive analysis and interpretation have been made to cover the concerned topic of study and make information more reliable and valid. A new initiative is taken to provide information about the mind and heart link.*

Paper Type: *Secondary clinical report analysis/interpretive paper*

Keywords: Psychological stress, Cardiac Arrhythmia, Tachycardia, Sudden Cardiac Death (SCD), ECG variations.

1. INTRODUCTION :

Acute mental stress (acute psychological stress) can cause significant atrial or ventricular arrhythmias or both, although the exact (bodily activities) physiological mechanisms are yet unknown. Electrophysiological processes of stress-induced arrhythmia can be elucidated using signal processing methods. In the lab, T Wave alternant, as well as further ECG measures of repolarization heterogeneity, rise with cognitive and significant mental/psychological stress, and may also increase in "real world" situations. Significant psychological stress and anger can precipitate intense cardiac pathology starting from arrhythmia [1]. Stress affects components of the signal-averaged electrocardiogram dissimilarities in the atrium. These alterations point to pathways via which common stresses might cause arrhythmia. Recent research has found that psychological variables have a key role in the etiology and manifestation of Coronary Artery Disease popularly known as CAD. The research allied CAD risk to five particular psychosocial spheres such as-

(1) Depression
(2) Anxiety
(3) Personality variables and personal characteristics
(4) Social isolation
(5) Chronic life stresses (CLS)

All these factors make up the majority of the aforementioned evidence. Behavioral mechanisms, in which psychosocial circumstances result in a far above the ground occurrence of negative fitness behaviors like meager diet and smoking, and straight pathophysiological mechanisms, like neuroendocrine and hyper platelet activation in the body, are some of the pathological physical activities underlying the correlation in between these clinical entities and Coronary Artery Diseases. Researchers have also been identified the direct connection of psychological stress with cardiac arrhythmias and other fatal issues A wide study was conducted to identify the link between psychological stress and cardiac issues during the period of the US presidential election in the year 2016. The result of this clinical study was positive. A direct connection between mental stress and cardiac problems has been identified [2]. Chronic psychosocial stressors can cause aggravation of Coronary Artery Atherosclerosis, as well as Temporary Endothelial Dysfunction, and then can even lead to cellular necrosis, at last, organ degeneration. The same incidents have been reported by a large body of scientific clinical data from animal model experiments like the cynomolgus monkey (Macaca Fascicularis).

2. RELATED WORKS :

Many studies and researches have been done to identify the relationship between psychological stress and its connection with cardiac arrhythmia. Even an illiterate person is well aware of the fact that mental stress can trigger develop heart attack (myocardial infarction) but only a few people know about the connection between psychological distress and cardiac arrhythmia. Most of the clinical studies and research have been conducted from the area of cardiology and electrophysiology. Not much clinical studies are not available in the domain of psychiatry/psychology or any other subspecialties. But it does not mean that the domain of psychiatry or psychology is lacking clinical study on the topic of

psychological stress and its effect on the heart. Some of the major researches in the domain of psychological stress and its effect on electrocardiogram variations are given below.

Significant psychological stress can contribute negatively to generating cardiac arrhythmia starting from tachycardia to intensive supra ventricular tachycardia (SVT). If the patient is already suffering from any kind of cardiac problem, significant psychological stress can generate electrocardiogram variations. If it is diagnosed and treated within the golden hour, the major pathological output can be reduced [3]. In some other research studies, it has been identified that there are only temporary alterations in electrocardiogram results if the person is healthy and does not have any existing cardiac problem. Once the psychological state becomes normal, electrocardiogram results will also become normal. It is only in the case of temporary psychological stress. But, if the patient is having ICD, the scenario has to be monitored properly. People with ICD may not tolerate even the least psychological stress [4]. In persistence and significant psychological stress T Wave altrnans can be visible and that causes significant changes in electrocardiogram also. Persistent psychological stress is one of the risk factors to develop cardiac arrhythmia [5]. Psychological stress-induced arrhythmias can be classified by taking differential diagnoses and the degree of stress. Temporary stress relief will show only a trivial alteration in electrocardiogram results. Clinically significant stress should not be neglected since it may precipitate cardiac problems [6]. It is also identified from clinical studies that psychological stress can cause major heart rate turbulence and severe ventricular arrhythmias. It can also cause ventricular hypertrophy as well as bundle branch blocks [7]. Many other scientific clinical research findings have also been given below to ascertain the point that psychological stress can precipitate cardiac arrhythmias even the person is physiologically healthy.

Table 1: This shows the link between psychological/psychiatric illness and its correlation with cardiac disease.

Sl. No.	Title of the paper	Focus area/Findings	Reference
1	*"Mental Stress and Ventricular Arrhythmia"*	Ventricular arrhythmias can be triggered by rage. Arrhythmias are also made more vulnerable by long-term unpleasant emotions. Autonomic alterations, which modify repolarization, presumably increased in people who are having sympathetic denervation, which then in return produce highly morbid Polymorphic Ventricular Tachycardia, are among the pathways relating to rage and arrhythmias.	Lampert, R. (2016). [8]
2	*"Mental Stress and Sudden Cardiac Death: Asymmetric Midbrain Activity as a Linking Mechanism"*	Two ECG-derived indexes of repolarization non - uniformity were used to quantify proarrhythmic alterations in the heart, which were linked to alterations in the amplitude and lateralization of local brain function represented in regional blood flow.	Critchley, H. D. et. al. (2005). [9]
3	*"The Effect of Mental Stress on the Non-Dipolar Components of the T Wave: Modulation by Hypnosis"*	In the lack of ischemia, psychological stress affected repolarization heterogeneities through altering the autonomic equilibrium.	Taggart, P. et al. (2005) [10]
4	*"Stress and Cardiac Arrhythmias"*	It is generally known that emotions such as psychological stress and sadness have an impact on cardiac autonomic regulation. Persons with the largest abnormalities in cardiac neural control, such as reduced	Johnson, B. et al. (2014). [11]

		parasympathetic tone combined with growing sympathetic activity, are at the highest risk for having deadly ventricular arrhythmias, according to a growing body of research.	
5	*"Depression and Cardiac Dysautonomia in Eating Disorders"*	When contrasted with the non-depressed patient group and controls, the prevalence of depression in ED patients reduced heart rate variability, even more, indicating that more awareness and a comprehensive treatment strategy may be necessary for these individuals to eliminate cardiac arrhythmia problems.	Jelinek, H. F. et al. (2018) [12]
6	*"Heart rate Variability in Psychiatric Disorders"*	Addresses the relationship between psychiatric disorder and its link with cardiac problems. This paper also stresses the influence of the sympathetic, parasympathetic, and autonomous nervous systems and their direct connection to propagate cardiac arrhythmia.	Yang, A. C. et al. (2010) [13]

3. OBJECTIVES :

This paper is prepared to shed light on the topic of psychological stress or in another popular term mental stress and its connections to generate cardiac problems, more particularly cardiac arrhythmias and sudden cardiac death. People are well aware about cardiac arrhythmia and sudden cardiac death, but only a least number of people are aware about the fact that psychologicapj65l/mental stress has a direct connection to generate cardiac arrhythmia. So the prime objective of this paper is to discuss about psychological stress and its relationship to generate cardiac arrhythmia on an ECG paper. More objectives are listed below.

(1) To identify the effect of psychological/mental stress to cause cardiac arrhythmia
(2) To identify the relation between psychological stress and ventricular arrhythmia.
(3) To rule out the effect of psychological stress in the atrium and its notable changes in ECG.

4. METHODOLOGY :

This paper is built upon secondary data. Scientific and systematic analysis has been done to obtain the final result and conclusion. Even though this paper is based on secondary data, the personal clinical experience gave some insight to make the data more accurate. To retain and enhance the reliability and validity of the paper, expert opinion has been taken from the side of psycho-diagnosis specialists, psycho endocrinologists, clinical psychologists as well as cardiologists. To get valid and reliable clinical data, authorized websites and journal papers have been scientifically analyzed. In this regard, more than eighty journal papers including published and unpublished journals have been systematically analyzed.

5. OVERVIEW OF CHANGE IN ECG VARIATIONS IN VENTRICLE DUE TO STRESS :

In epidemiological and medical studies, psychological distress is characterized as a consciously perceived disparity between expectancies and the observable situations connected to anxiety sensitivity and avoidance has been found to trigger ventricular or atrial arrhythmias, or in some cases both [14]. Data indicating increases in Sudden Cardiac Death (SCD) following stress-inducing community trials such as earthquakes or war provide the first evidence suggesting associating acute psychological distress with ventricular arrhythmias [15]. Increases in cardiovascular and Sudden Cardiac Arrest (SCA), for example, were documented in Israel during the Iraqi missile war in 1981 and after airstrikes on Zagreb [16]. The reported unexpected deaths at some stage in each of these residents' catastrophes were not linked to physical injuries or any direct physical participation, showing that psychological stress played a role rather than physical stress. Ischemic or arrhythmic episodes can trigger SCD, and

the impact of stress on ischemia has long been known. On the other hand, authentic data from ICD patients indicating an increase in ventricular arrhythmia following the 9/11/2001 assaults on the World Trade Center suggests that autonomic alterations due to stress may directly control arrhythmogenesis [17]. Stress and arrhythmia have also been linked in clinical research. Using a test case of ICD patients, we take a closer look at whether rage and other emotions can cause ventricular arrhythmias. Patients were instructed to keep a journal of their actions and feelings within the first fifteen minutes or two hours before receiving an ICD shock, according to this research. To act as controls, they were requested to fill out a comparable journal one week later at the same time. Rage levels were higher before receiving an adequate shock for ventricular arrhythmia than during the control periods, indicating that anger can cause ventricular arrhythmias [18]. Anger-Induced Cardiac Arrhythmias were further likely to be polymorphic, PVC- Initiated, and Pause- Dependent, all of which are risk factors for death. A good diagnosis is needed to differentiate the rhythm variation is due to psychological stress or any other reason. Various other ECG variations due to significant mental stress have also been given below to state the link between psychological stress and its correlation between cardiac arrhythmia and various other cardiac pathologies. For a better understanding, a portion of a normal ECG is also shown below.

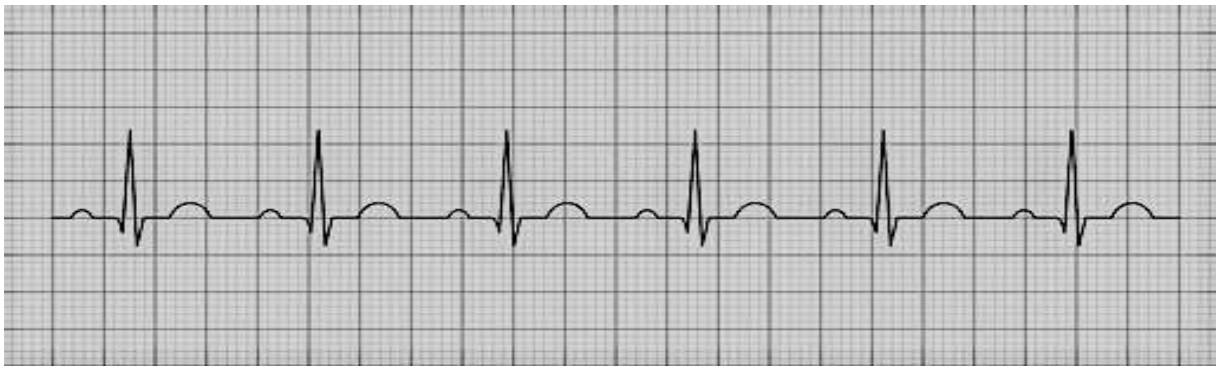

Fig. 1: Normal Sinus Rhythm. Normal P- wave, QRS complex is well arranged and T- wave is also normal in nature. No any abnormalities found in this ECG. PR Interval, QRS Interval and QT Interval seem to be normal [19].

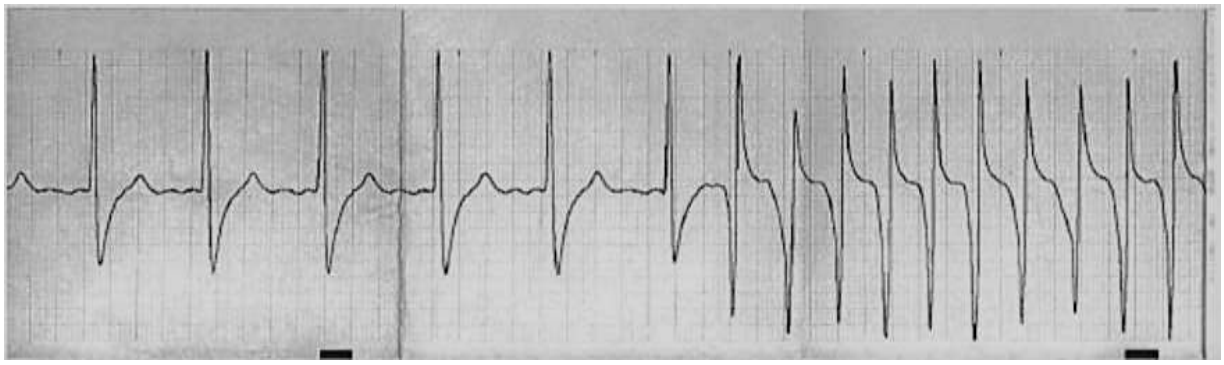

Fig. 2: Monomorphic sudden-onset non-anger generated cardiac arrhythmia [20].

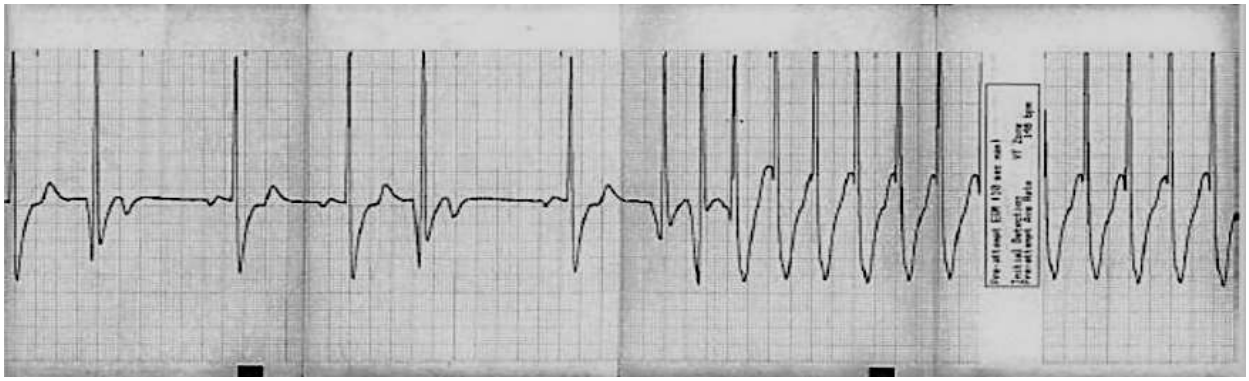

Fig. 3: Monomorphic Premature Ventricular Contraction (PVC) initiated Pause- Dependent Cardiac Arrhythmia. (Anger-Triggered) [20].

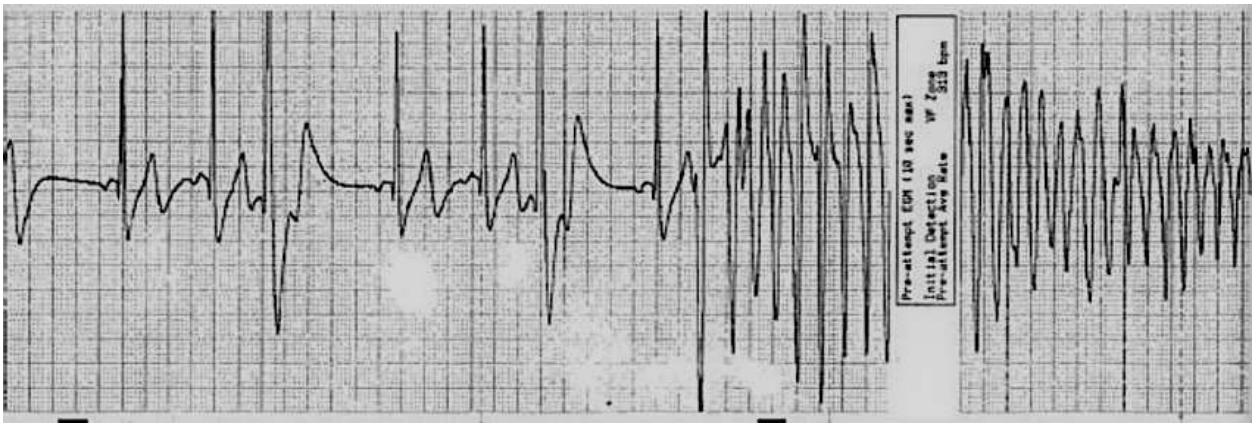

Fig. 4: Polymorphic Premature Ventricular Contraction initiated Pause- Dependent anger-triggered cardiac arrhythmia [20].

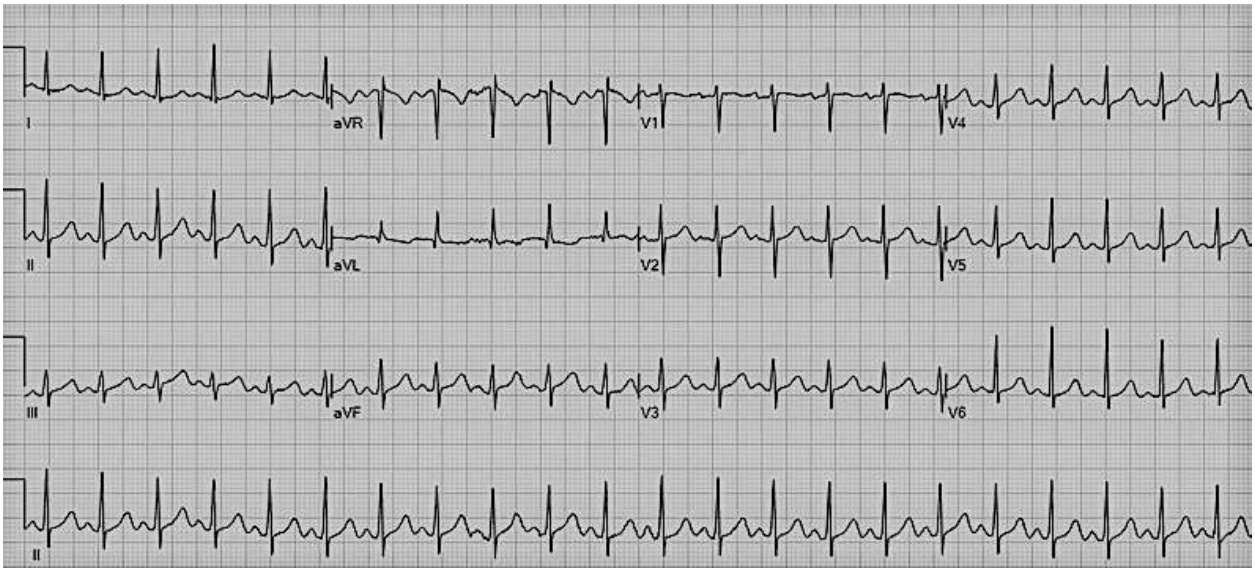

Fig. 5: Sinus tachycardia with estimated approximate 132bpm [21].

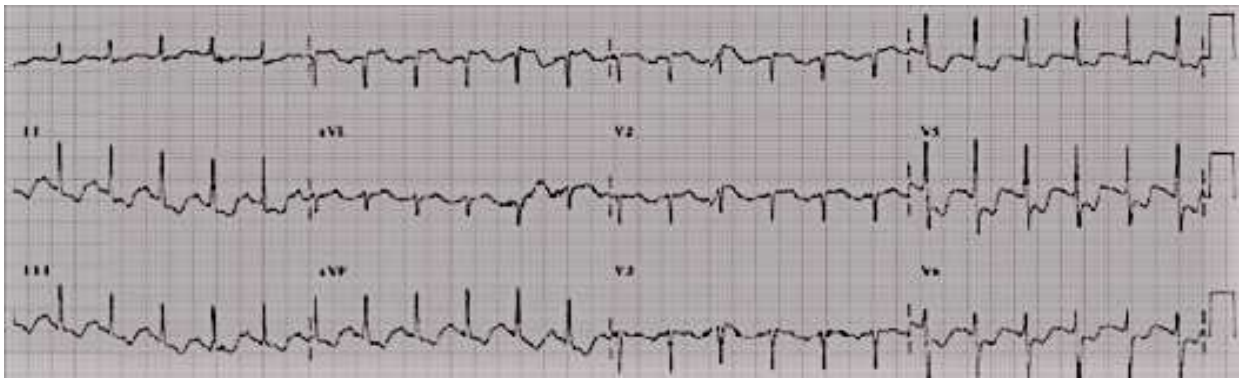

Fig 6: Anxiety induced ST wave depression [22].

Electrophysiological processes of stress-induced arrhythmia can be elucidated using signal processing methods. Toivonen et al investigated QT wave variations under scientifically induced stress via controlled trial using the human model of stress rather than doing the animal study. To attain the result, randomly selected on-call medical house officers have been selected [23]. The QT - intervals were contrasted to periods of physiological and psychological rest with identical heart rates (HR) during times of arousal owing to a page [26]. They discovered QT- hysteresis during stressful situations, with

prolonged QT than at rest, comparable QT hysteresis and it has also been reported during intensive exercise. Overstatement of QT- hysteresis in reaction to physical exercise is hypothesized to be one of the main mechanisms driving sudden death in athletes, and it might also be a component in stress-related SCD [24].

By monitoring T-wave alternans (TWA) throughout an experimental laboratory psychological stress regimen, we looked straight at the contact of psychological distress and its clinically significant outcome on the heterogenic effect of cardiac repolarization as a component of long-recognized factor to develop cardiac arrhythmogenesis. It is possible to induce psychological distress in the laboratory under the supervision of a practicing and experienced psychologist or psychiatrist employing several maneuvers or approaches, somewhat analogous to how we may create physical stress in a TET (Treadmill Exercise Test). These can include requiring the selected patient to execute mental arithmetic, just like serial number addition or subtraction of any three-digit figure within a particular time, or giving an oral/vocabulary-related assignment amid an emotional setting/component including emotional contents in higher level. We use a stressor termed "rage recall" in our lab. Just like if they were addressing a buddy afterward in the day, we also ask the participants to discuss a current occasion that made them upset or furious.

In one clinical trial T- Wave alternans commonly called TWA, T- Wave amplitude or T'amp, and T-Wave area (T area) were systematically calculated at the moment in time - regularity here of influence by using Interbeat middling procedure to analyze the influence of psychological pressure on trio-plane metrics of dissimilarity that can also be ascertained from another systematic monitoring method called Holter monitoring technique: TWA, T amp and T area [25]. A psychological hasslemodus Operandi combining intellectual reckoning and revoke was used on around thirty-three individuals in the connection of implantable cardioverter defibrillators and narration of different ventricular arrhythmias in this investigation [26]. During mental stress, TWA rose from 22uV as a baseline degree to 29uV. Mental distress raised all additional indexes of heterogeneity as well. (For better understanding refer to Figures 7, 8, and 9) during times of stress, Broad- Range repolarization inadequacy, which encompasses Non-Alternans periodicity, rose as well [27]. Mental stress was found to enhance TWA in similar research also [28].

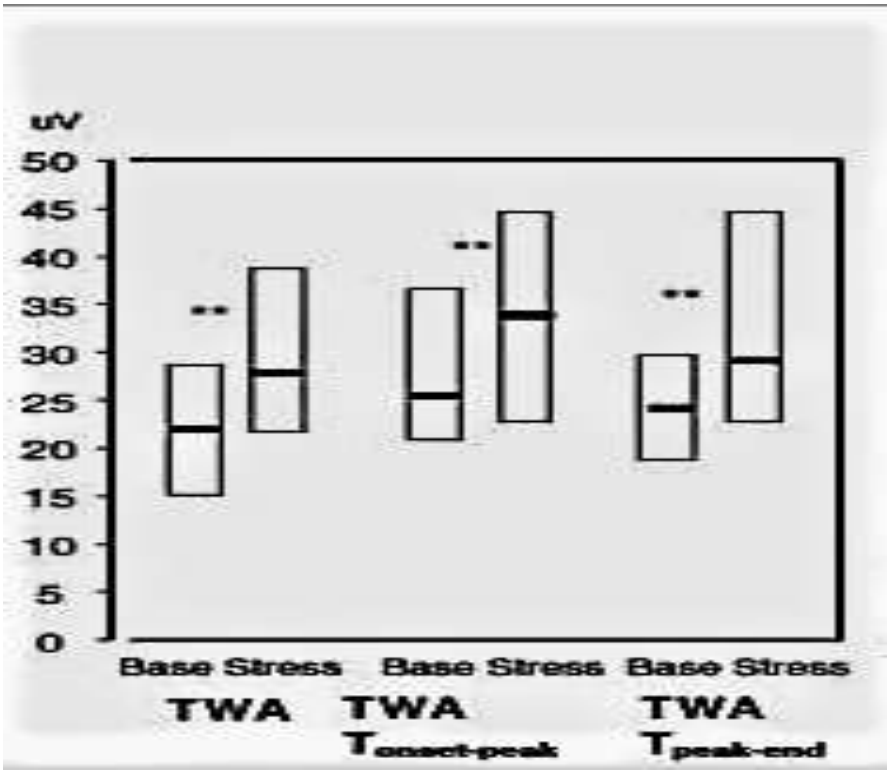

Fig. 7: Repolarization changes due to mental stress. Block plots indicates median and interquartile ranges. TWA= T-wave alternans [20].

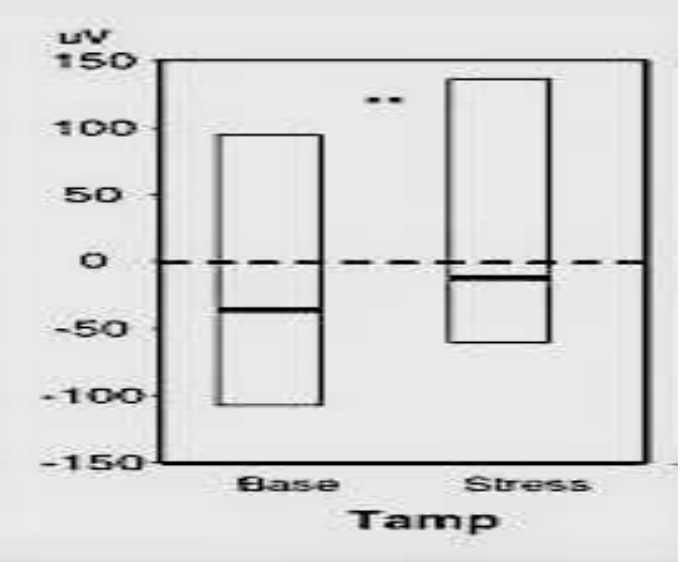

Fig. 8: T-wave amplitude changes [20].

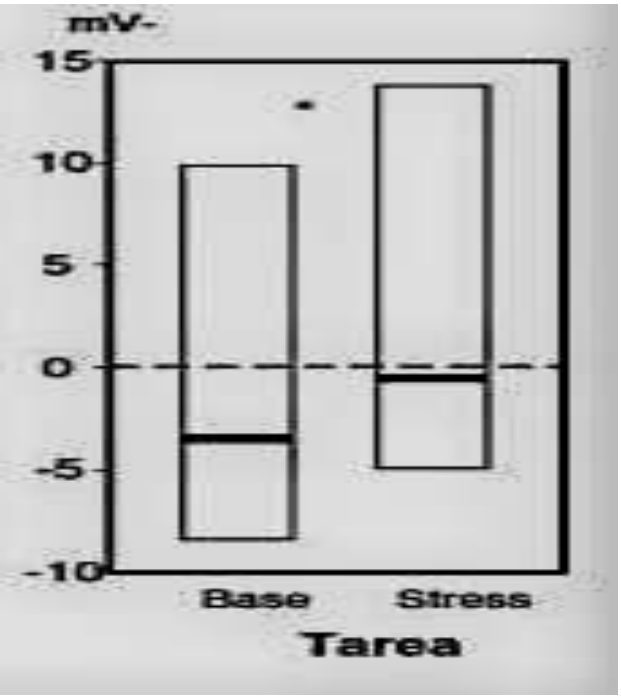

Fig. 9: Tarea = T-wave area changes [20].

There are no recognized pathways via which emotional stress might enhance TWA [29]. Kop and colleagues were capable of ascertaining that the effect of rage on T- Wave alternans was distinct from ischemia by using concurrent SPECT-perfusion imaging [30]. TWA can be affected by increasing heart rate solely (for example, by atrial pacing), however, there was a little rise in heart rate with mental stress in our research (as is typical with mental stress). We did notice a link between increases in TWA and catecholamines, implying a straight sympathetic action on cellular repolarization. Previous research has also demonstrated that sympathetic activity of the body, in addition to the effects of heart rate, may enhance TWA. TWA is abolished by patellectomy and increased by stellate ganglion stimulation in experiments [31]. Intravenous beta-blockade [24] reduced the size of TWA in clinical investigations, and TWA caused by exercise is larger than that induced by atrial pacing at the same heart rate [32].

The effect of rage on TWA in the lab predicted real-life arrhythmias. In a follow-up investigation of our anger and T Wave Alterens study, we discovered that anger-induced T Wave Alterance was a powerful determinant of arrhythmia, well with risk of ICD-treated ventricular arrhythmia for those in the upper quartile of anger-induced in the laboratory being over 10 times higher than other subjects (Figure 7). After correcting for traditional arrhythmia predictors including ejection fraction, preceding acute arrhythmia, and broad QRS, anger-induced TWA stayed prognostic [33]. Numerous research findings have also shown that TWA determines future arrhythmia, notably, anyone using the time-domain method of analysis, like the Modified Moving Average procedure, about which our Interbeat Average is similar and a recent consensus result supports its use, scientific studies involving over 12,000 patients: "Overall, we believe that TWA examination should be considered if there is a concern of sensitivity to deadly cardiac arrhythmias [34]. Furthermore, there is no compelling proof from interventional studies that it can be used to guide therapy at this time [35]. Time-domain approaches have the benefit of not requiring an elevated heart rate (as spectral methods do), allowing investigations to be conducted while on prescription beta-blockers. The predictive value of the test is dramatically reduced when beta-blockers are used [36]. This conclusion is consistent with the observations about the influence of autonomic variables on TWA: TWA testing performed in an autonomic state comparable to the patient's typical condition is more predictive of future outcomes. Our investigation was hampered by its size, the fact that it only included males, and the fact that it used corrected arrhythmias as an endpoint at an age before evidence-based programming. The strong predictive value found here, though, provides an interesting prospect: experimental mental stress-induced TWA may be an even greater sensor of arrhythmia risk, as this approach captures the interplay of activator and substrate that can lead to arrhythmogenesis.

6. OVERVIEW OF HOW ECG VARIES DUE TO SIGNIFICANT PSYCHOLOGICAL STRESS IN THE ATRIUM :

Stressful stimuli have been linked to AF in small case studies. In two studies published in the year 1968 and 1999, two to thirty percent of AF occurrences were caused by "emotional or physical weariness," with particular cues identified as a family funeral or waking up to an alarm [37]. The first prospective research of emotional AF triggering was just published. Ninety-five patients with sporadic AF finished an e-Diary inquiry of their emotions such as anger, anxiousness, despair, strain, and pleasure for the foregoing (proximal) thirty minutes, (Figure-7) moreover at the end of every date, abbreviation of patients emotions have been analyzed in the particular day [38]. Emotions recorded on the e-Diary for the thirty minutes leading up to arterial fibrillations were analyzed to individuals who recorded cardiac rhythm during the last 24 hrs using the Holter monitoring technique to make a correlation in sinus rhythm. End-of-day emotion summaries for days before a day with AF were contrasted to end-of-day mood summaries for days leading up a day without AF in the same way. Forty participants reported a total of 228 symptomatic AF occurrences [39]. On the e-Diary, there were one hundred and sixty-nine episodes with proximal sensation reports, and approximately eleven thousand five hundred and sixty-three psychological sentiment reports were identified all through Holter-long-established sinus rhythm, approximately one hundred and twelve ends of day summation sentiment entries past time amid incidents of atrial fibrillation, plus approximately 14663 ends of day summation sentiment findings proceeding days without AF. Sense of loss, worry, aggression, and stress all raised the risk of an AF event by 2-5 times. Pleasure reduced the incidence of AF by 85%. Rage and tension, as measured by end-of-day emotion summaries, both increase risk of AF the next day. The processes behind stress's arrhythmogenic impacts on AF are less well known. In most but not all investigations, sympathetic stimulation reduces repolarization as evaluated by the atrial effective refractory time in invasive tests. Furthermore, when paired with vagal impulses, sympathetic activation has a synergistic effect [40].

The signal-averaged P wave may be used to quantify atria conduction noninvasively, and a lengthy SA – P period has been linked to recurrent AF. Sympathetic stimulation with isoproterenol diminishes SA-P duration in the normal atrium; beta-blockers, on the other hand, impede conduction. After the beta-blockade, atropine reduced the SAP period demonstrating that Vagal stimulation delayed conduction. Furthermore, P- Wave duration determined since 24- hrs Holter surveillance is reduced during the day, indicating that variations in sympathovagal balance affect atrial conduction [41]. We've focused on the impact of psychological distress in people with significant AF who have aberrant Atrial Conduction. In one clinical research, approximately 97 patients who are suffering from AF and around 25 controlled volunteers were subjected to comparable mental stress assessment in the lab as stated afar

[42]. By and large, P- Wave conduction duration was greater than at its normal baseline also at some stage in aggression than in controls, although both groups' P-wave duration reduced with anger comparably [43]. Furthermore, late potentials rose in AF patients but reduced in controls, as measured by RMS-40, meaning Root Mean Square Voltage of the last forty mille seconds and there was some negative correlation in between the shift in P – Wave duration as well as the variation in RMS-40 in AF patients [44]. The electrophysiological processes behind this discovery have yet to be discovered. It is also likely that anger-related organic autonomic alterations exacerbated the pre-existing diverseness of cardiac conduction in individuals with established AF base, case in point, by decreasing cardiac conduction periods in normally healthy tissues while leaving defective tissue unchanged, resulting in AF susceptibility [45].

7. MENTAL STRESS AND VENTRICULAR ARRHYTHMIAS :

Investigations of population stresses have revealed that anger and other unpleasant emotions can cause unexpected mortality [46]. Anger has been shown to cause ventricular arrhythmias in people with implanted defibrillators in clinical research. Arrhythmias are also made more vulnerable by long-term unpleasant emotions [47]. Autonomic alterations, which modify repolarization, presumably increased in individuals having Sympathetic Denervation and which in return produce possibly fatal significant Polymorphic Ventricular Tachycardia are among the mechanisms relating to rage and arrhythmias. In individuals with implanted cardioverter defibrillators, treatments that reduce emotional distress and the resulting autonomic reactions may be beneficial [Fig 10 and 11].

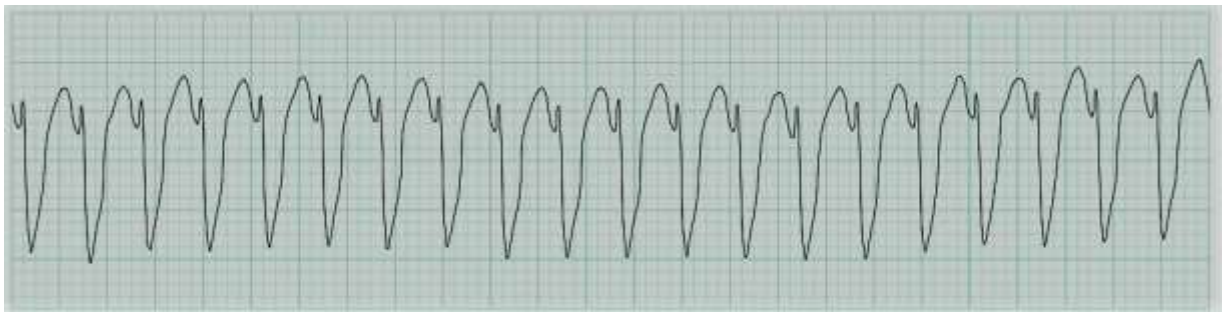

Fig. 10: Monomorphic ventricular tachycardia (Regular and broad complex in nature) [48].

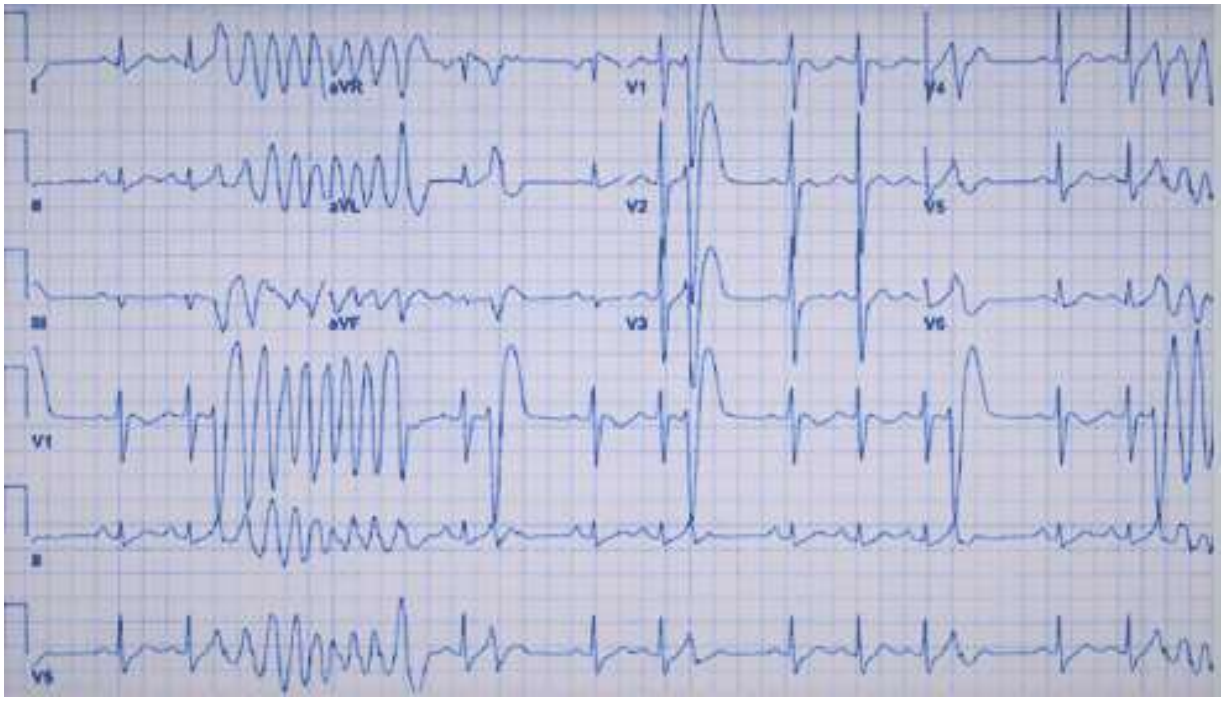

Fig. 11: Intermittent polymorphic ventricular tachycardia [49].

8. SIGNIFICANT CLINICAL IMPLICATIONS :

Depending upon our systematic clinical analysis and several other researchers' discoveries that anxiety or any other significant mental stress may cause atrial or ventricular arrhythmias and in some extreme cases periodically changing pattern of both of these symptoms can be seen and it appears exceptionally prone that therapies focused at reducing mental strain or physiological impact of psychologically significant stress on the body, will reduce cardiac arrhythmia incidence in vulnerable individuals. The above-mentioned ECG indicators of psychological stress can act as submitters while also providing mechanistic information. We're testing if an eight-week stress management program targeted for lowering depressing feelings will help people with ICDs have fewer arrhythmias. This particular clinical study is also trying to test if the tension decline technique can decrease the elevation of significant TWA associated with the laboratory stress test [50]. The result is also stated above to fully understand the mechanisms of action. Yoga and other complementary therapies have been demonstrated to reduce atrial and ventricular arrhythmia signs. To reduce stress-induced arrhythmias, more study is needed into both classic psycho-educational and complementary treatments.

9. CONCLUSION :

Both atrial and ventricular arrhythmias can be associated with stress. ECG stress profiles can give mechanistic insight as well as serve as surrogate endpoints in investigations looking into treatment options. However there are a variety of methods for assessing repolarization as well as repolarization heterogeneity in the ventricle, there are fewer alternatives for studying atrial electrophysiology noninvasively, and that is an important subject for future studies.

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