In the control trial of low-dose heparin among 4,121 patients, referred to by the Council, Kakkar reported 158 cases (7.7%) of wound hematomas and 182 cases (8.9%) of excessive bleeding in 2,045 heparin-treated patients for a combined complication rate of 16.6%. The Special Report mentioned only 158 cases and mislabeled these as "excessive bleeding." Many surgeons who are familiar with Kakkar's work have not thoroughly analyzed his data; the inaccuracies in the Special Report could be misleading.

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The authors reply:
To the Editor:

The letter by Drs. Pletcher and Riles is correct in calling attention to an error in the Special Report. On the left hand side of page 426A, 4th paragraph, line 15, 182 (8.9%) should be substituted for 158 (7.7%); and on line 17, (6.1%) should be substituted for (5.6%). From the data in the multicenter trial, it is not evident, as the writers claim, that the patients with excessive blood loss and those with hematoma formation were entirely separate and therefore additive.

Support for the slightly increased risk of bleeding does not, however, rest primarily on the large trial already mentioned. In a review of five other trials of low-dose heparin in which specific attention was given to the measurement of blood loss during and after elective general surgery, there was no clinically significant difference in bleeding between the control and heparin treated groups and in no instance was a fatal bleeding episode ascribed to heparin.

It is important to reiterate that the Special Report clearly states, in three separate sections, that there will be an increase in bleeding from low-dose heparin regimens. The Special Report emphasizes that patients considered for a low-dose heparin regimen should, prior to operation, be hemostatically competent. This term means that by history, physical examination and appropriate laboratory tests, the patient does not have a recognizable bleeding tendency. Particularly important in this assessment is abstinence from aspirin for five days prior to operation, since the combination of aspirin and low-dose heparin may lead to more bleeding in surgical patients than with either drug alone. Such a recommendation, it should be noted, has not been included in the published trials of low-dose heparin among surgical patients.

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References

The authors reply:
To the Editor:

We appreciate the comments of Dr. Abinader concerning increased understanding of the exercise electrocardiographic response. In particular we welcome his contribution to better interpretation of the manifestations of Barlow's (click-murmur) syndrome. But we are sorry that in writing that he disagrees with our exclusion of patients with LVH, intraventricular conduction defect, etc., he indicates that he misunderstood the thrust of our report. He and we are well aware that these conditions (also Barlow's syndrome) cause exertional ST-segment displacement which may not be due to coronary atherosclerosis. The purpose of our study was to identify additional patients whose exercise ST-segment displacement may correctly be attributed to coronary artery disease. We feel that we have succeeded in doing this, since in the group of patients we defined, an additional 0.1 mV of ST depression in Wilson lead V4, corresponded with significant coronary artery disease in 92%. By Dr. Abinader's own evidence the reliability of exercise ST depression as an index of coronary disease would have been degraded if any known sources of false positive results (such as Barlow's syndrome) had been included.

Much remains to be learned about electrocardiographic responses to stress, and progress in this field is aided by further investigation into the means by which specific disease entities may be identified in spite of recognizable confounding variables.

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Stress Testing and the ST Segment

To the Editor:

The article by Kansal and colleagues on stress testing with ST-segment depression at rest is a welcome contribution to the field of exercise electrocardiography, particularly as it correlates the ECG changes with coronary angiographic findings in this selected group of patients. However, we are at variance with the authors in relation to the conclusions reached concerning the patients which they have excluded, namely those with LVH, intraventricular conduction defects, and digitalis effect. The omission of the systolic click murmur syndrome (SCMS) from their excluded group warrants comment.

Resting ST-T changes are frequently encountered in patients with SCMS, and such changes may be accentuated on effort. Significant ST depression of 1 mm or more has been produced by submaximal or maximal exercise testing in ten to sixty percent of prolapse patients with a history of chest pain, thus yielding false positive results. When one considers the high prevalence of the SCMS in the population at large with the associated changes in repolarization, the authors' omission of these cases becomes even more significant. Furthermore, we are convinced that the ST-T changes in the SCMS are noncoronary in nature and as such should have been taken into account along with the other conditions which give rise to non-ischemic ST-T changes.

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References

The authors reply:
To the Editor:

I would like to call attention of the authors of the paper entitled "Oral hydralazine therapy for chronic refractory heart failure" (Circulation 54: 879, 1976) to the early work done by the following investigators:


Hydralazine Therapy

To the Editor:

I would like to call attention of the authors of the paper entitled "Oral hydralazine therapy for chronic refractory heart failure" (Circulation 54: 879, 1976) to the early work done by the following investigators:

Stress testing and the ST segment.
E G Abinader

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