passes the normal physiologic variability in net washout rate." Drs. Sklar et al. in their letter indicated that while this criterion is convenient and clinically satisfactory, it does not represent exactly ±2 s.d. from the mean of the slopes obtained from the normal group. This is an entirely correct interpretation of our paper.

Several questions concerning uptake and washout were raised in the letter. First, since the absolute washout rate depends upon several variables aside from myocardial thallium uptake, the washout rate cannot be used to imply or to substitute for the measurement of initial thallium distribution. The initial thallium distribution, redistribution, and segmental washout rates are probably best viewed as three separate entities (even though they are not completely independent). A myocardial segment can have reduced uptake and normal washout, which would be observed as a persistent defect. A myocardial segment may have reduced initial uptake with delayed washout, compared with normal myocardial segments, and this would produce classical redistribution (i.e., delayed normalization of the defect). In this case, the abnormal segment washes out more slowly than the normal segment, but does not necessarily have an absolute washout that is outside normal limits. In more severe defects, redistribution may result from increasing uptake of the abnormal segment. Increasing uptake in all myocardial segments in the absence of significant initial defects can occasionally be observed in cases of diffuse symmetric multiple-vessel disease, in which case no normal myocardial segment is available for comparison. In these cases, an *apparent* "reverse redistribution" can occasionally be observed when we compare two abnormal myocardial segments both of which have similarly reduced initial uptake but dissimilar washout rates. We have not quantitatively substantiated the case of *true* "reverse redistribution" resulting from a segment that has completely normal initial uptake but abnormal washout rate, which would produce a reverse defect in the delayed images. This would require a myocardial segment with normal blood flow and normal extraction coefficient, but with abnormal cellular washout rate, and would be illogical in the context of coronary artery disease. Reverse redistribution occasionally appears on scintiphoto images, but we have found on quantitative evaluation that it is nearly always the result either of comparing two abnormal myocardial segments under the incorrect assumption that one of the segments is "normal" or, in some cases, a photographic distortion resulting from the use of nonlinear gray-scale reproduction.

We wish to thank Drs. Sklar, Steele, and Kirch for their comments and for providing this forum for discussion.

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Re: Indium-111 Tropolone Versus Oxine

As a research biochemist having developed an aqueous ethanol-free In-111 oxinate preparation that proved to be an efficient cell-labeling agent especially for leukocytes and platelets (*1*), I would like to comment on the article by Dewanjee et al. titled: "Indium-111 Tropolone, A New High-Affinity Platelet Label: Preparation and Evaluation of Labeling Parameters (*2*)."

The statements concerning the solubility of oxine and the need for ethyl alcohol as a solvent are erroneous, and the statements about the ability of indium-111 tropolone to label platelets in a plasma environment are misleading and may raise false hopes in experiments.

The second line of the Summary contains the following statements: "Unlike oxine, which must be dissolved in ethyl alcohol, tropolone is soluble in isotonic saline." However, oxine used in the concentration levels current in cell-labeling procedures is soluble in saline without the help of ethyl alcohol or a solubilizer (*3*).

In the sixth line of the Summary I read that indium-111 tropolone would be able to yield 60-70% labeling efficiency with platelets in an ACD plasma medium. From Fig. 2, however, it is clear that only in cases of extremely low plasma concentrations, below 50 μl/ml, can labeling efficiencies between 40 and 50% be obtained. When the incubation mixture contains 250 μl/ml (25%) plasma, the labeling efficiency is only about 20%. For indium-111 oxinate and incubation mixtures containing more than 50% plasma, labeling efficiencies over 20 and up to 50% are obtained (*4*). Consequently there is no advantage in using tropolone instead of oxine. Is it realistic to speak of "plasma medium" if it contains only 50 μl plasma per ml incubation mixture?

In the Discussion there is an erroneous statement that HEPES or Tris buffer should be necessary as a solvent if acetylacetone is to be used. HEPES and Tris function as buffers. They don't function as solubilizers and they don't interfere with platelet function.

Let me conclude with a suggestion. Why not use the correct chemical names for indium chelates, such as indium-111 oxinate, indium-111 tropolonate, indium-111 acetylacetonate?

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Reply

I tend to disagree with Dr. Goedemans regarding the solubility of oxine and In-111 oxine in water. It is likely that a trace amount of oxine and In-111 oxine might be in solution, but the major fraction of In-111 oxine is in insoluble form without alcohol. The exact physical form of these neutral In-111 complexes in water is not known. A major fraction of the complex is retained in the filter paper (0.22 μm Millipore or Nuckopore filter), and most of these complexes tend to be sticky. The exact physical form is irrelevant as long as we obtain constant labeling efficiency maintaining cell viability.

In an ideal cell-labeling system, we would like to add minimum amounts and kinds of chemicals including buffer or organic solvent. In an In-111 tropolone preparation we use In-111 chloride, 20-25