

Prinzmetal's work and the "Sclarovsky-Birnbaum ischemia score" for acute myocardial infarction: a parallel in systematizing electrocardiographic knowledge

John E. Madias, MD, FACC, FAHA*

Mount Sinai School of Medicine of the New York University, New York, NY, USA

Division of Cardiology, Elmhurst Hospital Center, Elmhurst, NY, USA

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Abstract

In this viewpoint, the author traces back in time the original (? if possible) description of the electrocardiographic hallmarks of myocardial ischemic injury leading to myocardial infarction. In this venture, he detects a parallel between the work of Prinzmetal et al and that of Sclarovsky and Birnbaum and al, in their approach to research, which is based on systemization and utilization of electrocardiographic knowledge.

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Οὐδέν καινόν ὑπὸ τὸν ἥλιον

(There is nothing new under the sun).

Ecclesiastes

Truth springs from argument amongst friends

David Hume

Introduction

The objective of this viewpoint is to trace back in time the original (? if possible) description of electrocardiographic (ECG) attributes of acute ischemic injury, which if persistent are followed invariably by sequentially changing ECG attributes of acute myocardial infarction (AMI). Although the ST-segment elevation (+ST) and peaking of the T wave, which were described in the 19th century, are also dealt with, emphasis is focused on the changes in the QRS complex, that is, increase in the amplitude of the R waves, decrease in the amplitude, or disappearance, of the S-waves, which were noted much later. These last 3 ECG features have been used in the "Sclarovsky-Birnbaum ischemia score" (S-B score). It is not the intention of this communication to delve in the proven or potential merits of S-B score as a predictor of mortality, morbidity, and overall outcome after an AMI, for which the reader is

referred to the voluminous literature on the topic, but to draw attention to the similarity in the research approach of 2 groups, that of Sclarovsky, Birnbaum et al, currently active, and of Prinzmetal et al, who did their work 50 years or more ago. Finally, a few personal views about the S-B score are included, with questions that need to be answered and suggestions that may strengthen the scientific underpinnings of the S-B score and enhance its validity as a clinical index. The author begs the understanding of the readers about the substandard, for the *Journal*, quality of the reproduction of some of the figures, which had to be scanned from journal pages of 50 years old or more.

The "Sclarovsky-Birnbaum ischemia score"

Sclarovsky, Birnbaum et al, in 30+ papers spanning the years from 1988¹ to 2008² (complete list of references available on request), described and implemented the S-B score of myocardial ischemia, dealing with the hospital admission ECG in patients with AMI. Sclarovsky et al initially categorized patients with anterior AMI in those with tall and peaked T waves (first-degree ischemia), those with +ST and the above-noted T-wave changes (second degree ischemia), and those with the ECG features of second-degree ischemia, plus further +ST, *disappearance of the S-wave, and increase in the amplitude of R-wave* (third-degree ischemia).¹ The authors posited that patients with AMI may demonstrate 1 of these 3 ECG patterns of ischemia before the

* Division of Cardiology, Elmhurst Hospital Center, Elmhurst, NY 11373, USA. Tel.: +1 718 334 5005; fax: +1 718 334 5990.

E-mail address: madiasj@nychhc.org

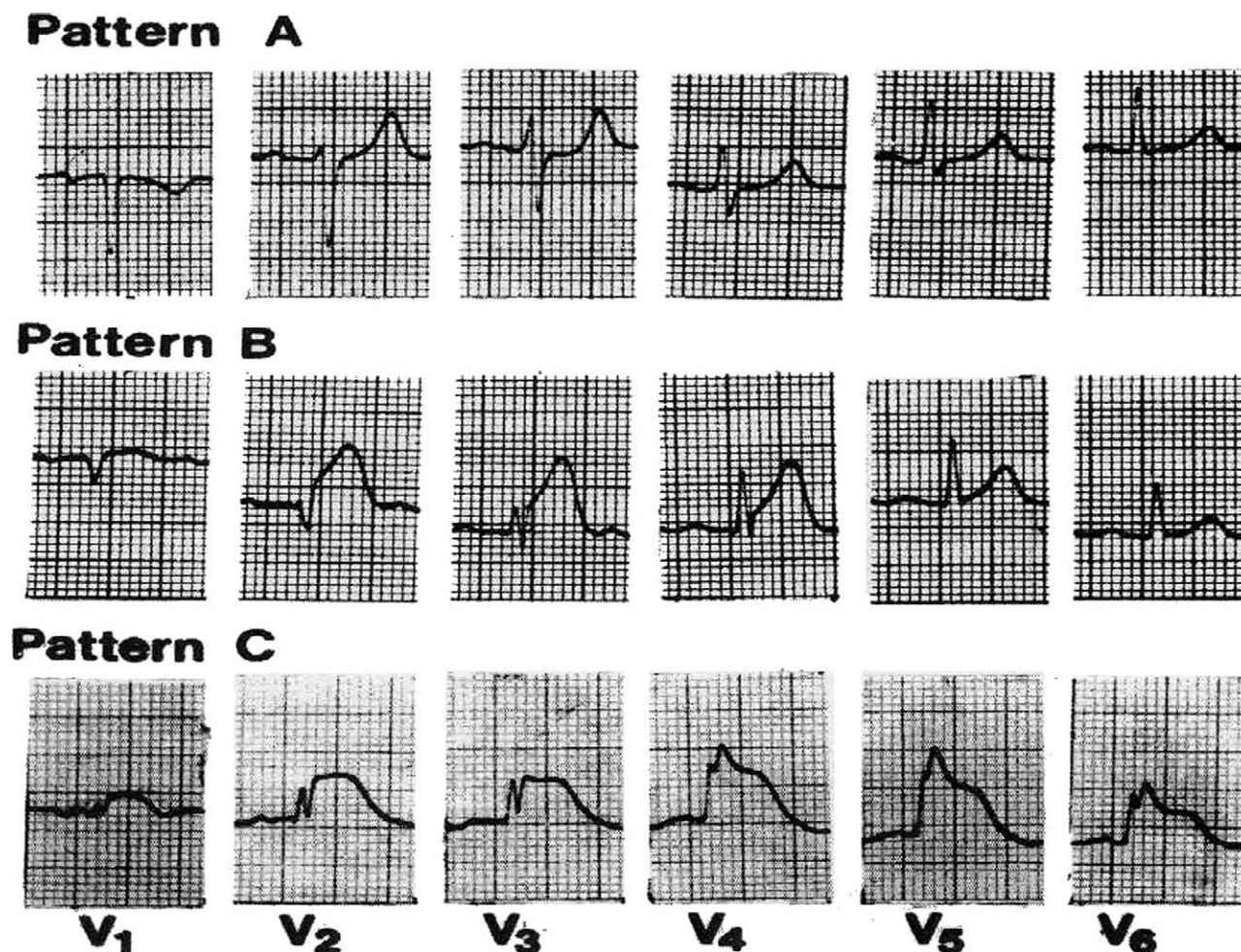


Fig. 1. Electrocardiographic patterns A (no ST-segment elevation with peaked upright T waves), B (ST-segment elevation without major changes in the terminal portion of the QRS complexes), and C (ST-segment elevation with major changes in the terminal portion of the QRS complexes) in 3 different patients with an anterior AMI of the “Sclarovsky-Birnbaum ischemia score,” involving the leads V_1 to V_6 . Note that the changes in the terminal portion of the QRS complexes in pattern C consist of a decrease or disappearance of the S waves; also note that the amplitude of the ST-segment elevation is equal to or less than the amplitude of the corresponding R wave in pattern B, whereas it is more than one half the corresponding R wave in pattern C. Adapted from reference [3], with permission from *Chest*.

emergence of T-wave inversion or Q waves, and that each of them have a typical evolution; no information on Q waves was included in that article.¹ Subsequently, the patients with an anterior AMI were stratified into those with tall and symmetrical T waves (pattern A), abnormal T waves and +ST (pattern B), and abnormal T waves, +ST of proportionally larger amplitude than in pattern B, and *distortion of the terminal portion of the QRS complexes* (pattern C); the distortion of the QRS complexes consisted of disappearance of the S-waves and/or emergence of the J point at a level above one half of the R-wave amplitude (Fig. 1).³ Patients with anterior AMI have been evaluated by these authors before the development of Q waves, using the admission ECG, indicating that the patients were observed early in the course of their AMI; these 3 ECG patterns were stable from admission to the time of the administration of thrombolysis.³ Similar patterns A, B, and C of the S-B score have been described in patients with inferior AMI (Fig. 2).⁴

Other synonyms for “grade 3” ischemia and “pattern C” of S-B score, which the authors used, are ST-segment elevation with terminal QRS distortion, emergence of a

J point at a level above the lower half of the R wave in leads with qR configuration, or disappearance of the S wave in leads with Rs configuration, QRS (+) pattern, and J point-to-R wave amplitude ratio of 50% or more. The authors felt that the patients with distortion of the QRS complexes did not represent a cohort with a shorter time interval between the onset of chest pain and the recording of the ECG.⁵ The distortion of the terminal QRS complexes has been attributed to severe ischemia (in the absence of collaterals, or “preconditioning”), involving the Purkinje system (ordinarily more resistant to ischemia than the contracting myocardium).⁶ The resultant slowing of conduction with late unopposed (or uncanceled) ventricular activation manifests as an increase in the R waves and disappearance of the S waves in the territory of AMI. Of note is that other workers have found in an animal model of acute myocardial ischemia⁷ that the increase in the amplitude of R waves was due to a *decrease in the intramyocardial conduction*, and it was *not associated with a change in the conduction of the specialized conduction pathways*. Clinical experience also has

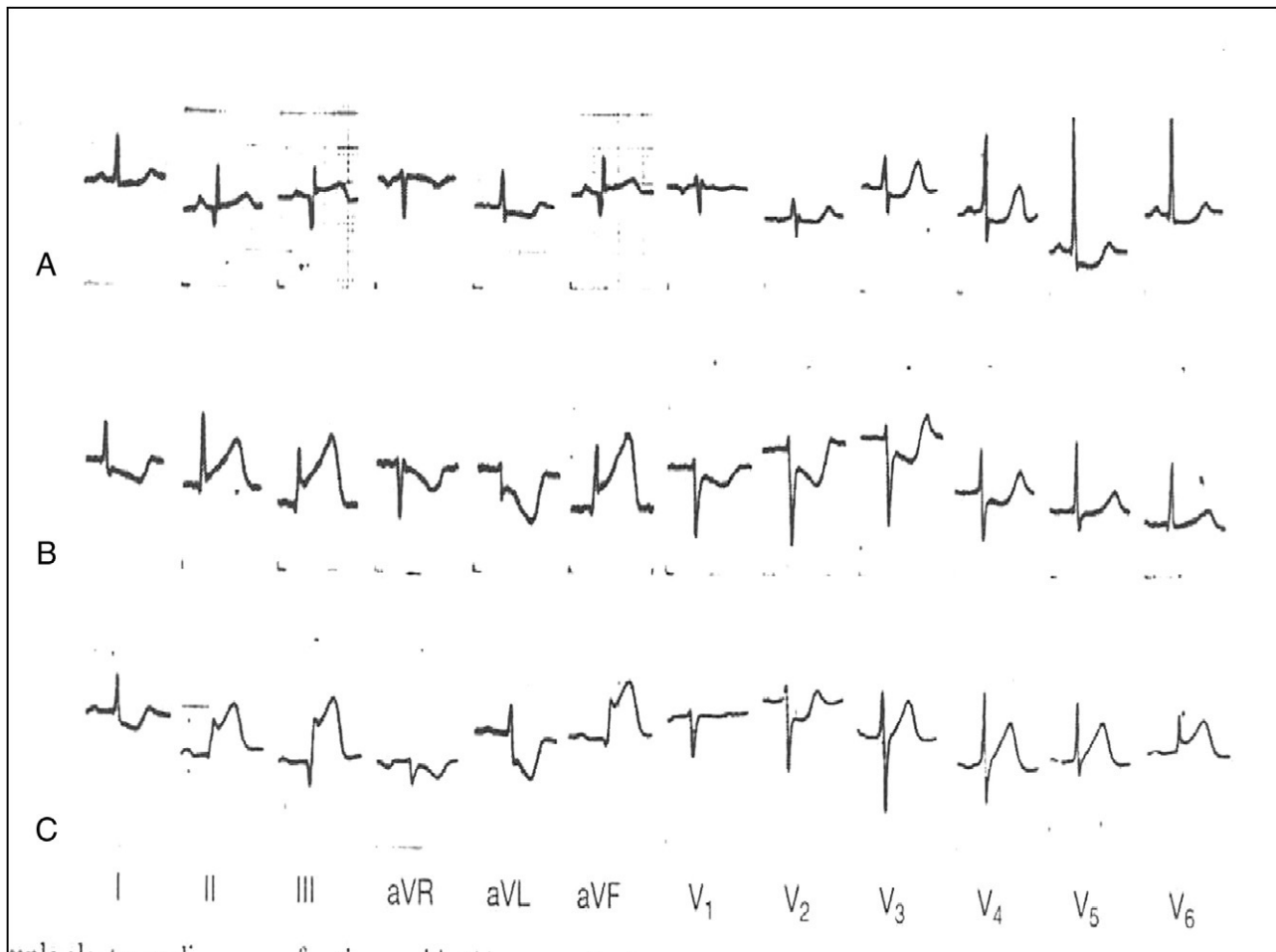


Fig. 2. Electrocardiographic patterns A (ST-segment depression), B (ST-segment elevation without major changes in the terminal portion of the QRS complexes), and C (ST-segment elevation with major changes in the terminal portion of the QRS complexes) in 3 different patients with an inferior AMI of the “Sclarovsky-Birnbaum ischemia score,” impacting leads II, III, and aVF. Note that the changes in the terminal portion of the QRS complexes in pattern C consist of disappearance of the S-waves, whereas there is a decrease in the amplitude of the S wave in pattern B; also note that the amplitude of the ST-segment elevation is equal to or less than one half the amplitude of the corresponding R wave in pattern B, whereas it is more than one half of the corresponding R wave in pattern C. A purist may object to a reference above of a “disappearance of the S-waves,” while there is a decrease in the amplitude of the S-wave” because the ‘S’ component of the QRS complex should not be called as such, if it stops above the isoelectric line; however, these expressions were retained because they were used in the work of Prinzmetal et al. More appropriate would be to consider the complexes with ST-segment elevation as represent qR configurations, not necessarily qRs configurations; accordingly, the grading of ischemia would be based on the height of the ST segment (J point) in relation to the R wave, not to changes in the S waves. Adapted from reference [4], with permission from *Clinical Cardiology*.

attributed the increase in the amplitude of R waves to regional slowing of the ventricular conduction.⁸ Practically, it may not make a difference whether there is slowed conduction in the Purkinje system with normal intraventricular conduction or vice versa because both scenarios would lead to late unopposed ventricular depolarization and enhanced somewhat widened R waves. The S-B score mostly refers to terminal distortion of the QRS complexes, in connection with “pattern C” of ischemia, reflecting behavior of the S waves, rather than an increase of amplitude of the R waves or widening of the R waves, although the latter are clearly shown in the figures of the authors’ articles (Fig. 1).³

Work of Prinzmetal et al on ischemic ECG changes

Prinzmetal et al, in 34 papers (complete list of references available on request), spanning the time interval between

1952 and 1970, dealt with the coronary circulation, classic effort angina and variant (Prinzmetal’s) angina, non-ST-segment elevation myocardial infarction, and +ST AMI. In that body of work, which involved both experimental animal models and patients, they described in detail the ECG changes resulting from partial or complete coronary occlusions in laboratory animals, and angina, AMI, and subsequent evolution in patients. In studying their work, one finds that those workers confirmed and used in further experiments the repolarization (ST-segment depression [–ST], +ST, and T-wave peaking or inversion) and the depolarization (Q waves and reduction of the amplitude of R-waves) changes, which had been noted previously in the laboratory and in patients.

What these workers described, concurrently with –ST or after it, in laboratory animals and patients with angina or mild AMIs, was a decrease (sometimes transient) in the R waves and an increase in the S waves; these changes

were consistent with the early phase of nontransmural myocardial ischemia, imparted by coronary ligation with or without associated hemorrhagic hypotension.^{9–12} In the early phase of transmural ischemic injury in canine experiments, they confirmed previous observations that, concurrently with +ST, the S wave becomes shallower and finally disappears, followed by an increase in the amplitude and a delay in the peak of R wave; no change in the slope of R wave was perceptible but the increased amplitude of the R wave was responsible for the delay in the peak of R wave; finally, the R wave increased in amplitude (occasionally called “giant R wave”) and duration to merge with the ST segment to form a monophasic potential (Figs. 3–5), a pattern occasionally simulating ventricular tachycardia, when the heart rate was rapid.^{10–13} The R-wave increase in the amplitude and width preceded the complete recession of the S wave and the formation of the monophasic potentials.^{10,11} Release of the coronary ligature led to decrease in the +ST and R-wave amplitude and increase in the amplitude of the S wave.^{10,11} These changes also could be exacerbated or ameliorated by hemorrhagic hypotension, or blood transfusions with restoration of the blood pressure, respectively, in the animals subjected to coronary ligation.^{10,11} All the above-described ECG

changes were more pronounced in the central zone of ischemia, as compared to the intermediate and peripheral zones, and in the process a *gradation* of +ST was observed in different locations of the ischemic territory (Fig. 4).^{10,11,13} Of interest is that all these changes in the ST-segment deviation and the amplitude of R waves and S waves could also be reproduced in animal experiments by changing the intracellular/extracellular potassium and sodium gradients, and in the absence of myocardial ischemia, although such changes were less impressive than the ones noted during coronary ligation.¹⁰ The same workers described in the course of severe attacks of variant angina, sometimes leading to AMI, along with the +ST, increase in the R-wave amplitude and width,^{10,14} and recession of the S wave.^{10,14} These changes in the QRS complexes were attributed to alterations in the action potentials, the electrical conductivity of the extracellular medium, and the ventricular propagation process.^{10,11}

A parallel in modus operandi of Prinzmetal et al and Sclarovsky, Birnbaum et al

In studying the work of the above 2 groups, one is struck by a similarity of the manner in which they carried out their

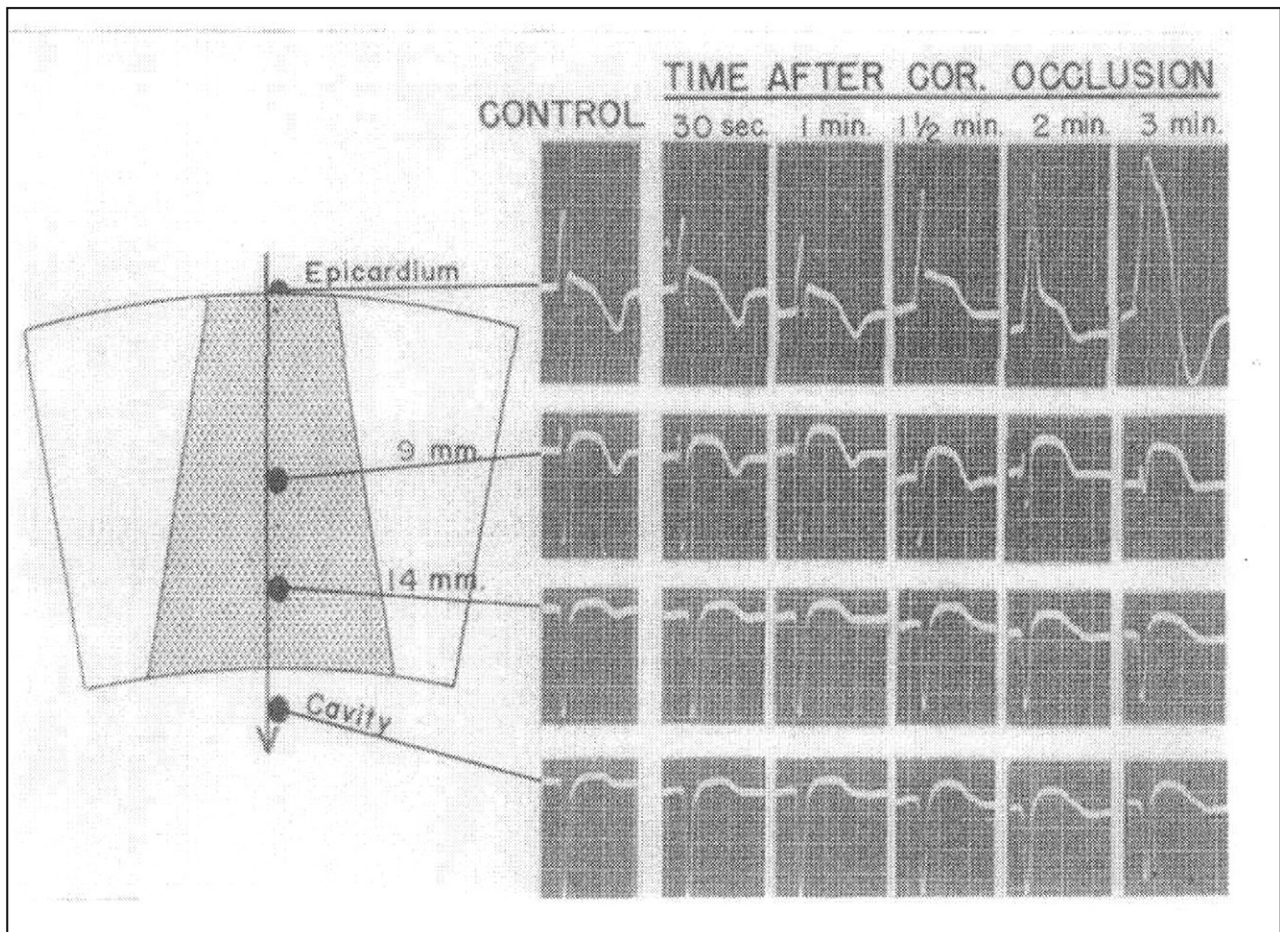


Fig. 3. Myocardial ischemia resulting from coronary ligation led to epicardial increase in progressively increased ST-segment elevation, decrease in the amplitude of S-wave with eventual disappearance, increase in the amplitude and eventually duration of the R-waves, and final merging of the ST-segment and QRS complexes and formation of monophasic curve. Adapted from reference [13], with the permission from the *American Heart Journal*.

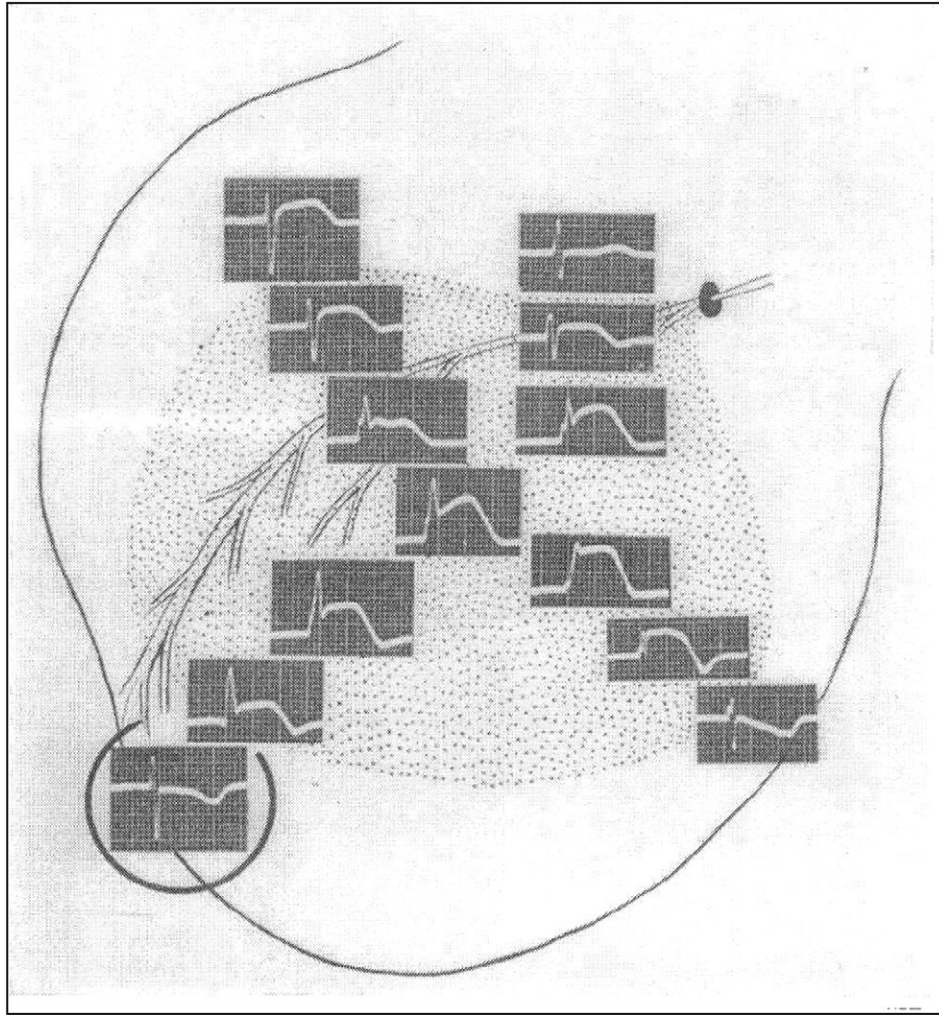


Fig. 4. Epicardial leads from ischemic injured area reveal ST-segment elevation and disappearance of S waves. Note that there is a descending gradation in the amplitude of the ST-segment elevation from the center toward the margins of the injury. The amplitude of the ST-segment elevation is more than one half of the amplitude of the corresponding R waves in the center of the injured area, whereas it is less than one half of the amplitude of the corresponding R wave toward the periphery of the injured territory. Adapted from reference [13], with permission from the *American Heart Journal*.

investigations. Both Prinzmetal et al and Sclarovsky, Birnbaum et al gleaned what was important in the work of their predecessors, put together a working model for their investigations, and proceeded to explore it in new scientific ventures. In reference to Prinzmetal et al, in many of their studies, they explored the attributes and utility of the QRS changes of acute ischemic injury in both the animal laboratory and the clinic to the fullest. Indeed, such work was so focused and intense, and so intimately linked to their group, that this author always attributed to them the original description of the underlying concepts, until one of the reviewers of the original submission of this communication to the *Journal* provided evidence that this was not the case. In fact, virtually all the ECG hallmarks of ischemic injury manifesting in repolarization and depolarization were well known many years before their use by Prinzmetal et al.

Early work on ischemic ECG changes

In an article by Wolferth et al,¹⁵ Burdon-Sanderson and Page were quoted as stating that “Engelmann in 1873 was

the first to demonstrate ‘monophasic’ curves resulting from cardiac injury in the frog”, and Bayliss and Starling are also quoted as stating that “Waller had recorded similar curves in mammalian hearts, considered them at first as a normal phenomenon, but attributed them later to injury.” Bayliss and Starling by the end of the 19th century showed beyond any doubt the relationship between ST-segment displacement and cardiac injury. In their experiments, Wolferth et al noted that epicardial injury is associated with a reversal of the inverted T waves to an upright position.¹⁵ Of note is that although the illustrations in their article revealed changes in the QRS complexes, no mention was made of them, probably stemming from the preoccupation at that time with the +ST.¹⁵ Hellerstein and Katz¹⁶ observed that subepicardial injury produced a decrease in the amplitude of the S-wave simultaneously with +ST with an increase in QRS complex duration, which they attributed to “local focal block”, and all these were preceded by changes in the T waves, but again no mention of changes in the amplitude of R waves was made.

In one of most widely quoted article of Prinzmetal et al,¹³ the authors referred to Hellerstein and Leabow,¹⁷

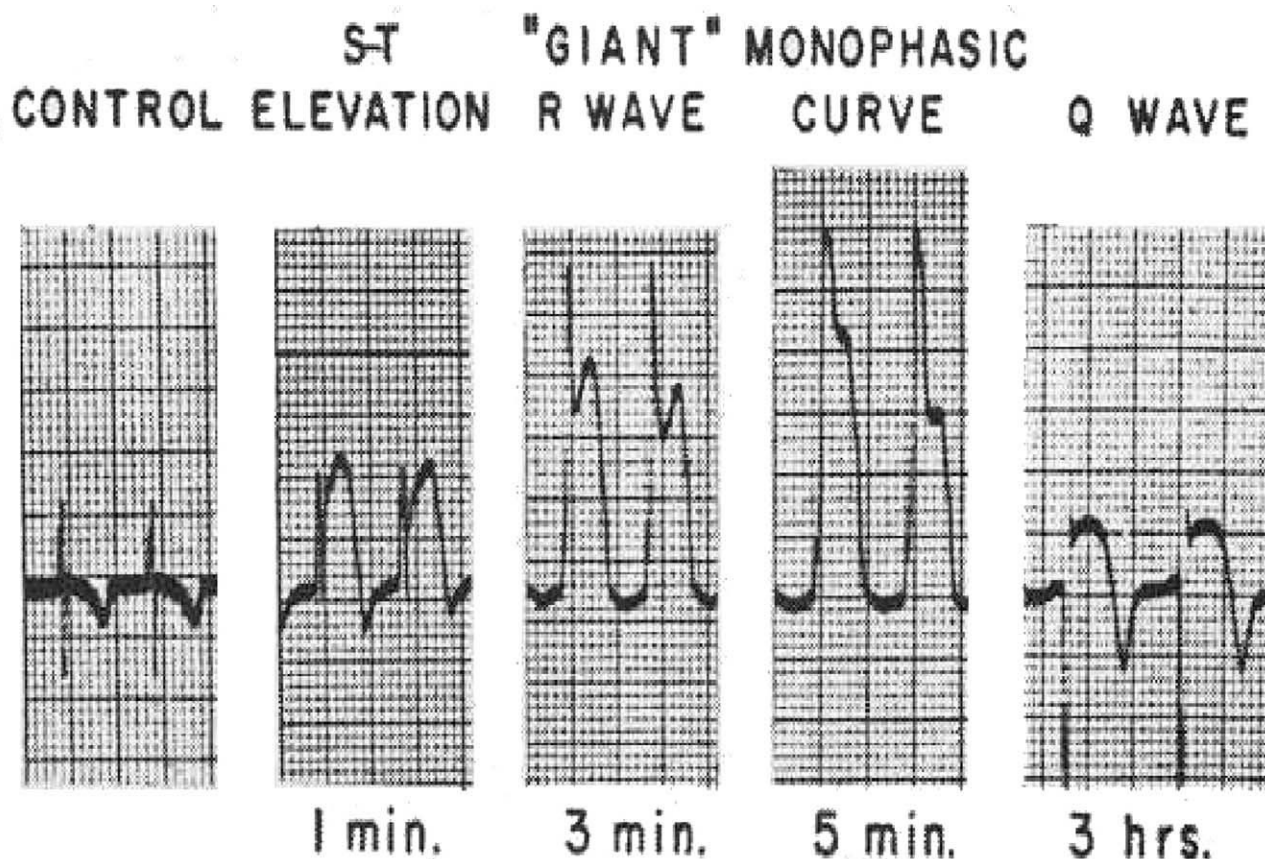


Fig. 5. Electrocardiographic changes after ligation of the left anterior descending coronary artery, which led to ST-segment elevation with progressively larger increases of the amplitude of the R waves, development of a “giant” R wave, widening of the QRS complexes, and development of monophasic potentials, with eventual progression to Q waves, decrease in the amplitude of the ST-segments and inverted T waves. Note that the amplitude of the ST-segment elevation is more than one half of the amplitude of the corresponding R wave. Adapted from reference [11], with permission from the *American Journal of Cardiology*.

and Sodi Pallares (personal communication), who “along with +ST showed delay in the peak of R-wave, an increase in the amplitude of R-wave, and a decrease in the amplitude of S-wave.” Sodi Pallares “attributed the QRS changes to a functional blocking of depolarization in the injured region” and elaborated on this matter in his memorable book (Fig. 6).¹⁸ Similar QRS changes, along with +ST, were described by Pruitt et al¹⁹ in subendocardial ischemic injuries, when the recording electrode was placed *intracardially*. The list of contributors to this concepts from the past 2 centuries is too long to be included herein. However, the contribution of Prinzmetal et al was in the systematic research exploitation of these concepts. In a similar vein Sclarovsky, Birnbaum et al considered all that had been done before, scrutinized the +ST and QRS changes in patients with an AMI, devised their S-B score, and applied it in earnest in all conceivable clinical scenarios.

A critique of the “Sclarovsky-Birnbaum ischemia score”

Even great works have their critics, and this author wishes to “air” some thoughts concerning the S-B score. The emphasis of Sclarovsky, Birnbaum et al for using a cutoff point of one half for the amplitude +ST in comparison with

the amplitude of the corresponding R waves to differentiate between “grade 2” and “grade 3” ischemia is central to the S-B score and appears, at least to this author, counterintuitive for continuous variables such as the +ST, R waves, S waves, and QRS duration, which are expected to occupy different loci in a spectrum and to continuously change, in a positive or a negative direction, in the early phase of AMI. Dichotomizing continuous predictors in general has been thought not to be a good idea.²⁰

The S-B score always relies on the admission ECG, a mere snapshot in the spectrum of changes, rather than having the advantage of considering the ECG appearance before the onset of the ischemic insult, or additional ECGs to examine progression or regression of the different grades of ischemia. It is understandable that the former is most often missing or unavailable in patients presenting with an AMI, and that the patients need to undergo revascularization expeditiously, but an effort needs to be made to record some ECGs, even in proximity to the admission tracing, while preparation for thrombolysis or percutaneous coronary intervention (PCI) is under way, to ascertain whether there are any short-term changes of “pattern C” to “pattern B” or “pattern B” to “pattern C.” Otherwise stated, are we referring here to a dichotomy and “qualitative” distinction, or a spectrum of changes and a “quantitative” difference, as in most things in natural systems? Intuitively, it appears

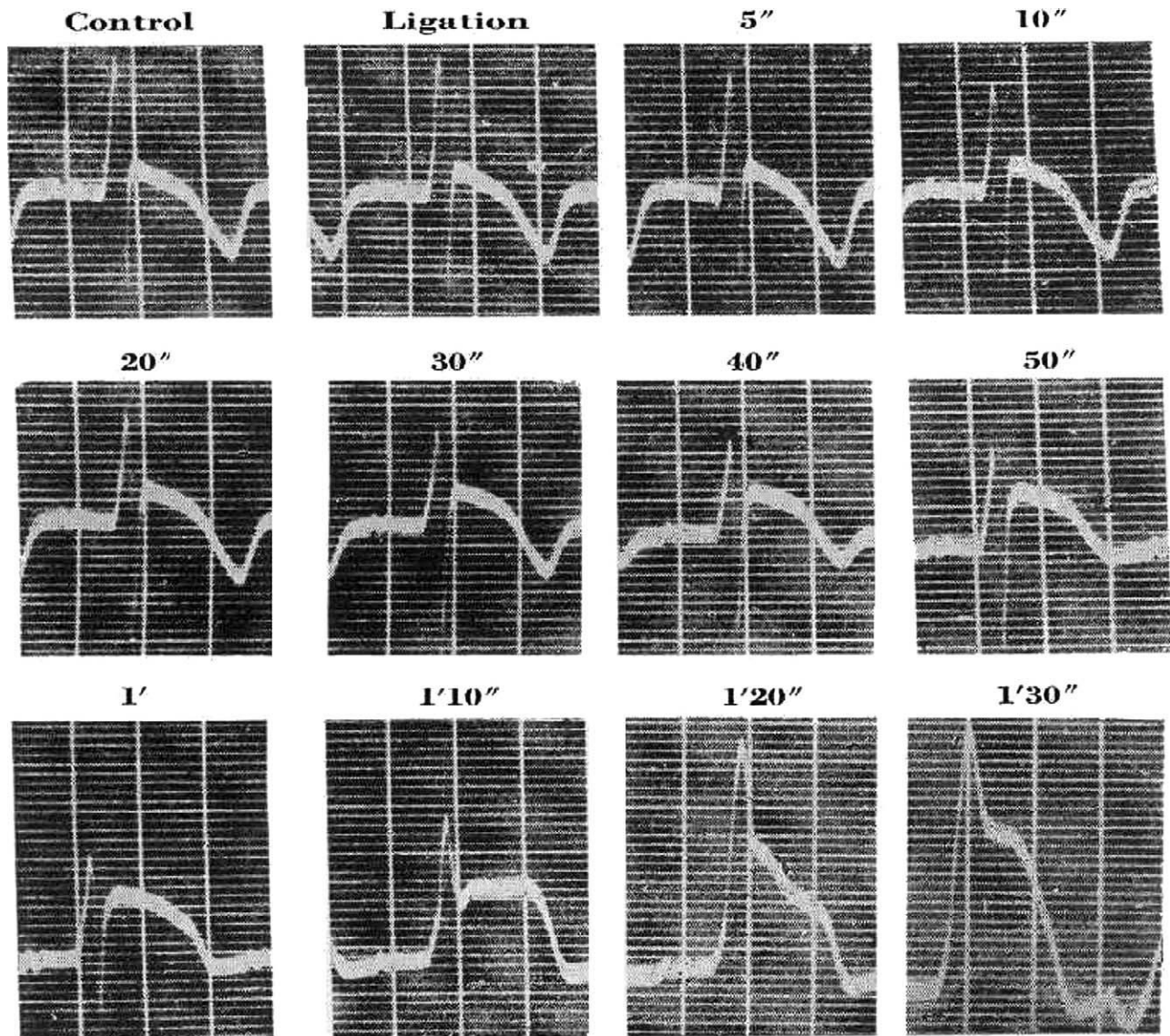


Fig. 6. Electrocardiographic changes a few seconds after ligation of the anterior descending coronary artery include delay in the intrinsicoid deflection, +ST, disappearance of the S wave, increase in the R-wave voltage, and widening of the QRS complexes; note that the appearance of the ECG at different time points could be categorized as “grade 2” or “grade 3” ischemia as per S-Bs, depending on the amplitude of +ST in comparison with the amplitude of the corresponding R wave. Adapted from reference [18], with permission from C. V. Mosby Co.

odd that certain +ST (>50%) in comparison to the corresponding R-wave amplitude has been found to be stable over long periods and thus differentiates patients to well-demarcated prognostic groups. One would expect more likely that the pattern of ECG changes is continuously changing, and what is viewed as “grade 3” ischemia may be just a phase, mostly early phase, in the process. Thus, this pattern would be expected to change, and thus the +ST would be expected to increase or decrease, and not to be characteristically “different” in different patients. Perhaps the best way to resolve this dilemma is by continuous ECG recordings instituted on first contact with a few patients with an AMI, and continued in the ambulance en route to the hospital and until implementation of reperfusion. Alternatively, insight could be obtained about the stability of “grade 2” and “grade 3” patterns of S-B score by serial or continuous ECG recordings during the course of balloon

inflations in PCI. It is conceivable that patients with an AMI presenting with a “pattern B” ECG have gone already earlier through a phase of “pattern C” ECG, and thus they are allocated to the “wrong” ischemic category based on their admission ECG. This is important when one uses a variable that remains stable and can be used as a predictor, as opposed to one that is subject to change and thus cannot serve for this purpose.

It is obvious that this author subscribes to the notion that the absolute and relative (to the corresponding R-waves) amplitude of +ST is subject to continuous change, and thus one cannot use a particular stage to define a specific degree of ischemia. This is also supported by the fact that both examples of “pattern C” and “pattern B” ECG features coexist in epicardial recordings after coronary ligation in canine experiments (Fig. 4) or in different ECG leads in patients with myocardial ischemia. Sclarovsky also noted

that the maximal ischemic changes in patients with AMI and ST-segment elevation were present in the leads representing the “core of ischemia,” whereas the border zone showed milder changes; typically, with occlusion of the left anterior descending coronary artery, the “core of ischemia” is present in leads V_2 to V_4 (“grade 2” or “grade 3” of ischemia), whereas leads V_5 to V_6 often show “grade 1” ischemia with prominent T waves.²¹ In some patients, ECG features of “pattern C” are seen only in a single ECG lead, and consequently categorization of such patients has been found by Sclarovsky and Birnbaum problematic. Indeed this has prompted the adoption of an ischemic “grade 2.5” between “grade 2” and “grade 3” in their S-B score, for patients who had ECG features of “grade 3” ischemia in a single lead.²² This inevitability results often when one “forces” a continuous variable to a categorical one.²⁰

On the other hand, the above may constitute a purist’s take of the matter, and thus one should not overlook the success of the implementation of the S-B score in so many facets in the study of patients with AMI. Besides there are many examples in medicine where continuous variables have been implemented after being transformed to ordinate scales with big success both in research and practice (eg, age, degree of aortic stenosis, and many TIMI group scores). Another important matter is the one associated with the effect of preconditioning and protection of ischemic regions, via emerging (during the balloon inflation) collaterals in PCI. Sclarovsky²¹ observed in patients undergoing coronary angioplasty of the left anterior descending coronary artery, with at least 3 inflations for at least 2 minutes duration, and in the absence of angiographic collateral flow, 4 different ECG patterns: (1) evolution to “grade 3” in all inflations, probably representing unprotected myocardium; (2) evolution to “grade 3” during the first inflation, and “grade 2” or “grade 1” in the next inflations, possibly representing the development of a protective mechanism by the myocardium; (3) evolution to “grade 2” during all inflations, possibly representing partially protected myocardium; and (4) evolution to “grade 1” in all inflations, probably reflecting well-protected myocardium.

Finally, one wonders whether the sequence of ECG features observed in the seconds/minutes in the process of PCI constitutes a suitable model for the changes occurring in patients with AMI; also, it may be argued that the animal models may not be appropriate for application in the human AMI. Thus, it is understandable that Sclarovsky, Birnbaum et al have voiced the same concern in their literature.^{2–6} This leads one to conclude that while we continue to implement the S-B score in practice and research, we should resolve whether the “grade 3” ischemia ECG pattern in patients with AMI is of the same nature as the ECG changes produced in the laboratory animal with coronary occlusion and in patients undergoing PCI.

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