In the meantime, it is essential that doctors become more aware of the risks of the sulfonylurea hypoglycemic drugs and use them cautiously, when all other therapeutic options have been exhausted.

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REFERENCES

Describing Patients With Discordant Ventriculoarterial Connections
I congratulate Blume et al. (1) on the excellence of their analysis of patients undergoing the arterial switch operation. I am surprised, however, when they state that the larger part of their patient group had "d-transposition," without providing further definition of this contentious term. Does this mean, for example, that they excluded those patients with complete transposition in which the aorta was to the left, such as those with mirror-imaged atrial arrangement (transposition [I,L,L])? And did they include those patients with a congenitally corrected transposition with right-sided aortas, such as those with the transposition [I,D,D]? The group from Boston should now describe the anatomy of their patients with the same accuracy of analysis which they apply to their results.

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REPLY
We thank Dr. Anderson for his comments. We agree with him that the importance of anatomic accuracy cannot be overemphasized. We did not exclude, on the basis of the spatial relation of the aortic valve relative to the pulmonary valve, any patient from the analysis. In fact, the interrelations between the semilunar valves were specified in the Results section of our report. Furthermore, we specified that only one patient had transposition of the great arteries with viscerocrural situs inversus (segmental anatomy [I,L,L]), and that patient was included in the analysis. We also specified that patients with physiologically corrected transposition of the great arteries [S,L,L] were excluded.

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Coronary Endothelial Dysfunction After Kawasaki Disease
Although the question addressed in the article by Yamakawa et al. (1) is interesting, there are several issues to be discussed with respect to the methods and interpretation of the results.

Study protocol. The dose–response curve of normal coronary arteries to acetylcholine (ACH) in their study is inconsistent with previously established pharmacologic properties of coronary arteries, on the basis of in vivo and ex vivo data in humans (including children) and animals (2,3). Although ACh, in fact, dilated normal epicardial coronary arteries only at high estimated concentrations (3.0 to 6.0 μmol/liter, estimated final blood concentrations) in their study, it has been established that ACh does so in a dose-dependent manner at 0.01 to 1.0 μmol/liter, at which level the contribution of nitric oxide to ACh-induced response has been demonstrated (2,3). This difference might be due to the short infusion time of ACh (30 s) in their study, as compared with 2 to 3 min in other studies. What could be the mechanism of the ACh-induced vasodilation at 3.0 to 6.0 μmol/liter, but not at 1.0 μmol/liter? In addition, the dose of ACh could be individualized when it is administered into either right or left coronary arteries in children of different ages. Because Yamakawa et al. (1) showed ACh-elicted paradoxic vasoconstriction at 10.0 μmol/liter, the relatively narrow range of effective ACh concentrations, as compared with those in previous reports, might in fact have produced highly variable responses to ACh.

Analysis of coronary angiogram. Although Yamakawa et al. concluded that normal coronary arteries but not regressed aneurysm exhibit a normal ACh response in patients with Kawasaki disease (KD), the angiograms could be investigated more carefully. They showed in Figure 3 that normal coronary arteries after KD (left main coronary artery and distal portion of the left circumflex artery [LCx]) dilated in response to ACh, whereas the regressed aneurysm (proximal portion of the left anterior descending coronary artery [LAD]) constricted. Readers might observe, however, that other segments (proximal portion of the LCx and...