different possible types of ventriculoarterial alignments (ACM, DORV, TGA, or DOLV), hearts with {S,D,L} segmental anatomy nonetheless constitute a syndrome or spectrum of anomalies that may have a similar morphogenesis.²⁻⁵

Otero-Coto chides us by saying that TGA {S,D,L} is "not so newly recognized" a complex as we said it was. His reaction in this respect is understandable, because he well remembers his own work on this subject.²⁻⁵

However, we think that Otero-Coto may not have fully understood what we meant. We said: "The transposition of the great arteries {S,D,L} complex is delineated for the first time from the anatomic, diagnostic, and surgical standpoints in this study of 26 cases: 16 surgical and 10 postmortem."¹ We did not mean to suggest that TGA {S,D,L} had never been published previously. Indeed, one of us (R.VP.) reported three postmortem cases of TGA {S,D,L} in 1968⁶ and six additional autopsy cases in 1977.⁷ In our 1995 paper,¹ we did not refer to our own previous reports of TGA {S,D,L},^{6,7} or to those of Otero-Coto²⁻⁵ or others,⁸ because our paper¹ was not intended to be a literature review.

Instead, one of the main purposes of our recent publication¹ was to demonstrate that TGA {S,D,L} is statistically significantly different from the more common TGA {S,D,D} in at least six different respects. This study¹ was the first time, to our knowledge, that TGA {S,D,L} was shown by statistical analysis to be a transposition complex that is significantly different from the more common form of TGA {S,D,D}.

Six anatomic features that were found to be significantly more frequent in TGA {S,D,L} than in TGA {S,D,D} were as follows:¹ (1) *ventricular septal defect*, in 96% versus 15% to 31% ($p < 0.01$); (2) *conal septal malalignment* in 80%, versus 21% ($p < 0.01$); (3) *right ventricular hypoplasia* in 50%, versus 0 ($p < 0.01$); (4) *pulmonary outflow tract stenosis* in 27%, versus 10% ($p < 0.01$); (5) *ventricular malposition* such as superoinferior ventricles and criss-cross atrioventricular relations in 23%, versus 0% ($p < 0.01$); and (6) *absent left coronary ostium* resulting in "single" right coronary artery in 23%, versus 5% ($p < 0.02$).

This is the first study,¹ to our knowledge, in which a quantitative mathematical method has been used to delineate what the TGA {S,D,L} complex really is.

Nonetheless, our findings and conclusions (summarized above) should be regarded as approximations. Further experience will continue to clarify the various anatomic features of the TGA {S,D,L} complex. Again, we thank Otero-Coto for his commentary.

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12/8/72122

Location of the conduction tissue in double-inlet left ventricle with leftward rudimentary right ventricle

To the Editor:

It seems to me that van Son and his colleagues¹ may be confused with regard to the location of the atrioventricular bundle in hearts with univentricular connection to a dominant left ventricle and leftward rudimentary right ventricle. They state in their article that hearts with {S,L,L} segmental anatomy are at risk of surgical heart block because "the nonbranching atrioventricular bundle courses at the superior-anterior margin of the BVF [bulboventricular foramen]." But this is true only when the bundle is viewed through the morphologically left ventricle! As we explained in an article by Cheung and associates,² cited by van Son and his colleagues (their reference 12), when viewed from the rudimentary and incomplete right ventricle, the bundle courses posteroinferiorly. Because of this, it is always safe to remove a wedge of muscular tissue from the apical trabecular septum by incising close to the obtuse margin of the ventricular mass (Fig. 1). This holds true whether the rudimentary right ventricle is right-sided or left-sided. These relationships

Fig. 1. The anterior view of the leftward rudimentary right ventricle from a heart with double-inlet left ventricle, showing the segment of apical trabecular septum that can safely be removed to enlarge the ventricular septal defect (VSD) without damaging the atrioventricular bundle.

are also discussed at length in the book *Double Inlet Ventricle*.³

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Reply to the Editor:

We have read the letter from Dr. Anderson regarding the location of the atrioventricular conduction bundle in {S,L,L} hearts and are in complete agreement with the location of the bundle in these hearts. In fact, we acknowledged Dr. Anderson's excellent work in this area in our article.

We wish to emphasize, however, that we are not confused about the location of the conduction system. The statement that we made in the article that "the non-branching bundle courses at the superior margin of the BVF [bulboventricular foramen]" refers to the anatomy when viewed from the transatrial approach, that is, from the left ventricular side. This is our preferred approach for examining the BVF, because it is the least destructive.

We are in complete agreement with Dr. Anderson that sometimes it is possible to resect a wedge of tissue from the apical trabecular septum by incising close to the obtuse margin of the ventricular mass. However, more extensive resection is often needed in severely restrictive BVFs, and this is especially more destructive in neonates and small infants. In such patients we prefer to perform a Damus procedure. Furthermore, the intraoperative manipulation necessary to expose the BVF through the rudimentary chamber, the great vessels, or the atrioventricular valve results in substantial distortion. When this problem is combined with the intrinsic fragility of the bundle in patients with {S,L,L} hearts, the risks of conduction injury are high.

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Surgical technique and atrial arrhythmias after total cavopulmonary connection

To the Editor:

We read with much interest the report by Hashimoto and associates¹ in the September 1995 issue of the Journal, in which a method of total cavopulmonary connection with an autogenous intraatrial tunnel was presented. Postoperative atrial arrhythmias are a widely recognized and very troublesome clinical entity after various forms of Fontan repair.² We have several questions pertaining to the occurrence of postoperative atrial arrhythmias after the technique described in this article.

The authors state that "preserving the crista terminalis and the sinus node and its arteries" prevented the development of postoperative atrial arrhythmias. Sinus node dysfunction and conduction abnormalities related to the crista terminalis have been linked to the genesis of atrial arrhythmias, both in patients who have had the Fontan operation^{3,4} and in naturally occurring human atrial flutter.⁵ Because we are presently working with an experimental model to elucidate more clearly the electrophysiologic importance of these factors with respect to atrial flutter after the Fontan operation, we are very interested to know whether the authors based their conclusions on