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Electrocardiographical myocardial ischemia and its clinical aspects

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ELECTROCARDIOGRAPHICAL MYOCARDIAL ISCHEMIA
AND ITS CLINICAL ASPECTS

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A good portion of the following material concerning the present day concepts of electrocardiographical myocardial ischemia, will be dealt with in terms of the spatial vector method. This method has been selected due to the dominating influence of Grant's "Clinical Electrocardiography- The Spatial Vector Approach" which initiated and guided many of my thoughts pertaining to the subject. In addition, selection was also based on the fact that "pattern reading and method" can easily be derived from vectorcardiography once the latter is understood, whereas the converse is not always so.
1.

The term "myocardial ischemia", is frequently used in medical practices today. Yet there are few such concepts which are so frequently misinterpreted and so genuinely misunderstood. Many are able to associate it with angina pectoris, depressed ST segment, pre-infarction state, digitalis effect etc. However, few can accurately correlate and evaluate these features and arrive at a pathophysiologically sound clinical-diagnostic picture. This paper will attempt such a correlation in hopes of alleviating such fallacious assumptions as are often postulated with regard to the subject.

Not intended to be an all encompassing volume of multiple facts and viewpoints, this paper endeavors to present important components in brief and fit them separately in to their places, hopefully arriving at a more clearly understood term of myocardial ischemia.

In the pages which follow, a knowledge of the physiologic basis for cardiology and its resultant manifestations is assumed on the readers part. It is felt that exclusion of such basic information is necessary for the clarity and brevity of this paper.

Due to a multitude of vague concepts and terms pertaining to the subject, it appears that establishment of an adequate definition is of paramount
importance initially.

The term "myocardial ischemia", is one to which various accurate definitions have been assigned by various branches of the field of medicine. The physiologist defines ischemia as a chemical stimulus which frequently produces visceral pain. It is felt that the pain is produced by the presence of abnormally large quantities of lactic acid, pyruvic acid, and perhaps a number of phosphoric acid compounds which are released into the respective tissues undergoing anerobic metabolism. In the heart, this anerobic environment and hence ischemic pain is often attributed to either physical narrowing and / or produced spasm of the nutrient coronary arteries. The frequently associated angina pectoris will be discussed in a future section. (4.662) The pathologist on the other hand used ischemia as a term applying to an area of tissue or the body which demonstrates a diminution or obliteration of its blood supply. When the specific area is myocardium, the subsequent pathology is specific to that locale and is consistent with degenerative changes, atrophy of structure, and loss of parenchymatous cells with replacement by fibrosis and / or adipose tissue. These changes are
found with gradual but incomplete diminution of blood supply with some degree of collateral circulatory insufficiency. (1.p.123)

If we are to combine these two concepts, the following conclusion is derived. Myocardial ischemia is that portion of myocardium which is undergoing anerobic metabolism secondary to an insufficient blood supply. The production of abnormal quantities of metabolites and the inability to rapidly rid the specific region of these compounds appears to be causational for the frequently accompanying angina pectoris.

Historically, the search for adequate correlation of the above definitions and for additional information pertinent to the subject was hardly realized or even postulated prior to the eighteenth century. Many of the old masters such as da Vinci, Versalius, Galen, and Harvey evidenced cognicence of coronary artery presence via their fine anatomical illustrations, but none were aware of their function.

Montpellier, Bellini, Desenac etc. were not only aware of the coronary arteries anatomy and course, but they also made many well documented observations as to their ossification, marked sclerosis, and
luminal obliteration at necropsy. Morgagni recorded a multitude of cases in which the patient exhibited many of the present day symptomatologies of angina pectoris, coronary thrombosis and occlusion and later at autopsy revealed coronary changes. In general, even these persons were not able to clearly outline coronary function or suggest correlation between symptomatology and pathological findings.

Heberden drew his remarkably accurate clinical picture of angina pectoris in 1768 but made no attempt to postulate etiology. Young Edward Jenner and Caleb Parry were among the first to believe that the ossified and narrowed coronary arteries which were found at occasional necropsies, were in fact the pathological lesion which was responsible for the anginal syndrome. They helped to substantiate their hypothesis by going as far as to accurately predict prior to his death or autopsy that coronary changes would be found in their friend and mentor, the great John Hunter who had suffered from angina for years.

Hence, it was the English physicians who are credited with the origin of the "coronary theory" as it later became known, and in his volume of 1779 Parry revealed a clear conception of the ischemic
heart muscle and its gross etiology of production. Throughout the following century this theory was greatly disputed between its respective proponents and antagonists. It was by frequent experimentation, the recording of clinical histories, and the constant observations of such pioneers as Virchow, Conheim, F. Smith, RennefMarie, and Cohn, to only mention a few, that further assisted in the establishment of this theory as nearly irrefutable.

It was not until the late 1800's and early 1900's that the present day's cardiologist's theory of myocardial ischemia began to meld however. For it was during this period that Mackensie's polygraph, Einhovern's electrocardiograph, and the work of Sir Thomas Lewis became available. These combined with the efforts of such men as Bayley, Wilson, MacLeod, and Grant gave to us the following present day concept of myocardial ischemia.(7.p.19-32)

Of the above four investigators, Grant's interpretation is one of the most recent and straightforward explanations of the subject that is available, and in the main it imitates most of the other's viewpoints regarding ischemic electrocardiography. Dividing his criteria into two categories,
Grant feels the following T and S-T changes are characteristic. (3. p. 85-107)

The T wave change is described as a primary T vector abnormality. These are differentiated from secondary T vector abnormalities in that the latter are due to conduction defects. The former primary changes are indicative of an altered myocardial cellular metabolism which results in delayed repolarization at the affected region. Since the T vector from the uninvolved or non-ischemic regions of the heart tend to dominate the mean T vector, it characteristically points away from the affected region of the myocardium. The explanation of this and S-T change will be dealt with in the forthcoming section of pathophysiologic correlation with the electrocardiograph. A typical mean T vector is found to point anterior and to the right since the left ventricle is so frequently involved.

Since in pure ventricular ischemia, QRS complex alteration is not present, an abnormal wide QRS-T vector angle is resultant and hence is considered as a frequent finding in ischemic vector analyses.

A concomitant vectorcardiographical change which is characteristically found in the presence of
7.

mild myocardial injury or myocardial ischemia is the production of a S-T vector which is caused to point away from endocardial ischemic regions and towards epicardial areas of injury. This resultant S-T vector is manifested through electrocardiographic lead tracings best monitoring the ischemic area. These leads reveal a ST segment displacement from the base or isoelectric line. This displacement is usually found in the form of ST depression.

The next section contains an explanation of the pathophysiology which produces the above changes and will merely be a generalized description of the more pertinent factors involved.

In a broad sense, it can be stated that myocardial insufficiency can be caused by (1) a primary reduction in oxygen delivery to the coronary tree and / or (2) a primary increase in cardiac work so that the usually adequate oxygen supply is no longer sufficient for maintainence. A primary T wave change is secondary to such an oxygen deficiency state which in turn produces an altered myocardial cellular metabolism. (12.) This abnormal process is causative for a delayed repolarization at the affected site. Since by definition, the T wave is a specific indication of
ventricular repolarization, a delay in the process will usually be revealed by an altered configuration of the T wave. Present day theory has it that repolarization is indicative of restoration of membrane semipermeability. (3.p.66) To accomplish this restoration, it is necessary for cellular metabolic work to bring about ionic movement and therefore return the cell to the charged or polarized state. Under ischemic conditions, this active process is slowed with resultant alteration in T vectors generated from the ischemic region. Since the process is ultimately accomplished, and the more passive and somewhat spontaneous depolarization is once again brought about, there is characteristically no QRS vector or complex change and hence the rather specific production of a wide QRS-T angle. Since the instantaneous T vectors from the uninvolved ventricular regions dominate the mean T vector, the latter is correspondingly obliged to point away from the affected or ischemic region. Statistically, it has been found that the left ventricle is most frequently ischemic and most often infarcted. (8.) Ideally then, this would produce a mean T vector which would point anteriorly and to the right in the opposite direction
as the structure involved. For such vector production, depressed or inverted T waves in leads I, and AVL, nearly isoelectric in leads AVR, V5, and V6, and elevated in V1 and V2 would be characteristic of ischemic pattern qualities.\(^{(3.p.89)}\) The QRS complex is unaltered as is the corresponding mean spatial vector. There is no change in the magnitude of the repolarization vector.

In discussion of the second diagnostic category, Dr. Grant suggests that the phenomenon responsible for S-T changes is "a type of injury intermediate in severity between defective repolarization (producing vector abnormalities) and outright cellular death (producing QRS vector abnormalities)".\(^{(3.p.101)}\) Due to this injury, the cell is no longer capable of maintaining the polarized state. The charge presumably "drains" or leaks off the membrane due to the primary defect in semipermeability which is again precipitated by the lack of oxygen to the tissues. The resultant flow of current due to the constant "leakage" is recorded on various EKG leads as deflection from the isoelectric line and is classically described as S-T depression. The accompanying diagram from Grant,
10.

helps to explain why the so called depressed S-T segment is merely a more apparent and easily measured index which is secondarily created by an initially elevated T-P segment. (3.p.102)

Guyton's theory of S-T depression or "current of injury" is similar to this. The abnormality is explained as "being due to a damaged or ischemic area of heart which remains depolarized at all times and which sets up a constant flow of current between the pathophysiologically depolarized portion and the normal polarized areas." He feels that during ischemic conditions, energy required for repolarization is simply not available. Therefore it is postulated that a current of injury occurs when the refractory period of a specific portion of the myocardium becomes so prolonged that this muscle fails to repolarize prior to
11. the next cardiac contraction or depolarization of the surrounding normal muscle. (4, p. 313)

The resultant S-T segment depression produces a nonisoelectric entity and therefore a S-T or "injury" vector. If the injury of ischemia is confined specifically to the epicardium musculature, as is the case with pericarditis and myocardial infarcts, the S-T vector points toward the area of injury. In angina pectoris, myocardial insufficiency, or sub-endocardial infarcts, the resultant injury is most frequently confined to the endocardium, and in direct opposition to the T vector, the "injury" vector points away from the ischemically involved area. (3, p. 102)

These diagrams summarize and combine the two classically diagnostic vectors which might be found in uncomplicated left ventricular ischemia.

Fig. 2

Vector direction is dependent upon:
1. Heart position
2. Area affected
3. Layer affected - endocardial vs. epicardial

In the previous two sections, the definitions and necessary criteria for the diagnosis of myocardial
ischemia have been laid down in a clear-cut fashion and have in general pertained to the findings consistent with the pure or unadulterated form of the subject. In actuality, ischemia is probably an entity which is frequently found in conjunction with and complicating many other forms of cardiac pathology. A complete and detailed differential diagnosis of such abnormalities could comprise a small volume in itself. The following will be a succinct consideration of a few of the more pertinent and most frequently confused states. As was done with "ischemia", they shall be dealt with in their uncomplicated classical modes of presentation.

A differentiation between certain conduction defects i.e. left bundle branch block, and myocardial ischemia is one which many a physician might encounter. As was previously brought out, the T vector abnormality produced by a conduction disturbance is a secondary T wave defect and hence is dependent upon the initially produced QRS vector defect. The latter's presence is conspicuously absent in the classical ischemia vectorcardiogram. The accompanying shift of the S-T segment produced in this conduction defect is attributable to forces of
repolarization and not to a current of injury. Here again, we find the defect to be secondary and dependent upon the initial QRS complex changes. A necessary increase in magnitude of repolarization vectors joins the growth of the QRS complex area and results in an enlarged T vectors. These in turn alter S-T segment position. Hence it is found that in conduction defects the S-T and T vectors most often are parallel and pointing in the same direction. When current of injury is present, these spatial vectors are usually not parallel. (3.p.107)

A method which is often diagnostic between the two types of T wave abnormalities is that of utilizing Frank Wilson's ventricular gradient. (13) This method is diagramed and explained in the following:

Fig. 3

Diagram A. is a control showing the relative magnitude and direction of the normal VG or ventricular gradient. It will be noticed in B that although the mean QRS and T vectors have changed in both magnitude and direction the VG remains equal in both direction and magnitude with that in A. According to Wilson, the absence of ventricular gradient change is indicative
of secondary T wave changes. The VG change in C. is felt to be evidence of primary T wave change.

Diagram D. reveals an unchanged VG but an abnormal wide QRS-T angle. Grant feels this is not consistent with primary repolarization abnormalities since VG remains the same. (3. p.133)

In today's vastly expanded and improved world of medicine, the electrocardiographical picture which most frequently mimics and defies differentiation from myocardial ischemia, is one which is produced by the physician himself. The produced changes are resultant from an entirely different pathophysiologic origin. The vector and pattern lead findings are so typical of the ones attributable to "ischemia", that most experienced internists and cardiologists today probably do not attempt to definitely distinguish between the two entities unless indicated. The similarity in question is produced by the effect of digitalis, its derivatives and the more modern synthetics.

As with conduction disturbances, the digitalis influenced vector cardiographic changes are due primarily to altered repolarization. The drug accomplishes this in three ways. Initially it is able to produce a more rapid repolarization and hence produce the characteristic shortened Q-T interval. As repolarization time is shortened, the depolarization
sequence through the ventricles is more accurately traced by the forces of repolarization. As this comes about, the resultant mean QRS and T vectors become even more parallel and in the same direction with one another. Under normal circumstances, the slower repolarization time prevents this to an extent. Secondly, the digitalis is apparently responsible for producing a more efficient myocardial contractility which in turn is responsible for a reduced pressure gradient across the ventricular wall thereby causing repolarization and depolarization to be in more direct opposition with each other. Finally, and somewhat dependent upon the previous two digitalis effects, the mean T vector is found to be exquisitely responsive to the systolic-diastolic heart sizes and is seen to rapidly shift direction when the cardiac input and output mechanics are altered even slightly. (3.p.96)

Upon digitalizing, the produced mean S-T vector is frequently found to be in a 180° opposition with the mean QRS vector. Once more this is attributable to the earlier onset of repolarization and it becomes apparent from this that the S-T vectors also are representative of repolarization.
Although it is seldom done routinely in the daily reading of EKG's, Grant makes a hypothetical separation between digitalis effect and that produced by "ischemia" on standard electrocardiograms. It was felt that digitalis characteristically produces a mean T vector which is reduced in magnitude but not significantly altered in direction, while ischemic T vectors tend to change direction and hence widen their angle with the mutually stable QRS vector. The lack of "j" junctional depression with the hammocked shaped S-T segment is also found to be consistent with digitalis administration. These are by no means pathognomonic or diagnostic features pertaining only to digitalis effect. They are mentioned here so that along with the results of history, physical and other studies, they too can be used as check points in the lengthy process of ruling in or out possible etiologies.

Most sources consulted for this paper have been quick to bring out the fact that a great number of separate or coexisting processes may obscure or simulate the ST-T changes which have previously been described. The obscuring entities frequently mentioned were; tachycardia, ventricular strain, hypermetabolism, shock, hypertension, aortic valvular
disease, electrolyte disturbances, pericarditis, valsalva manuever, head tiltings etc.. It may be seen from this, that many other factors must be taken into account and used to supplement the EKG results in order to arrive at a valid diagnosis.

Up to this point in the paper, the reader may certainly have questioned the objective importance of the subject many times. An explanation of this has purposely been withheld up to this point so that adequate tribute to the following topic might be given. Prior to its advent, the presence of "ischemia" on the EKG was poorly correlated with accurate data, possible pathologic progression, or specific therapeutics.

In the period between 1925-1929, two scientists by the names of Masters and Oppenhiem, made their initial attempts at proving the theory that by increasing cardiac output and work load via exercise, that the correspondingly increased coronary perfusion would be inadequate in supplying greater oxygen needs of the myocardium if significant coronary artery obstruction or spasm existed. In other words, they simply expected to create an ischemic myocardial state in those individuals with inadequate coronary
flow under conditions of stress. Through the years the exercise test and its criteria for positive results have been frequently revised and modified. (9) The present method appears to be statistically effective in exposing most coronary artery disease victims whose resting 12 lead EKG's are essentially normal. It has only come into significant usage across the U.S. in the past decade. Master's states "extensive clinical experience has shown EKG findings are positive for ischemia following exercise in nearly 97% of patients with coronary disease". Furthermore, he and his proponents feel that by uncovering this ischemic tendency early, the individual's chances are significantly better in avoiding the possible progression to coronary occlusion and myocardial infarction. Naturally this statement is qualified to the extent that this same individual receives and follows proper therapeutic and prophylactic care and counseling.

The essentials of the test entail the patient making a specified number of trips up and down two nine inch high stairs. The number of trips are dictated by the age, weight and sex of the patient via a published chart. One half the number of trips are
performed in \( \frac{3}{2} \) minutes and is known as the "single two step test". The double two step test is performing the number of trips specified on the chart in a total of three minutes. In a recent review of his test, its author explains the criteria and considerations which he attends to prior to the testing. It is felt that close attention to detail of the following check list adds materially in the production of his few false positives and complete patient safety. These points are in general agreed upon by other sources consulted.(10.)

1. basal conditions - medications and smoking discontinued on the evening prior to testing.

2. good previous nights rest.

3. tranquil testing atmosphere with at least 10 minutes rest before test.

4. physical exam prior to test to rule out acute illness or the possibility of impending infarction.

5. a normal resting EKG prior to exercise.

6. complete explanation of procedure to the patient.

7. instruction to the patient that he is to immediately cease all exercise upon experiencing chest pain, discomfort or abnormal sensation.

Having fulfilled the above requirements, the patient is instructed and carefully timed in the
performance of the single two step test. Upon completing the prescribed number of climbs, the patient immediately lies down and the chest and limbs leads which were worn throughout the test are hooked into the machine. (Masters uses only leads II, V3, V4, and V5.) Tracings are taken immediately following hook up at 2 minutes and at 6 minutes. Should these results prove negative, the double two step test should always be done in an hour or so or preferably on the following day.

Originally, Master's criteria for a positive or abnormal two step test was the presence of an RS-T depression of 0.5mm or greater in the post exercise EKG's. He felt that such a finding was indicative of myocardial ischemia and consistent with the presence of definite organic coronary disease. These criteria went unquestioned and were held as valid until the mid 1950's. It was during this time that the method began to grow in popularity and numerous investigators and clinicians became familiar with its use. Coexistent with its increased utilization, there was a great percentage increase in the number of false positive tests which were later confirmed at autopsy. Many of the individuals using Master's
method were obtaining false positive incidence as high as 25-40% even though they apparently adhered quite well to the prerequisites etc.. The chief complaint appeared to be that the author's criteria failed to differentiate adequately between the valid "ischemic" type RS-T segment depression and the often false positive "j" junctional depression of the RS-T segment. Many critics came to feel that configuration of the depressed segment and not just mm's of depression should recieve some attention and be included in the definition of a positive exercise test. The 1956-57 reports of Robb and Mattingly appeared to establish a statistically valid difference between post-exercise "ischemic" and "j" junctional RS-T depression. They arrived at the conclusion that "any depression of the RS-T segment which is consistent with an initial horizontal configuration of at least 0.08 sec. or a definite sag of that segment was abnormal". In addition they were in agreement that depression confined to the junction of the QRS and S-T complex (j junction) was of intermediate or little significance. (8.)

These and other critics caused Master's to take a new and objective assay of his original findings.
Percieving that he had failed to take the "j" junctional RS-T depression type into account initially, he went on to review his last consecutive 596 patients in 1961(10.) and arrive at the following altered criteria for a positive Master's two step exercise test and the significance of its absence.

1. The absence of RS-T depression or a negative double two step test is exceedingly helpful in that it provides at least 30:1 assurance that no impairment of coronary circulation or myocardial nutrition exists in that patient. Hence the investigator is obliged to consider other and extracardiac reasons for the patient's initial complaints.

2. The QT interval respective to heart rate should be measured in the rare case of a negative double two step result although the history and clinical picture strongly suggest the presence of the anginal syndrome, a definite hypertension is in evidence or the patient is recovering from a well documented "coronary" heart attack. Should the actual QT interval prove to be 10% or greater than the ideal QT time for that heart rate, then the double two step test of that individual should be considered abnormal.

3. A RS-T segment depression which is completely horizontal or sagging for at least 0.08 sec., is presently felt by the author to be "ischemic" in nature. In addition, he considers the presence of the above (#3) to be at least 95% indication of significant organic heart disease or myocardial nutritional defect. This he holds to be true irrespective of the depth of the segmental depression.

4. Unlike many of his critics, Master's has found that approximately 30% of his patients who demonstrate "j" junctional depression only to
have evidence of organic involvement capable of producing myocardial ischemia. He limits this criteria with the following:

a. Junctional depression of 2mm or greater is nearly always evidence of coronary artery disease or obstructed nutritional flow to the myocardium.

b. Should "j" depression be less than 2mm, the QX/QT fraction is measured and pronounced as being consistent with ischemia if it is more than 50% and if the corrected QTR is more than 1.08. (adequate explanation of 4.b. is available in reference 10.)

The use of this exercise test has been proven effective statistically in exposing the greater majority of patients suffering from significant occlusive coronary disease. Eventhough no such evidence of this is existent on the resting 12 lead EKG, many positive exercise tests for ischemia are later confirmed at follow-up autopsy. It has also been well established by many investigators that those individuals whose EKG patterns and vectors fulfill either past or present criteria for a positive Master's double two step test reveal a definite increased incidence in future fatal and non-fatal coronary occlusions. Upon institution of the more recent and up to date diagnostic features for a positive Master's, quantity and frequent false positives have been sacrificed for a decreased volume of affirmative tests and greater
accuracy in diagnosis. Lepeschkin postulates that a patient with normal Master's double two step test has a life expectancy eight times greater than if he had a definitely abnormal response. However, he and other sources feel that no complete certainty exists that a patient might not suffer a fatal coronary occlusion just a few hours after a completely normal exercise test.\(^{(6)}\)

Without great attention to the detail of definition or mechanism and etiology of production etc., I feel it necessary to mention what significance should be attributed to associated anginal pain with myocardial ischemia and how specific its presence is to diagnosis.

Although much more is known today regarding the mechanism and etiology of angina pectoris, it still remains as a vague entity with a multitude of presenting variations. Harrison defines it as a "steady, unwavering chest pain which appears on exertion and begins to subside promptly upon resting. When such a history is present and the patient receives unquestionable and rapid relief of pain following adequate sublingual dosages of nitroglycerin, the diagnosis may be considered established.\(^{(5)}\) Through years of experimentation and clinical experience, most investigators today consider the origin of classical angina to be
ischemic myocardium.\textsuperscript{(2, p. 614)} In general, it is equally acknowledged that the deficiency is most frequently precipitated by occlusive coronary artery disease. It is important to emphasize that it is the presence of anginal complaints in the individual which frequently lead to the search for and ultimately the finding of electrocardiographic myocardial ischemia. The production of chest pain during a Master's test calls for immediate cessation of testing and the assumption that the test is positive.\textsuperscript{(9, 10)}

A vast amount of studies have been done on angina pectoris since Herberden's original description nearly two centuries ago. However, the published data to date attempting to ascertain what proportion of angina sufferers actually demonstrate myocardial ischemia on EKG still appears to be inadequate. Explanation of this fact can partially be attributed to such factors as persistence of unclear and hazy concepts of angina pectoris, the presence of a-typical angina, the additional effects of associated or co-existing pathology etc.

In the evaluation as to the possible clinical importance of myocardial ischemia, it is plausible to associate and even link this entity with the number
one cause of morbidity and death among civilized people today. This notorious killer is of course, coronary heart disease. The pathologic progression of this disease characteristically incorporates or is causative for myocardial ischemia in both its early and late phases. As this disease progresses it seems to have been fairly well established that actual acute occlusion or infarction is preceded in 50% of the cases by premonitory symptoms and signs accompanying the ischemic tracings. When present these findings are referred to in today's literature as impending infarction state or "preinfarction syndrome". Master's states in a recent publication, that it is vitally important to recognize these signs as early as possible in hopes of instituting proper therapy and prophylaxis immediately and thereby bettering the individuals chance of avoiding a clinical infarction. Statistical data is not as yet available, but he and others are of the opinion that only a small percentage of individuals manifesting signs of the pre-infarction syndrome will proceed on into the end stage or actual infarction. The major portion of these patient's findings subside in several weeks. Hence, it is the small proportion who ultimately will continue on into
27.
coronary occlusion and infarction that his article is pointed towards. There is apparently no method at present for detecting these individuals beforehand.

Although other investigators and clinicians may add or subtract from this list, the following are Dr. Master's check points for diagnosing the preinfarction state:(11.)

1. An abrupt increase in the frequency, intensity and / or the duration of a previously mild to moderate anginal pain.

2. Increased dosages of nitroglycerin necessary for anginal relief.

3. Significant decrease in the amount of stress or exertion required for the precipitation of anginal onset.

4. Onset of angina pectoris while at rest or asleep.

5. EKG findings of RS-T depression or T wave inversion which were previously absent.

6. Laboratory studies: WBC>10,000, Sed. Rate>30, SGOT>40 ± LDH elevation.

Correspondingly, he advocated the treatment of partial bed rest with only minimal activity, small sedative doses, moderately increased nitroglycerin dosages, and possible anticoagulation. The latter being a matter of personal preference. It is his opinion that anticoagulants have not as yet been shown to be of statistically significant value in the ultimate
prevention of myocardial infarctions at least where the impending infarction state is concerned.

With this in mind, the author implied that it had been his clinical experience that when treated and diagnosed in the above manner, that the percentage of individuals who would normally be expected to proceed into acute myocardial infarctions are significantly reduced in numbers. It is pointed out however, that he agrees with those who postulate that once "coronary occlusion is in the making, that nothing will alter its course". The criteria or definition of "occlusion in the making" is not discussed. Once again this might be accounted for on the basis of the difference in the varying concepts of terminology etc..

In summary, the paper has covered a brief discussion of the historical background of heart-coronary disease and myocardial insufficiency. This was accomplished after a suitable definition was derived from the existing concepts of myocardial ischemia as they pertained to various medical fields. Combining the pathologic and physiologic theories, it was decided that the term myocardium ischemia should pertain to a portion of myocardium undergoing anaerobic metabolism subsequent to an insufficient blood supply. Production
and persistence of abnormally large quantities of certain metabolites were considered to be the most likely source of stimulus for the production of angina pectoris. A resumé of Grant's concepts regarding the electrocardiographical diagnosis of myocardial ischemia followed. It was concluded that ischemic EKG readings were characterized by T and ST changes which were without accompanying QRS alterations. The specific pathophysiology of the abnormally widened QRS-T vector angle and the "injury vector" were outlined and summarized diagramatically.

Since adequate discussion of a complete differential diagnosis was not within the confines of the paper, basic pitfalls and diagnostic points in general were considered. The process of ruling out the presence of conduction defects i.e. left bundle branch block, and digitalis effect, as two of the more common entities, was covered succinctly.

It was during this explanation, that the usefulness and necessity of Wilson's ventricular gradient was demonstrated diagramatically in the separation of possible T wave change etiology.

A somewhat greater proportion of the paper was devoted to a relatively detailed discussion and ex-
plaination of Master's exercise test. The expanse of this discussion may be attributed not only to basic, practical interest, but also to the test's importance.

A short discussion of angina pectoris followed and its association with myocardial ischemia was covered quite generally and concisely. An attempt was made to relate both the symptom and the EKG finding to one another. This relationship was further discussed and probably more aptly demonstrated in the insuing involvement with the "pre-infarction syndrome". On covering angina and the impending infarct state, an attempt was made not only to summarize the important clinical points, but also to establish a more perceivable correlation between coronary disease and EKG ischemia.

Let me conclude by stating that although EKG myocardial ischemia remains as a somewhat vague entity with many clinicians today, I feel that its ascendancy from obscurity to relative clinical prominence with in the last decade, is indicative of its future importance. As additional progress is achieved through the years in the fields of electronics, medical therapeutics, and medical research, I am able to perceive the day when routine evaluations of ischemic
heart pattern status, will act as a specific index for the accurate prediction of coronary obstruction, and the dictation for medical or surgical treatment.

Regardless of its future significance, the position occupied by myocardial ischemia as it pertains to pathology, prevention, and production of acute coronary occlusion is an essential one. I can only hope that this paper was in some way able to illuminate this undeniable importance that the subject holds for one of mankind's greatest killers.
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