A new anatomic approach to the ventricular septal defect in interruption of the aortic arch.

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Objectives: To analyze the anatomy of the ventricular septal defect (VSD) in heart specimens with interruption of the aortic arch (IAA), in order to confirm the hypothesis of different embryologic mechanisms for the different anatomic types of IAA.

Material and methods: We examined 27 hearts from the anatomic collection of the French Reference Center for Complex Congenital Heart Defects with IAA, concordant atroventricular and ventriculoarterial connections, and 2 distinct great arteries. Hearts were classified according to Celoria and Patton: type A, interruption distal to the left subclavian artery (A), type B, between the left subclavian and the left carotid artery (B), type C, between the 2 carotid arteries (C). We focused on the anatomy of the VSD viewed from the right ventricular side.

Results: There were 10 A, 17 B, no C. One A (with aortopulmonary window) and 1 B had no VSD. The VSD was conoventricular, located between the 2 limbs of the septal band, in 4/9 A and 16/16 B (p=0.005), with posterior deviation of the outlet septum. In A, the VSD was conoventricular in 4, with muscular borders in 2 and fibrous extension of the posterior limb of the septal band in 2, muscular in 3, membranous in 2. In B, the conoventricular VSD had entirely muscular borders in 4, fibrous extension of the posterior limb of the septal band in 9, and was juxta-arterial in 3; there was no fibrous continuity between the tricuspid and aortic valve.

Conclusion: The VSD in IAA type B is always conoventricular, with posterior deviation of the outlet septum, but without any fibrous tricuspid-aortic continuity. The VSD in IAA type A can be of any type. This reinforces the hypothesis of different pathogenic mechanisms responsible for the 2 types of IAA, and the inclusion of IAA type B in the group of conotruncal defects. The absence of fibrous tricuspid-aortic continuity indicates that the fibrous extension of the posterior limb of the septal band found in some hearts may be due to the posterior deviation of the outlet septum rather than to a perimembranous extension of the VSD.