Review Article

A Systematic Review of Acquired Left Ventricle to Right Atrium Shunts (Gerbode Defects)

Shi-Min Yuan

The First Hospital of Putian, Teaching Hospital, Fujian Medical University, Putian, Fujian Province, People's Republic of China

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Address:
Shi-Min Yuan

The First Hospital of Putian
Teaching Hospital
Fujian Medical University
389 Longdejing St.
Chengxiang District
Putian, Fujian Province
People's Republic of China
shi_min_yuan@yahoo.com, shiminyuan@126.com

The left ventricle-right atrium (LV-RA) shunt, a rare and special type of ventricular septal defect (VSD), was first reported by Gerbode et al[1] in 1958, and was thus termed "Gerbode defect", after the first author. In 1967, Riemenschneider and Moss[2] classified Gerbode defects into two types: direct (or true) and indirect (or false). The direct type is a direct shunt between the left ventricle and right atrium across the membranous septum, while the indirect type is a VSD with a left-to-right shunt at the ventricular level associated with tricuspid regurgitation, with the high-velocity shunt being directed into the right atrium. Later, Sakakibara and Konno[3] termed the above two types as supravalvular and infravalvular types. In addition, they added a Type III (valvular type), an intermediate type (or a combination) of types I and II, to the categories. In the early years, it was estimated that the incidences of the three types were 32%, 62% and 6%, respectively,[4] and that type II accounted for an overwhelming majority.[5] More recently, Sinisalo et al[6] stated that the incidences of the three types had changed dramatically, accounting for 76%, 16% and 8%, respectively. The LV-RA shunt can be either congenital or acquired in nature. The congenital Gerbode defect is rare, and its clinical features have been well described.[4] By contrast, the acquired LV-RA shunts have actually outnumbered the congenital, as there have been more predisposing risk factors, including infective endocarditis (IE), trauma, cardiac surgical procedures, and myocardial infarction, responsible for the development of the acquired shunt in the current era.[7] The acquired LV-RA shunts have been reported individually case by case in recent years. A review of acquired LV-RA shunts was published recently; however, it included incomplete patient information, most probably based on abstracts without referring to the full texts of the articles, and also improper inclusion of duplicate publications.[8] Therefore, the clinical characteristics and mechanisms remain to be sufficiently elaborated.

Literature review

A comprehensive literature search was conducted in the PubMED database. The search terms included "Gerbode defect" and "LV-RA defect/shunt/fistula/communication", with an additional filter of "human" and without any year limit. Exclusion criteria were ruptured sinus of Valsalva aneurysm, aorta-atrium fistula, LV-RA shunt of congenital etiology or formed by tricuspid valve aneurysm perforation, and duplicate publications. A literature search of Highwire Press, the Google search engine, J-Stage, Thesis Re-

Table 1. Literature retrieval.

Database	Reviewed	Inclusive
PubMed	102	67
Highwire Press	44	3
Google	101	3
J-Stage	46	5
Thesis Relation	81	19
"Baidu" Scholar	76	4
Chinese Medical Current Content (CMCC)	68	1
Bibliographic references	103	103

lation, "Baidu" Scholar and Chinese Medical Current Content (CMCC), as well as bibliographic references cited in the articles, ensured the completeness of the collection (Table 1). The search ended on March 31, 2015.

Data were carefully extracted with details of the study population, including demographics, clinical features, etiologies, diagnostic techniques, cardiac structural pathologies, hemodynamics, managements, follow-up duration, survival, complication and mortality, etc.

Quantitative data were expressed as mean ± standard deviation with range and median values. Comparisons of frequencies were made using Fisher's exact test. A p-value <0.05 was considered statistically significant.

Results

After exclusion of duplicate publications,[9,10] the comprehensive literature retrieval resulted in a collection of 205 articles[4,11-214] involving 234 patients, published until March 2015. These articles included 134 (65.4%) case reports,[a] 24 (11.7%) conference abstracts/posters,[b] 20 (9.8%) case series,[c] 11 (5.4%) medical images,[d] 7 (3.4%) conference/education course titles,[e] 6 (2.9%) original articles,[f] 2 (1.0%) "how-to-do-it"s,[119, 144] and 1 (0.5%) courseware.[132]

The patients' age was 45.2 ± 22.5 years (range, 1 month to 86 years; median 48 years) (n=202). Of the 200 patients whose gender could be tracked, 138 (69%) were males and 62 (31%) were females, with a male-to-female ratio of 2.2:1. The male patients were much younger than the females (43.0 ± 21.8 years vs. 50.4 ± 22.7 years, p=0.0296).

There were a total of 237 acquired LV-RA shunts in 234 patients, with one of the patients having a single recurrence and another having a double recurrence of the shunts. The underlying etiologies for an acquired LV-RA shunt could be categorized into four: iatrogenic in 121 (51.1%), infective in 87 (36.7%), traumatic in 22 (9.3%), and ischemic in 7 (3.0%) patients (χ^2=195.8, p=0.0000) (Figure 1). The onset time of the acquired LV-RA shunt was reported in 137 patients for 138 shunts. The time was described as "many years" for 9 (6.5%) shunts in 9 (6.6%) patients from a single report.[206] In 15 (10.9%) patients, the acquired LV-RA shunts developed immediately after cardiac interventions, where the onset time was recorded as "0". In general, the shunt developed at 20.5 ± 38.1 months (median 2; range 0-408) (n=49) after the impact of the inducing factor. The iatrogenic LV-RA shunt took much longer to develop than did the infective shunt (30.5 ± 56.3 months vs. 6.5 ± 18.0 months, p=0.0244) (Figure 2).

Types of shunts were given for 193 patients with 198 shunts, 160 (80.8%) shunts in 156 patients (80.8%) were type I,[g] 24 (12.1%) shunts in 24 (12.4%) patients were type II,[h] (one patient had a type I shunt and a recurrent type II shunt[110]), and 14 (7.1%) shunts in 14 (7.3%) patients were type III[i] (one patient had a type I shunt with 3 additional VSDs[175]). Apart from the 14 patients with a type III shunt, 15 (6.4%) patients had an additional intracardiac shunt, including a ruptured sinus of Valsalva aneurysm in 4 (2.7%) patients,[j] patent *fossa ovalis*[k] and left ventricular outflow tract to left atrium shunt[l] in 3 (20%) patients each, congenital VSD[41,108] and paravalvular leak[50,142] in 2 (13.3%) patients each, and aorta to right atrium shunt[178] in 1 (6.7%) patient.

[a] Refs: 12-16,18,20-22,24,26,28,30,31,35,38,41,42,44-46,48-52,54-61,64,65,67,68,71-73,75-77,79-82,84,85,87,89-91,93-95,98-101,104-113,121-125,127-129,131,133,134,136,137,139-142,145,147,149,151-154,157-161,163,164,167-169,171-175,177,178,182,183,185,186,188,190-192,194,195,197,199,200,202-204,207-210,212,214

[b] Refs: 11,47,62,63,70,74,86,92,97,103,117,118,135, 155,165,176,180,184,187,193,196,205,206,211

[c] Refs: 17,23,29,32,34,36,37,43,53,66,88,115,116,138,143,150,170,181,201,213

[d] Refs: 33,39,40,69,83,120,126,146,148,156,189

[e] Refs: 19,25,78,114,166,179,198

[f] Refs: 4,27,96,102,130,162

[g] Refs: 13,15-18,21,22,26,29-31,33-39,42-50,52-57,59-62,64-66,68-72,74-77,79-85,87,89-91,95,96,99,100,102-107,109-113,115-117,119-123,125-128,131-134,136-140,143-150,152,153,155-160,163,165,167-174,177,178,180,182,185,186,188-194,197,199-201,205,207,208,210,212-214

[h] Refs: 4,11,20,24,28,32,37,41,58,98,108,110,118,124,129,138,141,154,176,183,202,211

[i] Refs: 12,14,67,88,94,101,110,115,164,175,195,203,204

[j] Refs: 133,171,196,197

[k] Refs: 100,150,157

[l] Refs: 42,107,132

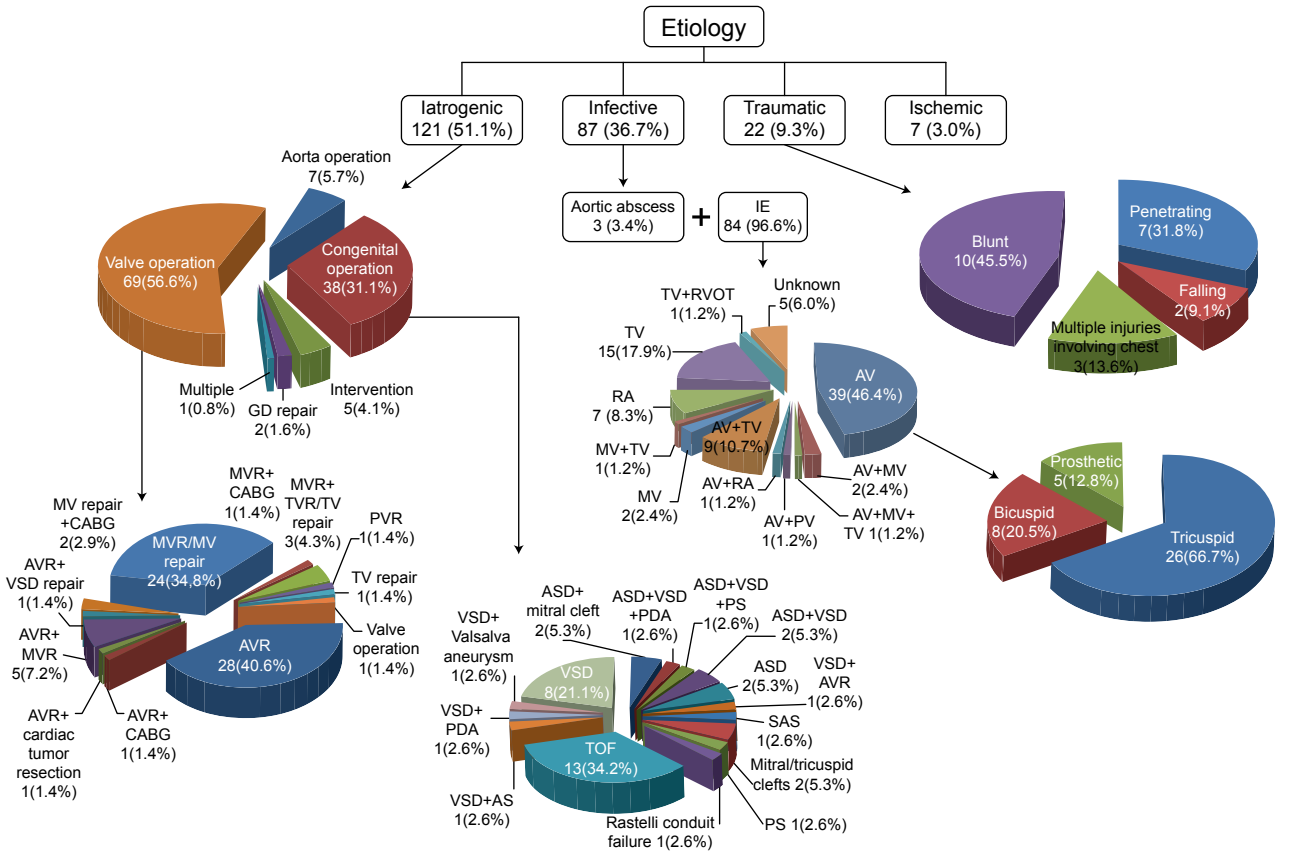

Figure 1. Distribution of predisposing risk factors of the acquired left ventricle-right atrium shunt. AS – aortic stenosis; ASD – atrial septal defect; AV – aortic valve; AVR – aortic valve replacement; CABG – coronary artery bypass grafting; GD – Gerbode defect; IE – infective endocarditis; MV – mitral valve; MVR – mitral valve replacement; PDA – patent *ductus arteriosus*; PS – pulmonary stenosis; PVR – pulmonary valve replacement; RA – right atrium; RVOT – right ventricular outflow tract; SAS – subaortic stenosis; TOF – tetralogy of Fallot; TV – tricuspid valve; TVR – tricuspid valve replacement; VSD – ventricular septal defect.

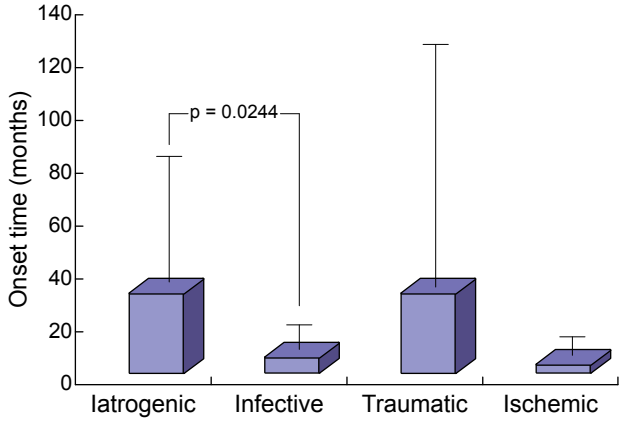

Figure 2. A comparison of the onset time between the four etiological subtypes of shunt.

Of the 121 patients with an iatrogenic shunt due to a previous cardiac operation or intervention, 102 (84.3%) were primary operations, 15 (12.4%) were redo operations and 4 (3.3%) were second redo operations (χ^2=214.4, p=0.0000). Aortic and mitral valve replacements were the two most common cardiac procedures responsible for the development of the acquired shunts (Figure 1). The multiple cardiac surgical procedures included myomectomy, coronary artery bypass grafting and mitral valve replacement,[26] while the interventional procedures were radiofrequency catheter ablation of the atrioventricular node in 4 patients[36,114,163] and endomyocardial biopsy after orthopedic heart transplantation in 1 patient.[99]

There were a total of 99 episodes of IE in 94 patients, with 94 (94.9%) primary and 5 (5.1%) recurrent IE episodes, 83 (83.8%) of which were a direct cause, 12 (12.1%) were an indirect cause, and 4 (4.0%) were not a cause of an acquired LV-RA shunt (χ^2=171.9, p=0.0000; Figure 1). Aortic valve IE was the most common among both direct and indirect IE causes. Tricuspid IE was more common among direct IE causes. Of all the IE cases, the underlying pathogens were indicated in 64 patients, includ-

Table 2. The underlying pathogens of infective endocarditis.

Pathogen	n (%)
Staphylococcus	23 (33.8)
aureus	19 (82.6)
methicillin-sensitive	4 (21.1)
methicillin-resistant	1 (5.3)
unknown	14 (73.7)
epidermidis	2 (8.7)
lugdunensis	1 (4.3)
capitis	1 (4.3)
Streptococcus	33 (48.5)
viridans	12 (36.4)
α-hemolytic	6 (18.2)
mitis	3 (9.1)
mutans	2 (6.1)
pneumoniae	2 (6.1)
agalactiae	1 (3.0)
anginosus	1 (3.0)
sanguis	1 (3.0)
Unknown	5 (15.6)
Others	10 (14.7)
Pseudomonas aeruginosa	3 (30)
Morganella morganii	2 (20)
Cardiobacteria hominis	1 (10)
Gemella morbillorum	1 (10)
Neisseria elongata	1 (10)
Salmonella, non-typhoid	1 (10)
Gram-positive cocci (microscopy)	1 (10)
Mixed infections	2 (2.9)
Staphylococcus aureus + Streptococcus viridans	1 (50)
Staphylococcus aureus + Enterococci	1 (50)

ing 2 cases of recurrent IE (one of them had a second recurrence), 2 combined infections, and 3 multiple specimen investigations, amounting to 68 identified pathogens (Table 2). The specimens for pathogen cultures were described in 38 (52.8%) cases, including 30 (78.9%) blood, 4 (13.3%) resected vegetations or valve tissues, 2 (6.7%) catheter tips, 1 (3.3%) urine, and 1 (3.3%) sternal wound sample.

The clinical manifestations of this patient setting are shown in Table 3. The definite diagnosis of LV-RA shunt was described in 164 (70.1%) patients. There was failure to diagnose by medical imaging before operation or before death in 17 patients, whose diagnosis was established by surgical exploration in 11 (64.7%) patients and by autopsy in 6 (35.3%). All other patients were diagnosed with the aid of medical imaging, in particular transthoracic and/or transesophageal echocardiography. In 19 (8.1%) patients, a high jet flow was detected in the right atrium by transthoracic (n=16) or transesophageal echocardiography (n=1), or by cardiac catheterization (n=2), but the flow origin was uncertain (χ^2=33.3, p=0.000). The diagnosis of LV-RA shunt was missed in 22 (9.4%) patients by transthoracic (n=17) or transesophageal (n=4) echocardiography, or by cardiac catheterization (n=1) (χ^2=29.6, p=0.000). A misdiagnosis was made in 17 (7.3%) patients, by transthoracic and transesophageal echocardiography in 14 (82.4%) and 3 (17.6%) patients, respectively. They were misdiagnosed as ruptured sinus of Valsalva aneurysm into the right atrium (n=5), VSD (n=3), paravalvular leak (n=3), aorta to right atrium shunt (n=2), tricuspid insufficiency (n=2), or tricuspid insufficiency with pulmonary hypertension (n=2).

The shunt size was reported in 104 patients, with 15 (14.4%) of them being described as "small", "restrictive", or "insignificant". The VSD size of the patients with a traumatic LV-RA shunt was larger than that of the patients with an iatrogenic or an infective LV-RA shunt. No differences were noted in the measurements between the iatrogenic and infective groups in terms of total pulmonary blood flow to total systemic blood flow ratio (Qp/Qs), peak velocity, pressure gradient across the VSD, or pulmonary artery pressure (Table 3). There were 52 patients with 55 Qp/Qs measurement results: 10 (18.2%) were <1.5 and 45 (81.8%) were ≥1.5 (χ^2=44.5, p=0.0000). Moreover, a significant difference was found between the patients with a Qp/Qs <1.5 and those with a Qp/Qs ≥1.5 (1.3 ± 0.1 vs. 2.3 ± 0.7, p=0.0001).

Interventional treatment of the LV-RA shunt was only noted in the iatrogenic group patients. Surgical treatment was applied more in the infective shunt than in other subtypes (χ^2=84.4, p=0.0000; Table 4). Among the 40 patients who received no intervention for the shunts, 7 (17.5%) were asymptomatic because of an insignificant shunt; 5 (12.5%) were advised to observe a close follow up; 14 (35%) were treated conservatively, including antibiotic therapies (one of them developed IE later and then required an operation); 1 (2.5%) was in a deteriorated condition and operation was not indicated; 2 (5%) patients declined an operation; and 11 (27.5%) patients lost the chance of a surgical operation because of sudden death. The patients were on a follow up of 13.2 ± 16.0 months (range, 0.5-96; median, 3) (n=51). Of the surgically treated patients, one patient had once-recurrent[160] and another had twice-recurrent LV-RA shunts.[153] The former was managed conservatively and the latter had to undergo two more surgical procedures. Both eventually did well. The overall morbidity and mortality were 12.0% (28/234) and 12.4% (29/234), respectively. Clinical outcomes did not re-

Table 3. Clinical features of Gerbode defect.

Variable	All	Iatrogenic	Infective	Traumatic	Ischemic	Iatrogenic vs. infective	
						χ^2	p-value
Clinical manifestation							
Asymptomatic	11	11 (100)				22	0.0000
Symptomatic	115	42 (37.8)	57 (51.4)	8 (7.2)	4 (3.6)	4	0.0463
Chest pain	7	3 (42.9)		2 (28.6)	2 (28.6)	6	0.0143
Dyspnea	41	27 (65.9)	9 (22.0)	4 (9.8)	1 (2.4)	16.1	0.0000
Effort intolerance	4	3 (75)		1 (25)		6	0.0143
Fever	39	3 (7.7)	35 (89.7)	1 (2.6)		50.6	0.0000
Fever, dyspnea	9		9 (100)			18	0.0000
Palpitation, dyspnea	3	1 (33.3)	2 (66.7)			0.7	0.4142
Palpitation	1				1 (100)		
Abdominal pain	2	2 (100)				4	0.0455
Hemodynamic compromise	6	2 (33.3)	3 (50)		1 (16.7)	0.4	0.5271
Edema	4	3 (75)	1 (25)			2	0.1573
Heart murmur	123	54 (43.9)	48 (39.0)	15 (12.2)	6 (4.9)	0.7	0.4008
Systolic	89	43 (48.3)	26 (29.2)	14 (15.7)	6 (6.7)	7.4	0.0062
Diastolic	3		3 (100)			6	0.0143
Systolic and diastolic	23	9 (39.1)	14 (60.9)			1.4	0.2380
Systolic+diastolic+continuous	1			1 (100)			
Continuous	1		1 (100)			2	0.1572
Heart murmur phase not stated	6	2 (33.3)	4 (66.7)			1.3	0.2482
No heart murmur	2	1 (50)	1 (50)			0	1.0000
Associated disorder							
Bicuspid aortic valve	23	6 (26.1)	17 (73.9)			8.7	0.0028
Heart failure	61	30 (49.2)	23 (37.7)	6 (9.8)	2 (3.3)	1.4	0.2437
Membranous/valve aneurysm	20	4 (20)	13 (65)	3 (15)		7.5	0.0053
Cardiac chamber dilation	113	61 (54.0)	28 (24.8)	23 (20.4)	1 (0.9)	24.5	0.0000
LA	15	9 (60)	4 (26.7)	2 (13.3)		2.5	0.1152
LV	19	6 (31.6)	10 (52.6)	3 (15.8)		1.1	0.2890
RA	48	29 (60.4)	9 (18.8)	9 (18.8)	1 (2.1)	19	0.0000
RV	31	17 (54.8)	5 (16.1)	9 (29.0)		11	0.0007
IVC dilation	7	6 (85.7)	1 (14.3)			7.1	0.0075
HV dilation	2	1 (50)	1 (50)			0	1.0000
PA dilation	3	2 (66.7)		1 (33.3)		4	0.0455
Pulmonary hypertension	19	8 (42.1)	6 (31.6)	3 (15.8)	2 (10.5)	0.1	0.7064
Valve/prosthesis dehiscence	12	7 (58.3)	3 (25)	2 (16.7)		1.8	0.1797
Aortic/mitral leaflet destruction	26	3 (11.5)	22 (84.6)	1 (3.8)		25.9	0.0000
Valve insufficiency (paravalvular leak)	84	31 (36.9)	47 (56.0)	5 (6.0)	1 (1.2)	5.8	0.0160
AR/AR+MR+TR	38	6 (15.8)	32 (84.2)			32.9	0.0000
MR	15	8 (53.3)	5 (33.3)	1 (6.7)	1 (6.7)	0.6	0.4338
TR	31	17 (54.8)	10 (32.2)	4 (12.9)		2.7	0.1016
Aortic/mitral annular/wall abscess	18		18 (100)			32.1	0.0000
Hemolysis	6	4 (66.7)	2 (33.3)			1.3	0.2482
Atrioventricular block	36	12 (33.3)	15 (41.7)	8 (22.2)	1 (2.8)	0.3	0.5867
First-degree	13	5 (38.5)	7 (53.8)*	1 (7.7)*		0.7	0.4142
Second-degree	2		2 (100)				
Third-degree	21	6 (28.6)	7 (33.3)†	7 (33.3)	1 (4.8)	0.2	0.6949

Echocardiographic measurement						
VSD size (mm)	8.6 ± 5.1 (range, 1-30; median, 7) (n=89)	7.5 ± 4.2 (range, 2-22; median, 6) (n=43)	8.3 ± 4.4 (range, 1-18; median, 7.5) (n=30)	12.2 ± 7.9 (range, 1.7-30; median, 10) (n=12)	11.3 ± 4.3 (range, 7-15; median, 11.5) (n=4)	0.4104 (iatrogenic vs. infective); 0.0065 (iatrogenic vs. traumatic); 0.0456 (infective vs. traumatic)
Total pulmonary blood flow to total systemic blood flow ratio	2.1 ± 0.8 (range, 1.2-5; median, 2) (n=55)	2.0 ± 0.7 (range, 1.2-5; median, 2) (n=26)	1.9 ± 0.6 (range, 1.2-3; median, 1.7) (n=15)	2.4 ± 0.8 (range, 1.8-4; median, 2) (n=7)	2.7 ± 1.0 (range, 1.7-4; median, 2.5) (n=7)	0.5472 (iatrogenic vs. infective); 0.1453 (iatrogenic vs. traumatic); 0.2898 (infective vs. traumatic)
Pressure gradient (mmHg)	103.1 ± 31.2 (range, 60-144; median 116) (n=7)	109.2 ± 30.1 (range, 70-144; median 102.5) (n=4)	121.5 ± 7.8 (range, 116-127; median, 121.5) (n=2)	60 (n=1)		NS
Peak velocity (cm/s)	4.8 ± 0.8 (range, 2.5-6; median, 4.7) (n=25)	4.8 ± 0.9 (range, 2.5-6; median, 4.7) (n=15)	4.8 ± 0.7 (range, 4-5.7; median, 5) (n=6)	4.6 ± 0.6 (range, 4.1-5; median, 4.6) (n=2)	4.5 (n=1)	NS
SPAP (mmHg)	58.4 ± 19.8 (range, 20-90; median, 60) (n=15)	63.8 ± 18.7 (range, 44-87; median, 70) (n=7)	55.7 ± 25.5 (range, 20-90; median, 57) (n=6)	52.5 ± 10.6 (range, 45-60; median, 52.5) (n=2)	48 (n=1)	NS

*First-degree heart block aggravated into third-degree. †Third-degree heart block alleviated into first-degree. AR – aortic regurgitation; HV – hepatic vein; IVC – inferior *vena cava*; LA – left atrium; LV – left ventricle; MR – mitral regurgitation; NS – non-significant; PA – pulmonary artery; RA – right atrium; RV – right ventricle; SPAP – systolic pulmonary artery pressure; TR – tricuspid regurgitation; VSD – ventricular septal defect.

veal any significant differences between the four etiological subtypes of shunts; however, the surgically managed patients showed the highest survival, the interventionally treated patients had the highest post-interventional complications, and the no-intervention patients had the highest mortality rate (Tables 5 & 6). Nine patients in the interventional group had 13 complications, including a residual shunt in 9, heart block in 3, and hemolysis in 1 patient. The surgically treated patients were associated with more frequent postoperative heart block and the interventionally managed patients showed more residual shunts (Table 7).

Discussion

It is well-known that any factors leading to ischemia, necrosis, weakening or injury of the septum may predispose to the formation of an acquired LV-RA shunt. Currently, acquired LV-RA shunts have been increasingly reported as a result of the increasing underlying etiologies, including cardiac surgical/interventional maneuvers, infections such as subannular abscess or IE, chest trauma, or acute myocardial infarction.[215] These factors are shown in detail in Table 8.

However, some authors proposed that blunt trau-

Table 4. Management strategies of four etiological subtypes of shunts.

Management	All	Iatrogenic	Infective	Traumatic	Ischemic
Intervention	24 (10.3)	24 (100)			
Amplatzer duct occluder		8 (33.3)			
Amplatzer membranous VSD occluder		1 (4.2)			
Amplatzer muscular VSD occluder		4 (16.7)			
Amplatzer septal occluder		3 (12.5)			
Amplatzer VSD occluder		1 (4.2)			
ASD occluder		1 (4.2)			
Amplatzer device, unspecified		4 (16.7)			
Vascular plug		1 (4.2)			
Unknown		1 (4.2)			
Surgical operation	139 (59.4)	48 (34.5)	65 (46.8)	22 (15.8)	4 (2.9)
Patch repair	52 (37.4)	9 (17.3)	30 (57.7)	12 (23.1)	1 (1.9)
Pledgeted suture	24 (17.3)	13 (54.2)	10 (41.7)		1 (4.2)
Direct suture	40 (28.8)	17 (42.5)	14 (35)	7 (17.5)	2 (5)
Intraoperative device	1 (0.7)	1 (100)			
Surgical technique, unspecified	22 (15.8)	8 (36.4)	11 (50)	3 (13.6)	
No intervention	40 (17.1)	19 (47.5)	18 (45)	2 (5)	1 (2.5)
Self-cured	2 (0.9)	1 (50)	1 (50)		
Unknown	29 (12.4)	23 (79.3)	5 (2.7)		1 (0.9)

ASD – atrial septal defect; VSD – ventricular septal defect.

Table 5. Comparison of clinical outcomes among four etiological subtypes of shunt.

Etiology	Survived	Complicated	Recurrent	Died
Iatrogenic	65 (71.4)	17 (18.7)		9 (9.9)
Infective	53 (67.9)	8 (10.3)	2 (2.6)	15 (19.2)
Traumatic	15 (75)	2 (10)		3 (15)
Ischemic	3 (42.9)	1 (14.3)		3 (42.9)
χ^2	6.6	3.0		6.6
p-value	0.0850	0.3930		0.0850

ma of the chest might lead to the enlargement of a congenital LV-RA shunt,[59] and this cast doubt upon the nature of an acquired LV-RA shunt subsequent to chest trauma. The LV-RA shunt was also noted to be adjacent to the VSD patch, and the incidence of the acquired shunt was 1.7% in patients who had undergone repair of tetralogy of Fallot.[206] The latency for the formation of an infective LV-RA shunt was within a very short period of time, as estimated by Wada et al.[193]

The classification of the etiologies of LV-RA shunts is still under debate. Cheema et al[216] defined an LV-RA shunt in a 31-year-old male patient with a history of two VSD repairs at 4 weeks and 4 years old to be of congenital origin. However, Taskesen et al[8] categorized this case as an acquired shunt type in their recent systematic review. In the present article, this patient was not included in the list of acquired shunts according to authors.

Transthoracic echocardiography, despite being the gold standard for the diagnosis of intracardiac

Table 6. Clinical outcomes of patients under different management strategies and a comparison between the first three subgroups.

Management	Survived	Complicated	Recurrent	Died
Surgical	103 (76.3)	16 (11.9)	2 (1.5)	14 (10.4)
Interventional	19 (63.3)	10 (33.3)		1 (3.3)
No intervention	15 (50)	2 (6.7)		13 (43.3)
Self-cured	2 (100)			
Treatment unspecified				1 (100)
χ^2	21.4	14.1		21.4
p-value	0.0000	0.0010		0.0000

Table 7. Complications in different management groups, n (%).

Management	Interventional	Surgical	No intervention	Interventional vs. surgical	
				χ^2	p-value
Heart block	3 (23.1)	12 (75)	1 (50)	5.7	0.0170
Residual shunt	9 (69.2)			16.1	0.0000
Infective endocarditis		1 (6.3)	1 (50)		
Hemolysis	1 (7.7)				
Respiratory insufficiency		1 (6.3)			
Atrial fibrillation		1 (6.3)			
Unknown		1 (6.3)			

Table 8. Underlying etiologies leading to an acquired left ventricle-right atrium shunt.

Etiologies
Iatrogenic
Direct surgical/interventional trauma
• Extended decalcification of the annulus in the posteromedial commissure or its vicinity during valve replacements[24,61]
• Aggressive debridement of the calcific aortic or mitral valve annuli[14]
• A tear of the tricuspid annulus at the membranous septum resulting in injury of the membranous septum[24]
• Inadvertent damage to cardiac structure during surgical maneuver at the level of the valve annulus[66]
• Suture line of septum involvement[14]
• Incidental trauma by percutaneous intervention[36,99,114,163]
Postoperative strain
• Erosion by the rigid prosthetic ring in postoperative patients[50,210]
• Blood flow impacting strongly on tricuspid annuloplasty ring[211]
Infective
• Vegetation or abscess invasions of the membranous septum[67,170]
Chest trauma
• Blunt
• Penetrating
Acute myocardial infarction

shunts, has shown notable deficiencies in the delineation of LV-RA shunts, in comparison with transesophageal echocardiography and other imaging modalities.[215] With pulsed Doppler echocardiography, an LV-RA shunt can be visualized.[190] However, the high-velocity LV-RA jet may be misdiagnosed as tricuspid regurgitation with pulmonary hypertension.[71,148] Zhang et al[217] praised the important role of real-time three-dimensional transesophageal echocardiography in the percutaneous closure of multiple *secundum* atrial septal defects. To further define shunt anatomy and to quantify the shunt ratio, cardiac magnetic resonance imaging was often performed.[126] Nevertheless, magnetic resonance imaging angiography and transesophageal echocardiography may occasionally lead to a false positive LV-RA shunt.[218] In brief, transesophageal echocardiography was the most sensitive diagnostic technique, with less diagnostic bias in the diagnosis of this lesion.[215]

The cardiac geometric/structural lesions associated with acquired LV-RA shunt are predominantly right atrial dilation.[215] A small restrictive defect was defined when the Qp:Qs ratio was <1.5:1, and the cardiac chambers and the pulmonary vasculature were usually normal. Larger shunts with a Qp:Qs >2.0:1 could be associated with volume overload of the left heart and elevated right ventricular and pulmonary arterial pressures.[186] Patients with an acquired LV-RA shunt are often associated with elevated right atrial (14 mmHg) and pulmonary (52/22 mmHg) pressures and a higher Qp:Qs (2.4:1).[79] This study revealed that more patients with an acquired shunt had larger Qp:Qs ratios, indicating an increased shunt flow via the acquired defect. Moreover, patients with an acquired LV-RA shunt who received interventional therapy, with the use of Amplatzer occluders to close the defect, showed satisfactory results.

Acquired LV-RA shunts, especially the infective and iatrogenic subtypes, are often associated with a series of comorbidities, including congestive heart failure, valvular leaflet perforation, subannular abscess, and complete heart block.[215] Hemodynamic de-

terioration, a new LV-RA shunt and valvular destruction with ring abscess might be noted.[154] Therefore, in most of the patients with an LV-RA shunt surgical correction is warranted. Yacoub et al[219] proposed that all LV-RA defects should be repaired, regardless of their size, to prevent the risk of IE, in light of the small chance of spontaneous closure and the very low morbidity and mortality. Toprak et al[185] proposed that patients with an insignificant LV-RA shunt with no symptoms and no right ventricular volume or pressure overload can be kept under close follow up, rather than undergoing surgical closure. Moreover, patch repair of an LV-RA shunt should be performed on the right atrial side in order to prevent postoperative heart block.[182] Two-side patch repair of the LV-RA shunt was reported by Inoue et al[87] and by Matt et al,[119] and both patients had a good surgical outcome. An oversized patch, larger than the shunt, was advocated in order to avoid placement of the stitches proximal to the conduction system.[87] However, Elliott et al[220] stated that LV-RA shunt repair might result in tricuspid valve malformations. Spontaneous closure of the LV-RA shunt has been confirmed by surgical exploration[83] and by postoperative echocardiographic examination.[102] It was stated that spontaneous closures are less likely in type I LV-RA shunt but more in acquired than in congenital shunts.[79]

Conclusions

Acquired LV-RA shunt is increasingly being reported. Patients who have an insignificant shunt, with no clinical symptoms or associated circulatory overload, can be kept under close follow up, whereas in those with a larger shunting flow, surgical or interventional therapy is warranted. Surgical trauma and infectious involvement of the septal membrane are the underlying mechanisms leading to the development of acquired LV-RA shunts. Hence, careful surgical maneuvers in primary heart surgery and effective prophylaxis of IE in this high-risk patient population would be good measures for preventing LV-RA shunt development.

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