Electrocardiographic Ischemic Patterns
Without Coronary Artery Disease*

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ST segment deviation is commonly accepted as a sign of myocardial ischemia. Whether indicative of actual myocardial ischemia or not, ST segment changes have been and continue to be generally interpreted as “ischemic ST deviation,” “subendocardial injury,” “coronary insufficiency,” or even “myocardial infarction.” The use of any of these terms intimates coronary artery disease and tends to leave one with an ominous feeling about the patient’s prognosis. Such ominous prognostic feelings on the part of the clinician may lead him to recommend drastic changes in the patient’s way of living. Such recommendations either directly or indirectly indicate to the patient that from that time forward his life is in jeopardy, and he must be extremely careful. Whether or not such drastic steps are necessary following all cases of actual proved coronary artery disease is not a point to be considered here, but it is obvious that such total-life modifying recommendations are contraindicated when there is no coronary artery disease. Iatrogenic heart disease is disabling to a patient, and is unnecessary if the clinician clearly understands when electrocardiographic changes are significant and when they are not.

“Ischemic patterns” refers to those ST segment changes (elevation and depression) which have been commonly considered to be the result of myocardial ischemia. These same electrocardiographic changes are encountered in a wide variety of clinical conditions without other evidence of coronary artery disease. “Non-ischemic” electrocardiographic changes have received sporadic attention, but little effort has been made to establish a common denominator to explain these ST changes. This paper represents an attempt in this direction.

Definitive understanding of the nature of ST deviation has been hampered, both clinically and experimentally by the relative lack of concern as to whether the direction of the shift was upward or downward. It has been generally assumed that epicardial ST segment depression is reciprocal to ST segment elevation on the subendocardial layers of the wall of the heart; elevation or depression in a given lead being dependent on the point of observation, i.e., on the position of the electrode with regard to the area of myocardial injury.

Recently published experiments' demonstrate that ST segment depression is a manifestation of primary epicardial change, as is ST segment elevation. Primary ST segment depression over the epicardial surface of the heart has been experimentally produced without ischemia by per-
fusion of the coronary artery with specific concentrations of electrolytes sufficient to alter the extracellular concentration.

In these perfusion experiments, no myocardial ischemia was produced, yet both ST segment elevation and ST segment depression were recorded from the same perfused area, the direction of the deviation depending on the electrolyte composition of the perfusate. Perfusion of coronary arteries with solutions of high potassium (4 m.Eq./L.K+) or low sodium (103 m.Eq./L.Na+) concentration resulted in ST segment elevation. Perfusion with solutions of high sodium (171 m.Eq./L.Na+) or low potassium (0.5 m.Eq./L.K+) concentration led to ST segment depression. (Figures 1, 2, 3, 4). These ST changes, whether produced by changes in sodium or potassium concentrations, tended to disappear shortly after termination of perfusion.

It is a well established concept that the shape of the electrocardiographic curve appears to be related to the pattern of electrolyte distribution on both sides of the cell membrane, and that ST segment deviation reflects an alteration in these electrolyte concentrations or relationships.

Normally the ratio of intracellular to extracellular potassium is about 30:1 while sodium has an extracellular to intracellular ratio of about 10:1. It is this transmembrane ratio or gradient which has been temporarily altered in the previously mentioned perfusion experiments.

Either an extracellular increase in sodium or an extracellular decrease in potassium leads to an increased transmembrane gradient. The increased transmembrane gradient of either of these ions is reflected in the electrocardiogram as ST segment depression. If the transmembrane gradient of either sodium or potassium is decreased, ST segment elevation will be seen.

Clinically, ST segment is usually associated with lowering of the T wave while ST elevation is usually associated with elevation of the T wave.

Clinically, alterations in the electrolyte pattern, similar to those produced in perfusion experiments have been found in two different types

![Figure 1: ST segment response to perfusion of high concentration potassium solution (4 m.Eq./L.K+) in saline solution (142 m.Eq./L.Na+) into a coronary artery. (A) Control record shows an isoelectric ST segment. (B) ST segment elevation occurs with perfusion.](http://journals.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21352/ on 06/26/2017)
of myocardial ischemia: ischemia with ST segment depression and ischemia with ST segment elevation. Just as in the perfusion experiments, correlation of chemical determinations with electrocardiographic changes showed that ischemia with ST segment depression is related to an increase in the transmembrane gradient of sodium and potassium ions. In ischemia with ST elevation, there is a decrease in the transmembrane gradients, with the ischemic cell losing potassium to the extracellular compartment while the intracellular content of sodium probably increases.

The described perfusion experiments are of more than theoretical interest. ST segment deviations due to electrolyte changes have been reported in many non-ischemic clinical conditions. It is important to note that non-ischemic disease entities which are dissimilar in etiology and clinical picture often show similar changes in electrical potential, leading to identical ST segment deviations. The production of electro-
cardiographic changes by alterations in electrolyte balance in clinical conditions has up to now been limited largely to the obvious cases of potassium balance. However, in the perfusion experiments it has been shown that changes in sodium concentration account for ST segment deviation also. The concept of primary ST segment depression enables us to correlate the direction of ST segment shift with specific electrolyte patterns. In animal experiments and clinical conditions the direction of the ST segment response is usually the same whether on an ischemic or obviously non-ischemic basis.

It should be noted that in the perfusion experiments, the injection of solutions containing various ionic concentrations altered the extracellular fluid concentration of the heart only in that area supplied by the coronary artery being perfused. The ST segment deviations were observed a few seconds following the injections. Many different clinical conditions also reveal variations in serum ionic concentrations. These variations, of course, affect the ionic concentration of extracellular fluid of the entire heart. They may also affect the ionic concentration of the intracellular fluid. Also, the alterations of serum ionic concentration seen in these clinical conditions do not occur immediately, as they do in the perfusion experiments. The duration of the imbalance is infinitely greater in the clinical conditions than in the experiments.

There are many differences that make it difficult to compare acute experiments in animals with chronic or subacute disease states in man. However, similar electrocardiographic changes and alterations in extracellular fluid ionic concentrations are seen both clinically and experimentally. Such similar findings lead us to speculate regarding the correlations between experimental evidence and the clinical conditions described in this paper.

The speculations in this paper can only be considered preliminary in view of the insufficient data now available. It will be many more years, even decades, before all of the factors that affect ST segment deviation are known and understood. Simple acute experiments, such as these cited in this paper, are valuable, since they do help explain the "abnormal" electrocardiogram. Perhaps the most important point of this entire paper is the evidence that is presented showing that an "ischemic" electrocardiogram does not necessarily mean an ischemic heart.

FIGURE 4: ST segment response to perfusion of hypotonic saline solution (103 m.Eq./L.Na+ with 2 m.Eq./L.K+) into a coronary artery. (A) Control record shows an isoelectric ST segment. (B) ST segment elevation occurs with perfusion.
In the remainder of this paper some of the non-ischemic clinical conditions showing ST segment deviation will be reviewed and discussed. For purposes of presentation these clinical conditions have been separated into four general groups, divided according to their commonly accepted etiology:

1. electrolyte imbalance
2. hormonal influence
3. hemoglobin deficit or blockage
4. enzyme system blockage

**Group 1: Electrolyte Imbalance**

The mechanism responsible for electrocardiographic alterations evoked by abnormal electrolyte distributions is already understood to a certain degree. A decrease in the transmembrane gradient of cations is probably responsible for low membrane potential of the cell and for the elevation of the ST segment. An increase in the transmembrane gradient of cations, on the other hand, is related to an increase in membrane potential, the latter showing ST segment depression.

The ST segment responses to various experimentally induced electrolyte imbalances are identical in pattern to those encountered in a number of clinical conditions which are known to involve the same electrolyte imbalances. Under clinical conditions, however, many other factors are present, making the picture more complex. Nevertheless, it has been stated that the electrocardiogram is more sensitive than any chemical method in reflecting the seriousness of an electrolyte imbalance. It would seem that this is not always true.

**Electrolyte imbalance associated with ST segment depression.**

*Diabetic coma and hyperglycemia:* In diabetic coma, important electrolyte changes are seen both before and during specific treatment. Before treatment is initiated, the potassium stores of the body are markedly depleted but this is masked by dehydration and the true deficit is reflected in the electrocardiogram. During treatment with insulin and sodium salt infusions, the serum sodium level rises and the previously masked depletion of potassium stores becomes manifest through rehydration. This deficit is reflected in a low serum potassium level. Serum potassium is further depressed as potassium accompanies glucose into the cell under the influence of insulin. Both the high sodium and low potassium concentrations in the extracellular fluid contribute to depression of the ST segment. With the infusion of appropriate amounts of potassium salts the ST segment depression will disappear.

Similarly, ST segment depression occurs after injection of hypertonic glucose or the ingestion of huge quantities of glucose. The endogenously released insulin will promote the movement of glucose and potassium into the cells, and the decreased extracellular potassium will be reflected electrocardiographically as ST segment depression.

*Familial periodic paralysis:* During attacks of familial periodic paralysis extracellular potassium probably migrates into the cells, resulting in a higher transmembrane gradient of this ion. ST segment depression is often observed during these attacks.
Artificial kidney: The changing electrocardiographic picture during extracorporeal hemodialysis\textsuperscript{7,8,9} from ST segment elevation to an isoelectric line, or even ST segment depression, follows closely the changing serum level of potassium. The low serum potassium level after hemodialysis usually shows ST segment depression along with other electrocardiographic changes.

Gastrointestinal conditions: ST segment depression has been observed in the following conditions: therapeutic peritoneal lavage, intestinal lavage, prolonged diarrhea,\textsuperscript{10} in the course of severe malnutrition,\textsuperscript{11,12} dysentery, intestinal obstruction,\textsuperscript{13} bile fistula, and prolonged vomiting. All of these conditions are characterized by potassium loss and a low extracellular concentration of potassium. In these gastrointestinal states ST segment depression may occur without any indication of myocardial ischemia.

Salicylate toxicity: A toxic dose of salicylates directly stimulates the respiratory center, resulting in hyperventilation, loss of carbon dioxide and respiratory alkalosis. In salicylate toxicity the stage of respiratory alkalosis is associated with low serum potassium and occasionally visible ST segment depression. Marked ST depression is sometimes found.\textsuperscript{14,15} Treatment with potassium salts has been found to return the depressed ST segment to the isoelectric line.\textsuperscript{16}

There is evidence for the assumption that alkalosis potentiates electrocardiographic changes induced by hypokalemia, while acidosis obscures the hypokalemic pattern.\textsuperscript{17,18}

Anxiety: ST segment depression has been encountered in acute anxiety reactions and is commonly ascribed to respiratory alkalosis secondary to hyperventilation.\textsuperscript{19,20} In clinical practice, false positive exercise tests may be brought on by incidental anxiety in the emotionally labile patient.

Diuretic therapy: Cohen\textsuperscript{21} recently described a case of muscle paralysis due to hypokalemia which had developed during the course of chlorothiazide administration. In this case ST segment depression was recorded while the serum potassium level was as low as 1.65 m.Eq./L. The serum sodium level was elevated. Hypokalemia is noted in about 40 per cent of patients during chlorothiazide treatment. ST segment depression has also been observed during treatment with mercurial diuretics.\textsuperscript{22,23}

Low sodium diet: A very restricted low sodium diet used in the treatment of arterial hypertension has occasionally led to disappearance of the ST segment depression connected with left heart strain. Simultaneously, a substantial decrease in serum sodium and a corresponding increase in serum potassium was reported.\textsuperscript{24} This observation raised the question as to whether the electrolyte changes secondary to severe sodium restriction were alone responsible for the electrocardiographic phenomena.\textsuperscript{25} Using potassium in the treatment of patients with left heart strain gradually returned the electrocardiographic changes characteristic of hypertensive disease to normal. Since norepinephrine and mineralocorticoids show a specific ability to promote a shift of sodium into the cell, such electrolyte changes may be involved in the production of arterial hypertension.
**Electrolyte imbalance associated with ST segment elevation.**

**Shock:** The complex and changing mechanisms of profound traumatic shock may result in either elevation or depression of the ST segment. Usually, extracellular potassium is elevated while extracellular sodium is low.4' ST segment elevation was found in electrocardiograms taken following war injuries, and in acute pulmonary embolism with shock.4 Experimental ischemic compression of an extremity has led to ST segment elevation,4 the elevation becoming more pronounced after massage of the ischemic areas. This phenomenon was explained on the basis of potassium being released from the ischemic tissues. ST segment depression, in contrast, was observed in post-operative patients who were maintained on intravenous feeding; this ST segment depression was corrected by the administration of potassium chloride.4

**Miscellaneous conditions:** Elevation of the ST segment may be encountered in clinical entities characterized by increased levels of extracellular potassium; as in some types of acidosis, following ingestion of excessive potassium salts,4 and in uremia. In this last condition elevation of the ST segment may mask the electrocardiographic picture of “left heart strain.”4'4' High levels of serum potassium may also be observed when there is an excessive release of cellular potassium, as in hemolytic transfusion reactions,4' hemolytic anemias, crush syndrome,8' severe attacks of malaria,8' starvation, and various other conditions associated with cell destruction. Wener et al.8' encountered hyperkalemia and ST elevation after injection of hemolyzed red blood cells during animal experiments.

**Group 2. Hormonal Influence**

It is suggested that ST segment deviation induced by some hormonal factors can be explained on the basis of changes in electrolyte distribution. In different hormonal syndromes the ST segment may deviate up or down, depending on the serum concentrations of sodium and potassium. Some observers have succeeded in correcting such ST segment deviations by administration of appropriate electrolytes.

**Adrenal cortical hypofunction:** In severe acute adrenal insufficiency, ST segment elevation is encountered, usually combined with a high, peaked T wave; these changes disappearing after the serum potassium is returned to normal levels. In adrenal insufficiency urinary excretion of sodium is increased while excretion of potassium is decreased.19 There is an associated loss of water with resultant depletion of extracellular fluid, reduction of plasma volume, and dehydration.4' Fluid loss is further aggravated by migration of water into cells. Robertson et al.17 found a concomitant decrease of intracellular sodium in adrenal insufficiency.

**Adrenal cortical hyperfunction:** In contrast to the ST segment response observed in adrenal insufficiency, administration of large doses of ACTH or adrenal cortical steroids over a long period of time may result in ST segment depression.4'4'7 Such ST segment deviation was returned to the isoelectric line following administration of potassium salts.4 ST segment depression induced by treatment with desoxycorticosterone acetate was found to be potentiated by 1 per cent sodium chloride,4' but after administration of potassium chloride the changes were quickly reversed and the ST segment returned to the isoelectric level.4'
Although the alterations in potassium are largely secondary to those of sodium metabolism, it is believed that the adrenal cortex exercises some specific effect on potassium metabolism. Cortisone in doses above 200 mg. per day increases urinary excretion of potassium, lowers the serum potassium, and results in salt retention. These changes lead occasionally to hypopotassemic hypochloremic alkalosis and signs of hypokalemic "toxicity." The same direction of electrolyte shift is induced by continued administration of large doses of corticosterone, desoxycorticosterone, and ACTH.9

Attention has been drawn recently to the effect of aldosterone, a hormone which has a sodium retention power 500 times that of hydrocortisone. ST segment depression is considered to be a diagnostic feature of aldosteronism and has been traced to hypopotassemia. Examples of secondary aldosteronism can be encountered in nephrosis, congestive heart failure, hepatic cirrhosis, eclampsia, and idiopathic edema. In tracings from patients with hepatic cirrhosis and hepatic coma ST segment depression has been observed.4

ST segment depression has been observed in some cases of Cushing's disease. Increased formation of adrenal steroids with salt retaining effects leads frequently to reduction of serum potassium and elevation of serum sodium. In a case of Cushing's disease Teabeaut restored the ST segment to the isoelectric level by potassium infusion.42

Epinephrine: Infusion of large doses of epinephrine has been reported to produce ST segment depression under experimental conditions.43 This response may be explained on the basis of electrolyte changes in cardiac muscle. With infusion of a large amount of epinephrine, Robertson et al.44 observed an initial rise in serum potassium followed within a few minutes by a steady decrease; at the same time there was an increase in serum sodium and glucose.

The ST segment depression encountered in anxiety states has been attributed by some authors to the action of released epinephrine.45 An increase in the depth of ST segment depression was demonstrated in some emotionally labile patients after the injection of small doses of epinephrine. In normal subjects, small doses of epinephrine have no effect on the ST segment.46

Toxemia of pregnancy: As previously noted, epinephrine infusion may produce ST segment depression.46 In normal pregnancy, epinephrine and other vasoconstrictor amines are inactivated by monoamino oxidase present in the placenta and the decidua. The activity of this enzyme, however, may be dependent on the oxygen tension within the placenta. In toxemia of pregnancy, placental ischemia has been demonstrated and these ischemic changes are believed to be responsible for a substantial lowering of oxygen tension and a significant decrease in the activity of monoamino oxidase.

This factor may at least partially explain the ST segment depression sometimes observed in toxemia of pregnancy.

Sex hormones: While there is some clinical evidence that the gonadal hormones affect the electrolyte balance of body fluids, this area is in need of more extensive investigation. ST segment depression has been reported
in menopause and was found to disappear with estrogen treatment\(^*\) although the published changes do not appear to be great.

**Insulin:** Under the influence of insulin, both glucose and potassium leave the extracellular fluid and enter the cell. As the transmembrane gradient of potassium increases, ST segment depression appears. This electrocardiographic finding has been long observed in insulin shock in diabetes and in insulin coma treatment for psychiatric disorders.\(^*\) In insulin-induced hypoglycemia Parrish\(^*\) and Judson et al.\(^*\) have correlated ST segment depression with the changes in serum glucose and potassium.

**Group 3: Hemoglobin Deficit or Blockage**

ST segment depression is a frequent finding in a group of clinical entities characterized by a deficit in active hemoglobin. The deficit may be due either to a decrease in total body hemoglobin or to a blockage of its physiological activity. In both instances, there is an identical result, i.e., an insufficient supply of oxygen to the cell.

**Hemoglobin deficit**

Hypoxia impairs aerobic metabolism of glucose and the cell compensates by increasing its anaerobic glucose turnover. This less economical latter process requires considerably more glucose, and greater quantities of glucose and potassium enter the cell.\(^*\) There is a concomitant compensatory shift of sodium to the extracellular fluid. The resultant changes in myocardial cell transmembrane gradients of sodium and potassium seem to be manifest in the electrocardiogram as ST segment depression.\(^*\)

**Anemia:** Most cases of mild anemia present no electrocardiographic abnormalities because the hemoglobin deficit is compensated for by an increased rate of blood flow. In severe anemia, regardless of type, the state of the myocardial cell is affected and ST segment depression is often observed.\(^*\) The same electrocardiographic finding has been reported after acute hemorrhage.\(^*\)

In severe degrees of anemia, instances of anginal pain without overt coronary disease have been reported.\(^*\) Relief of the anemia resulted in the simultaneous disappearance of both angina and the electrocardiographic abnormalities. It is important to note that anemia produced anginal pain and ST segment depression identical to that caused by ischemia. The common cause for the changes in both conditions is probably a similar disturbance of cell metabolism. It would seem that the manifestations of ischemia should not be regarded as specific.

**Hemoglobin blockage**

Blockage of hemoglobin activity, or the presence of abnormal hemoglobins, may change the carbohydrate metabolism of the cell in the same way as in absolute hemoglobin deficit.

Such functional deficits of hemoglobin occur in the presence of carboxyhemoglobin, methemoglobin, and sulfhemoglobin. There are numerous reports\(^*\) of ST segment depression in the presence of these abnormal hemoglobins and the electrocardiographic changes can be uniformly explained by hemoglobin blockage. Occasional ST segment elevation is
observed with carbon monoxide poisoning and this may be due to an extreme degree of hemoglobin blockage. The reversal of direction in the ST segment response resembles the difference observed in varying degrees of ischemia, i.e., ST segment depression with mild ischemia and ST segment elevation with severe ischemia.

An insufficient oxygen supply to the alveolar surfaces of the lungs will decrease the quantity of available oxyhemoglobin and increase the amount of circulating reduced hemoglobin. This occurs in chronic pulmonary diseases, in some normal subjects during anoxic tests, and in the presence of low atmospheric pressures at high altitudes. ST segment depression in these conditions has been extensively documented and reported. In the final stage of anoxia, ST segment elevation may occur.

Insufficient ventilation during anesthesia results in an accumulation of carbon dioxide which combines with hemoglobin, thus reducing the amount of hemoglobin available for oxygen transport. This strong affinity of hemoglobin for carbon dioxide is more apparent in carbon dioxide poisoning which may lead to ST segment depression or to ST segment elevation in severe cases. Altschule and Sulzbach showed that inhalation of 5 per cent carbon dioxide and 95 per cent oxygen results in marked ST segment shifts from standard leads. These shifts were rapidly reversible upon stopping the inhalation. Their important conclusion was that such ST segment changes occur in the absence of anoxia or ischemia.

Group 4: Enzyme System Blockage

The correlation of electrocardiographic changes with alterations in complex enzymatic reactions, with present knowledge, is largely based on reasoning by inference. However, a certain amount of direct confirmation of such changes is available.

Among the common poisons of enzyme systems, heavy metals are accountable for ST segment depression. Definite changes in the ST segment have been encountered following intravenous injection of mercurial diuretics, a few hours after injection of neoarsphenamine, and following treatment with antimony. Heavy metals act like sodium, inhibiting enzyme systems in the Krebs cycle (aconitase, isocitric enzyme) and in fatty acid degradation.

The experimental injection of mono-iodo-acetic acid, which inhibits glucose metabolism by blocking trioxophosphodehydrase, was found to induce ST segment depression.

In the above examples, enzyme system blockage limits aerobic metabolism and ST segment depression may result. When the suppression of enzymatic reactions is more pronounced, ST segment elevation may occur. Cyanides, for example, suppress all oxidative tissue pressues and when given in large doses, ST segment elevation can be expected. The same result has been observed in some cases of veratrine intoxication. On the other hand, in patients with angina pectoris, the injection of cytochrome C, which acts as a catalyst for oxidation, prevents the ST segment changes customarily found when these patients are subjected to exercise or low oxygen pressures.

The problem of oxidation also enters into the probable explanation of ST segment deviation occurring in various vitamin deficiencies. It is
known, for example, that deficiencies of the Vitamin B group inhibit dehydrogenase and decarboxylase, thus affecting the aerobic breakdown of carbohydrates. Experimentally induced thiamine deficiency in dogs produced a depletion of myocardial glycogen stores and ST segment elevation; this was reversed after thiamine was added to the diet. In clinical reports ST segment depression has been occasionally ascribed to avitaminosis B1.

Depression of the ST segment in some cases of pellagra was corrected by treatment with niacin. In some cases of myocarditis, angina pectoris, and atypical hypothyroidism, the depressed ST segment was corrected by administration of niacin. Also experimentally, a diet free of pantothenic acid has produced ST segment depression.

Discussion

In this report, mainly changes in extracellular concentrations, or transmembrane gradients, of potassium and sodium ions have been taken into consideration. It is suggested that in many different clinical conditions variations in serum cationic/cationic gradients play an important part in producing ST segment depression or elevation. However, other ions, or as yet unknown factors may also be involved. There are also some exceptions which do not fit the concept of sodium and potassium electrolyte gradients presented in this paper.

Shifts of potassium and sodium ions produced ST segment changes in cases of myocardial ischemia identical to those found in the experimental and clinical conditions cited in this paper. Potassium shifting into the cell, while sodium moves into the extracellular compartment, results in ST segment depression, as observed in the classic form of angina; potassium leaving the cell, while sodium enters, leads to ST segment elevation, as observed in the variant form of angina or early myocardial infarction.

Electrocardiographic changes in ischemia can probably be explained on the basis of specific alterations in the metabolism of the ischemic cell, which in turn affect intracellular and extracellular electrolyte distribution.

SUMMARY

ST segment deviations (elevation and depression) can be indicative of myocardial ischemia or injury secondary to coronary artery disease. In a wide variety of clinical conditions, however, these ST changes are “non-ischemic,” reversible by appropriate therapy and should not be interpreted as pathognomonic of coronary artery disease. Clinical conditions showing these ST changes without myocardial ischemia are reviewed in this paper.

It is postulated that ST segment deviation, whether or not the result of myocardial ischemia is related to changes in potassium and sodium gradients across the myocardial cell membrane. It is further postulated that an increased transmembrane gradient of either of these ions produces ST segment depression and that a decreased transmembrane gradient produces ST segment elevation. Clinical and experimental evidence is presented supporting these postulates. However, there is little doubt that factors other than sodium and potassium are involved.

RESUMEN

Las desviaciones del segmento ST (elevación y depresión) pueden ser indicadoras de isquemia del miocardio o lesión secundaria a la enfermedad coronaria.

En una gran variedad de estados clínicos sin embargo estos cambios de ST son “no isquémicos,” reversibles por la terapia adecuada y no deben interpretarse como patognomónicos de enfermedad coronaria. Las condiciones clínicas que muestran estos cambios de ST sin isquemia del miocardio se pasan en revista en este trabajo.

Se supone que la desviación del segmento ST, sea o no resultado de la isquemia miocárdica, está en relación con los cambios del potasio y del sodio en sus gradientes a través de la membrana celular del miocardio. Se supone además que un gradiente creciente a través de la membrana, de cualquiera de estos iones produce una depresión del segmento ST y que el decrecimiento del gradiente a través de la membrana produce una elevación del Segmento ST. Se presentan evidencias clínicas y experimentales que sustentan esta interpretación. Sin embargo, no hay duda de que existen otros factores además del sodio y del potasio.

RESUMÉ

Les déviations du segment ST (élévation et dépression) peuvent indiquer une ischémie myocardique ou une altération secondaire à une affection de l’artère coronaire. Dans une grande diversité de conditions cliniques, cependant, ces modifications
zu segment ST sind "non-ischémique," réversibles par une thérapeutique appropriée, et devraient être interprétées comme caractéristiques de l'atteinte de l'artère coronaire. L'auteur passe en revue les conditions cliniques montrant ces modifications du segment ST sans ischémie myocardique.

Il admet le postulat que la déviation du segment ST, qu'elle soit ou non le résultat d'une ischémie myocardique, est en rapport avec les modifications des gradients de potassium et de sodium à travers la cellule membraneuse myocardique. Il part ensuite du fait qu'une augmentation du gradient à travers la membrane de l'un de ces ions produit une dépression du segment ST et que la diminution du gradient produit une élévation du segment ST.

L'auteur apporte une preuve clinique et expérimentale à l'appui de ces postulats. Cependant, il y a une certaine possibilité pour que d'autres facteurs que le sodium et le potassium entrent en ligne de compte.

ZUSAMMENFASSUNG


Es wird die Forderung aufgestellt, daß eine Abweichung der ST-Strecke, sei sie nun die Folge einer Ischämie des Myocards oder nicht, in Beziehung gesetzt wird zu Veränderungen im Calcium- und Natrium-Druckgefälle durch die Herzmuskel-Zell-Membran. Es wird weiter als gegeben vorausgesetzt, daß ein erhöhtes transmembranes Druckgefälle eines dieser Ionen eine Senkung des ST-Stückes bewirkt, und daß ein herabgesetztes transmembranes Druckgefälle zu einer Hebung des ST-Stückes führt. Es werden klinische und experimentelle Zeugnisse zur Unterstützung dieser Postulate angeführt. Wenig Zweifel besteht jedoch darüber, daß noch andere Faktoren außer dem Natrium und Calcium im Spiele sind.

Complete reference list will appear in the reprints.

FDA RULING ON CHLORAMPHENICOL

Commissioner of Food and Drugs George P. Larrick has announced (January, 1961) that a panel of distinguished physicians appointed by the National Research Council at the FDA's request has found that the antibiotic, Chloromycetin (chloramphenicol) is a valuable drug that should remain on the market for use in treating serious infections under medical supervision both in hospitals and for treatment of patients in the home. According to the ruling of the FDA, the following is to be clearly stated:

(Immediate container label)

"WARNING: Blood dyscrasias may be associated with the use of chloramphenicol. It is essential that adequate blood studies be made (see enclosed warnings and precautions)."

(enclosed insert)

"WARNING: Serious and even fatal blood dyscrasias (aplastic anemia, hypoplastic anemia, thrombocytopenia, granulocytopenia) are known to occur after the administration of chloramphenicol. Blood dyscrasias have occurred after short-term and with prolonged therapy with this drug. Bearing in mind the possibility that such reactions may occur, chloramphenicol should be used only for serious infections caused by organisms which are susceptible to its antibacterial effects. Chloramphenicol should not be used when other less potentially dangerous agents will be effective or in treatment of trivial infections such as colds, influenza, viral infections of the throat, or as a prophylactic agent."

"Precautions: It is essential that adequate blood studies be made during treatment with the drug. While blood studies may detect early peripheral blood changes, such as leukopenia or granulocytopenia, before they become irreversible, such studies cannot be relied upon to detect bone marrow depression prior to development of aplastic anemia."