LETTERS TO THE EDITOR

Letters to the Editor will be published, if suitable, and as space permits. They should not exceed 1,000 words (double spaced) in length, and may be subject to editing or abridgment.

Prognostic Value of ST-Elevation

To the Editor:

The paper by Nielsen in the August 1973 issue of "Circulation" on the prognostic importance of the ST segment elevation in acute myocardial infarction touches a point which has not been dealt with in the literature heretofore: the quantitative assessment of the ST segment elevation in the 12 lead standard ECG. The latter has been only qualitatively utilized in the diagnosis of myocardial infarction, although changes of the magnitude of ST shifts — elevation or depression — have long been suspected by the practicing physician to reflect changes in the clinical status of patients.

Good correlation between sums of ST shifts and magnitude and extent of ischemic injury have been demonstrated in several studies. Follow-up of patients with myocardial infarction has been facilitated, and extension of the initial ischemic damage has been detected using various precordial mapping systems. The question arises if the standard 12 lead ECG can similarly be used as an objective index of ischemic injury. Nielsen has found good correlation of the magnitude of ST elevation and prevalence of cardiac arrest, congestive heart failure, death, shock and certain arrhythmias, and conduction defects, using the standard 12 lead ECG. These correlations existed despite the fact that a random baseline ECG was used for the analysis, and no serial studies were done.

Some information is presently available regarding the relative merits of the 49-lead and conventional 12-lead systems in evaluating both magnitude and extent of ischemic injury. In a systematic comparison of the two systems, we have demonstrated the superiority of the 49-lead system in assessing magnitude of ischemic injury. Similarly, Reid and his colleagues noted that extension of infarction was detected by 48-lead maps in a considerable number of patients in whom changes were absent in the conventional 12-lead ECG (fully one-third of a series of 12 patients).

Thus, although the standard ECG "scans" only a fraction of the recording sites of the mapping system, the study of Nielsen should stimulate clinicians to look at the standard ECG in a more quantitative fashion, since mapping techniques have not been widely applied as yet. Summation of ST elevation derived from the six precordial leads for patients with anterior myocardial infarction and leads II, III, and aVF for patients with inferior myocardial infarction may produce an index useful in the follow-up of patients in the Coronary Care Unit. For the latter type of infarct, as well as for nontransmural myocardial infarction, the sums of ST depressions in the standard precordial "V" leads may also prove useful.

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References


Thrombosis and Myocardial Infarction

To the Editor:

In the second of his series of two excellent editorials discussing the role of thrombosis in atherosclerosis and acute myocardial infarction (Coronary thrombosis and fatal myocardial ischemia, Circulation 49: 1, 1974) William Roberts listed five factors which tend to indicate that coronary thrombosis is a consequence, rather than a precipitating cause of acute myocardial infarction. His first two points, the low frequency of
thrombi in patients dying suddenly and the increased frequency of thrombi with increasing intervals between infarction and death, may not necessarily support the hypothesis that thrombosis is secondary rather than primary.

Fibrinogen is composed of three pairs of subunit polypeptides, designated Aα-, Bβ- and γ-chains. Thrombin cleaves fibrinopeptide A and B from Aα- and Bβ-chains, respectively, and the resulting fibrin monomers (αβγ)n align to form noncrosslinked fibrin [(αβγ)n]n.1 In the presence of fibrin stabilizing factor (FSF, factor XIII), a transglutaminase, intermolecular covalent ε-(γ-glutamyl) lysine crosslinks are rapidly formed between COOH-terminal regions of the γ-chains. The α-chains crosslink more slowly (over the period of several hours) to give highly crosslinked fibrin. Polymerization of the α-chains is known to markedly increase the resistance of fibrin to lysis while fibrin containing no crosslinks or γ-chain crosslinks is more susceptible to digestion by plasmin.2 Thus freshly formed fibrin clots are easily lysed by plasmin, where as fibrin that has had the opportunity to fully crosslink is resistant to lysis.

Since the hypoxia and acidosis that immediately precede or accompany death can markedly stimulate fibrinolysis, freshly formed clots that have not crosslinked extensively could be lysed and would not be found at autopsy, particularly if there is a delay of several hours between death and postmortem examination. Therefore, the absence of thrombi in patients dying suddenly and the increased frequency of thrombi with duration of infarction may reflect the extent of crosslinking of α-chains of fibrin and consequent susceptibility of fibrin to removal by plasmin rather than the initial presence or absence of thrombi.

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References

The Fourth Heart Sound
To the Editor:

I trust that Dr. Tavel will publish a reasonably prompt retraction of the totally false attribution to me of a “statement” cited in his Editorial on the fourth heart sound.1 This ran: “Notwithstanding Spodick’s statement to the contrary, we are able to record splitting of the first heart sound in most normal individuals.” No such “statement” exists. The citation purports to be from a letter by me (Tavel’s reference 12) which nowhere states anything of the sort.2 The nearest thing to it was a plea for an appropriately designed controlled study of the split first sound to establish both its audibility and prevalence.9

Perhaps it is significant that the source of the erroneous “statement” contains a final summation which bears on — and indictsa - the traditionalist ex cathedra approach of the erroneous Editorial, to wit: "In any case you cannot beat a formal investigation with a noninvestigation, nor a study that tries to minimize bias with any other kind of study."9 This implies that Tavel’s message might be dead right — but his case is not made in an acceptable way. Our findings on the prevalence of S4 (phonocardiographic, auscultatory, or both) in older normal persons cannot be seriously refuted by the editorialist’s impressionistic old chestnut, "It is my experience . . . ."1 Our findings might indeed be mistaken, but in today’s world proving this requires an appropriately designed study which must include minimizing observer biases. To be sure, the experience of an expert of Tavel’s stature cannot be ignored (and much of it conforms to my own impressions). Yet, uncritically examined common “experience” led for centuries to the quite reasonable conclusion that the Earth was flat.

One can easily accept Dr. Tavel’s discussion of the mechanism of production of the pathologic S4 and the coupling of pathologic audibility of its A-wave counterpart. However, extrapolation from this kind of demonstration does not establish the practical threshold of audibility. One can make further reasonable assumptions based on the logarithmic response of the human ear to the frequency-amplitude product of artificial test sounds. But any auscultatory study requires actual study of auscultation itself — i.e., observer performance in a controlled investigative protocol. In this regard, figures 1 and 2 of the Editorial prove nothing but the acknowledged coincidence of S4 with the A-wave peak (in what appear to be low time-constant ACGs).1 Indeed, in figure 2, the inaudible S4 is either superimposed on or merged with the low frequency component of S1; moreover, S4 consists of a train of vibrations. The question naturally arises as to which individual vibrations were, in fact, audible in such a configuration. In our department, auscultation in conjunction with oscillographic PCG monitoring demonstrates that it is often difficult to identify any individual "audible" vibration.

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