

Prevention of residual ventricular septal defects with fibrin sealant.

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Abstract:

BACKGROUND: Modern echocardiography now allows for the detection of a substantial number of residual ventricular septal defects (VSDs) after surgical patch repair that remained hidden in the past. Mostly without hemodynamic significance, residual VSDs may have clinical consequences (progressive dehiscence, hemolysis, prophylactic antibiotic treatment, endocarditis). To reduce the number and size of residual VSDs we performed an experimental and a clinical study.

METHODS: (1) In an experimental setup, burst pressure of 60 fibrin glue-sealed defects (calibrated between 1.0 and 5.0 mm in diameter) was determined using a computerized recording system and pressure loads up to 500 mm Hg. (2) In a prospective clinical trial with blinded postoperative echocardiographic controls VSD closure was performed in 36 consecutive patients (age, 37 +/- 40 months; range, 4 to 134 months) using a polytetrafluoroethylene patch and running sutures reinforced with pledgets (22 of 36 patients) or sealed with fibrin glue (14 of 36 patients) in accordance to the surgeon's preference.

RESULTS: (1) Experimentally, mean pressure load achieved was more than 500 +/- 0 mm Hg for 1.0-mm defects, 413 +/- 52 mm Hg for 2.5-mm defects, 363 +/- 58 mm Hg for 4.0-mm defects, and 313 +/- 48 mm Hg for 5.0-mm defects ($r = 0.873$, $p < 0.001$). (2) Clinically, all patients survived. Residual VSDs at echocardiography were observed in 16 of 22 patients (72%) for reinforced versus 5 of 14 patients (36%) for sealed with fibrin glue ($p < 0.05$). Diameter of residual VSDs accounted for 1.3 +/- 1.2 mm for reinforced versus 0.3 +/- 0.4 mm for sealed with fibrin glue ($p < 0.01$).

Hemodynamically significant residual VSDs were found in 2 of 22 patients (9%) for reinforced versus 0 of 14 patients (0%) for sealed with fibrin glue ($p =$ not significant).

CONCLUSIONS: Small defects sealed with fibrin glue resist physiologic pressure load. Fibrin glue sealing of prosthetic patches during intracardiac VSD repair allows for significant reduction of number and size of residual VSDs. Improved long-term outcome can be expected.