Report on experience in eight patients

The Konno procedure to treat prosthesis-patient mismatch and complex left ventricular outflow tract obstruction in adult patients

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Summary

BACKGROUND: The most common forms of congenital and acquired obstruction of the left ventricular outflow tract (LVOT) are valvular aortic stenosis, congenital tunnel-like obstruction, hypertrophic obstructive cardiomyopathy and, finally, the prosthesis-patient mismatch (PPM) following aortic valve replacement. The last is most probably underestimated and undertreated. Here, we report our experience with the Konno anterior aorto-ventriculoplasty in adult patients suffering from prothesis-patient mismatch.

PATIENTS: Between 2005 and 2020, 8 adult patients received a classical Konno anterior enlargement of the aortic anulus to treat a typical PPM (n = 5) or another complex obstructing pathology of the LVOT (n = 3). Median age was 42,5 years and 5 patients were female. All patients suffered from exercise dyspnea NYHA functional class III or IV. In addition to the Konno procedure, 3 patients underwent mitral valve replacement (n = 2) or repair (n = 1). Two patients received tricuspid anuloplasty to treat severe concomitant tricuspid regurgitation. All patients had undergone at least one previous operation, 3 patients had 2 prior procedures in their medical history and 1 patient was operated for the fifth time.

RESULTS: All patients survived the operation. One younger patient suffering from Hutchinson' type of progeria died in-hospital on postoperative day 45 following prolonged hemodynamic failure. She also had developed thrombosis of the mechanical mitral prosthesis, underwent thrombectomy and extracorporeal membrane oxygenation support (ECMO) but did not recover. One patient required a double chamber pacemaker implantation because of persistent atrio-ventricular block III. All surviving patients demonstrated very satisfactory postoperative hemodynamic patterns across the LVOT and clinical improvement.

The Konno anterior aorto-ventriculoplasty is a very efficient method to adequately enlarge the LVOT, independently from the initial pathology. This technique is only rarely used because it is considered as a complex intervention. However, this type of enlargement usually allows the insertion of a larger prosthesis than after a simpler Manougian or Nicks posterior anulus enlargement. For adult patients with severe PPM or other forms of complex LVOT obstruction, the Konno intervention is a valuable option that should be carefully discussed.

Introduction

Obstruction of the LVOT can occur as a simple pathology (valvular aortic stenosis) or as a more complex disease, like hypertrophic obstructive cardiomyopathy, membranous subaortic stenosis, tunnel-like subaortic stenosis and finally as an obstruction related to atrioventricular septal defects. These more complex diseases develop frequently independently from aortic valve pathology but they may be occasionally combined with valvular aortic stenosis and/or regurgitation.

A very small subset of patients presents with functional obstruction due to a too small valvular prothesis (a mechanical or a tissue valve) implanted to replace a defect native aortic valve. One of the reasons for this phenomenon which is called PPM may be patient outgrowth when valvular replacement was performed at younger age, excessive increase of body weight occurring late after valve replacement or simply the implantation of a too small valve at the initial operation.

PPM is present when the effective orifice area (EOA) of the inserted prosthetic valve is too small in relation to body size [1, 2]. The main hemodynamic consequence is that higher than expected gradients are generated through the normally functioning prosthetic valve. PPM is not an uncommon finding following surgical aortic valve replacement (SAVR), as it is described in up to 20–30% of the cases in series following surgical but also transcatheter aortic valve replacement (TAVR) [1–7]. PPM means worse hemodynamic function because of a higher afterload for the left ventricle. This may have adverse impact on the outcome: less regression of left ventricular hypertrophy, more adverse cardiac events, and lower survival during long-term follow-up [8–12].

As opposed to the majority of risk factors that may lead to adverse outcome following SAVR or TAVR, PPM can be prevented by using a wise strategy at the time of a

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surgical intervention. Aortic root enlargement is an established procedure to overcome a small aortic anulus at initial surgery. In certain patients (children and adolescents with an expected important outgrowth as well as adult patients with a very narrowed native aortic anulus and finally those suffering from massive obesity), placing an adequately sized prosthesis to prevent PPM is of paramount importance. Aortic root enlargement adds some technical complexity to a simple SAVR but should be carefully evaluated on the basis of preoperative calculations and intraoperative inspection of the aortic root.

We present our institutional experience with a small series of adult patients who received the most efficient but also complex procedure (the anterior Konno aorto-ventriculoplasty) to overcome either a PPM situation following SAVR or to treat another form of complex stenosis of the LVOT.

Patients and methods

Between February 2004 and July 2020, we performed an anterior aorto-ventriculoplasty operation after Konno in 8 adult patients (3 males and 5 females). We excluded younger patients (<20 yrs) who received either a modified Konno or a Ross-Konno procedure at the neonatal age or in the childhood during the same period.

The median age was 42,5 years (from 20 to 66 years) and the body weight ranged from 38 kg to 105 kg (median 75 kg). Patients data were analyzed retrospectively from hospital records and from the clinic-specific cardiac database. Data included preoperative diagnosis with a summary of all investigations, the operative notes, the post-operative course and the follow-up examinations. The main patients' characteristics are described for each patient and summarized in **table 1**. All patients underwent 1 to 4 previous operations and they were all in NYHA functional class III or IV regarding symptoms of dyspnea.

Case Age (yrs) Basic clinical problem Previous operation Interval since last surgery Intraoperative particularities 1 55 PPM aortic prosthesis 1 6 yrs AVR 21 mm SJM replaced through 24 mm ATS dp mean 53 mmHg – AVR mech, MVR, TVR TVRep Physio Ring 30 mm severe TR2 28 PPM aortic prosthesis 4 21 mths AVR 21 mm SJM replaced through 25 mm SJM dp 43/68 mm Hg – Commissurotomy (in prior tube valve conduit) – Homograft miniroot – AVR in homograft – Composite mechanical3 26 PPM aortic prosthesis 1 5 yrs AVR 18 mm ATS replaced through 26 mm ATS dp mean 35–40 mm Hg – AVR mech (revision because of residual VSD at one week)4 66 PPM aortic prosthesis 1 27 yrs AVR 19 mm SJM replaced through 22 mm ATS severe MR/MS and TR – AVR mech MVR 23 mm SJM dp mean 40 mm Hg TVRrep Physio Ring 28 mm5 61 Severe AV / MV stenosis

Table 1A: Clinical problem, previous surgery, interval since previous surgery and intraoperative particularities.

Case	Age (yrs)	Basic clinical problem	Previous operation	Interval since last surgery	Intraoperative particularities
1	55	PPM aortic prosthesis dp mean 53 mmHg severe TR	1- AVR mech, MVR, TVR	6 yrs	AVR 21 mm SJM replaced through 24 mm ATSTVRep Physio Ring 30 mm
2	28	PPM aortic prosthesis dp 43/68 mm Hg	4CommissurotomyHomograft minirootAVR in homograftComposite mechanical	21 mths	21 mm SJM replaced through 25 mm SJM (in prior tube valve conduit)
3	26	PPM aortic prosthesis dp mean 35–40 mm Hg	1AVR mech	5 yrs	AVR 18 mm ATS replaced through 26 mm ATS (revision because of residual VSD at one week)
4	66	PPM aortic prosthesis severe MR/MS and TR dp mean 40 mm Hg	1- AVR mech	27 yrs	AVR 19 mm SJM replaced through 22 mm ATSMVR 23 mm SJMTVRrep Physio Ring 28 mm
5	61	Severe AV / MV stenosis Hypoplastic Ao anulus	1- VSD patch closure	55 yrs	AVR 21 mm SJMMVR 27 mm SJM
6	20	PPM aortic prosthesis dp 40/65 mm Hg	2- ballon valvuloplasty-AVR mech + Nick enlargement	7 yrs	AVR 16 mm ATS replaced through 22 mm ATS
7	33	Degeneration bio valve dp 70/37 mm Hg	1- AVR bio	7 yrs	AVR 19 mm Edwards replaced through 18 mm ATSMVR 23 mm ATS
8	51	VSD and endocarditis	1- AVR and MR repair	3 mths	AVR 25 mm Edwards replaced through 27 mm EdwardsMVrep Physio Ring 32 mmVSD closure together with Konno patch

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Table 1B: Additional intraoperative parameters, ICU and follow-up.

Case	Age (yrs)	ECC duration (min)	Ischemic time (min)	ICU duration	Follow-up	Symptoms
1	55	150	124	3 d	5 yrs	NYHA I (BMI 41)
2	28	135	112	1 d	12 mths	NYHA I-II
3	26	105	98	3 d	17 yrs	NYHA I
4	66	306	216	4 d	12 mths	NYHA I (BMI 30)
5	61	354	198	2 d	24 mths	NYHA I
6	20	144	118	1 d	12 mths	NYHA I
7	33	265	148	34 d		ECMO – mitral valve thrombosis
8	51	326	219	4 d	13 yrs	NYHA I, then lost to follow-up

PPM: patient prosthetic mismatch; SJM: St-Jude Medical; AVR aortic valve replacement; ATS: Medtronic ATS 360 AP; MVR: mitral valve replacement; Edwards Perimount Magna tissue valve; MV rep: mitral valve repair; NYHA New York Heart Association; TV rep: tricuspid valve repair; ECMO: extra-corporeal membrane oxygenation; VSD: ventricular septal defect; d: days; dp: pressure gradient; mths: months; mech: mechanical prosthesis, yrs years; bio biological prosthesis

1 55 yrs AVR 21 mm SJM Hypoplastic Ao annulus - VSD patch closure MVR 27 mm SJM6 20 PPM aortic prosthesis 2 7 yrs AVR 16 mm ATS replaced through 22 mm ATS dp 40/65 mm Hg – ballon valvuloplasty – AVR mech + Nick enlargement7 33 Degeneration bio valve 1 7 yrs AVR 19 mm Edwards replaced through 18 mm ATS dp 70/37 mm Hg – AVR bio MVR 23 mm ATS8 51 VSD and endocarditis 1 3 mths AVR 25 mm Edwards replaced through 27 mm Edwards – AVR and MR repair MVrep Physio Ring 32 mm VSD closure together with Konno patch.

The underlying anatomic diagnoses were severe aortic stenosis with bicuspid aortic valve, narrowed LVOT and severe mitral stenosis in one patient, destructive mitral and aortic valve endocarditis with annular abscess and a very small aortic anulus in one patient, while a classical PPM was the indication for surgery in 5 patients. Among them, one patient presented with a salmonella endocarditis following aortic homograft implantation, one had an additional subvalvular LVOT obstruction caused by septal hypertrophy, and one had a significant additional mitral and tricuspid insufficiency.

The operative technique was adapted to the individual pathology. A Konno aorto-ventriculoplasty was performed in 7 patients, while a double enlargement, Manougian combined to a Konno aorto-ventriculoplasty was performed in one patient.

Concomitant surgical procedures included tricuspid valve reconstruction in two patients, xenopericardial patch closure of an aortic root abscess combined to ascending aortic replacement and mitral valve reconstruction in one patient. In one patient enlargement of the RVOT was necessary to treat a subpulmonary stenosis and finally resection of an asymmetric septal hypertrophy was performed in one patient.

Brief description of the patients' history

Patient 1

A 55 yrs old female patient had undergone a triple valve operation (aortic and mitral valve replacement as well as de Vega tricuspid annuloplasty) in 2010 in Jordania because of rheumatic heart disease. She had received a 21 mm mechanical aortic valve and developed a massive adipositas with a body mass index (BMI) of 41 late after surgery. She suffered from dyspnea functional class III-IV and echocardiography demonstrated a mean pressure gradient of 53 mm Hg across the otherwise normally functioning aortic valve prosthesis. In addition, she suffered from permanent atrial fibrillation.

Because of the normally functioning mitral valve prosthesis, we decided to proceed with anterior aorto-ventriculoplasty to avoid posterior enlargement and therefore the region of the aorto-mitral continuity. The Konno procedure was performed using two distinct Biocor® (St-Jude Medical, Abbott Laboratories, Chicago IL, USA) xenopericardial patches, one for the enlargement of the LVOT and the aortic root, the second for the closure of the right ventricular incision. A 24 mm Medtronic (Medtronic, Minneapolis, MN, USA) mechanical prosthesis was implanted. In addition, the tricuspid valve was re-reconstructed using a 30 mm Physio® II annuloplasty ring (Edwards Lifesciences, Irvine, CA). Intraoperative echocardiography demonstrated a mean pressure gradient across the aortic valve of 5 mm Hg after weaning the patient from cardiopulmonary bypass. At follow-up examination 6 months post-operatively, echocardiography (performed at a heart rate of 100/min) showed a good function of the aortic prosthesis with a pressure gradient of 11/21 mm Hg and an aortic valve area of 2.03 cm^2. The maximal RV/RA gradient was 37 mm Hg, corresponding to a moderate pul-

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monary hypertension. At last follow-up visit, the patient was doing fine with a slight dyspnea (NYHA functional class II) while obesity and the persistent deconditioning were considered as possible causes of the residual symptoms.

Patient 2

This 28 yrs old male had received an aortic valve commissurotomy combined to a limited subvalvular resection at the neonatal age because of a critical valvular aortic stenosis. At the age of 5 years, he had undergone replacement of the aortic valve because of severe restenosis through a homograft mini-root with re-implantation of the coronary arteries.
Unfortunately, the homograft valve presented severe degenerative changes very soon after the operation and 2 years later a 23 mm mechanical valve was implanted in the homograft following annular enlargement of the latter using Manougians' technique. Following this 3rd operation, the patient did very well during the next 19 years and was completely asymptomatic until 2018. He then presented with a salmonella prosthetic valve endocarditis and a suspected abscess below the left coronary ostium. Following a 4 weeks antibiotic treatment, the prosthetic valve and the homograft were resected and a composite-graft replacement using a 21 Medtronic mechanical conduit was implanted. The patient recovered well from this 4th operation but he developed exercise-induced angina NYHA functional class II-III 18 months after the procedure. Echocardiography showed an elevated pressure gradient of 43/68 mm Hg at rest across the composite graft, corresponding to a severe prothesis-patient mismatch. Under exercise, the gradient was measured as high as 83/110 mm Hg. Valve cine showed an unrestricted motion of both prosthetic leaflets. Re-operation was found to be indicated and a Konno procedure was performed using a large xenopericardial patch for anterior aorto-ventriculoplasty. This allowed the implantation of a 25 mm Masters Series St-Jude Medical valve. Intraoperative echocardiography showed a pressure gradient of 20/9 over the aortic valve. However, first postoperative controls showed still an elevated maximal gradient of 40 mm Hg probably due to hyperdynamic conditions which resolved with time. At 12 months follow-up the pressure across the valve was still somewhat elevated with a mean of 20–25 mm Hg, the patient was asymptomatic excepted during very strong exercises when he described a rather smooth angina equivalent. The left ventricular hypertrophy regressed from 143 to 136 g/m^2 (LV-mass index).

Patient 3

This 26 yrs-old female had been treated at the age of 21 years at another institution because of a combined aortic valve disease with predominant stenosis. At the initial operation, Manougians' anular enlargement had been performed and a 21 mm Masters Series St-Jude valve was first implanted but the leaflets motion was not completely normal because of subvalvular tissue. During the procedure, the 21 mm St-Jude valve was exchanged against a 18 mm ATS AP 360 (Medtronic-ATS) mechanical valve. A few years later, a paravalvular leak was diagnosed with a clinically significant hemolysis.
The patient complained about exercise-induced dyspnea functional class NYHA II to III and echocardiographic assessment demonstrated a mean pressure gradient of 35 mm Hg across the mechanical valve. Due to persistent symptoms even after beta-blocker treatment, re-operation was considered.
A typical Konno procedure was performed and a 26 mm ATS (called today a Medtronic AP 360 series) mechanical valve was implanted. One week postoperatively, the patient had to be re-operated because of a significant ventricular septal defect at the basis of the left-ventricular septal patch. Most probably the size of the first left-ventricular sided pericardial patch was to small and tension caused a tear in the muscular septum. An additional xenopericardial patch was used to allow tension-free closure of the VSD and the patient recovered well. The intraoperative gradient across the valve was 6/10 mm Hg. The patient recovered well and echocardiography before discharge showed normal biventricular function and a stable pressure gradient across the mechanical prosthesis. At last examination 14 years following the Konno procedure, the patient was doing well, the aortic valve area was 2.23 cm^2 and the gradient 7/11 mm Hg. There was still a small restrictive VSD (dd coronary fistula) with a restrictive gradient of 65 mm Hg. The left ventricular function was normal with an ejection fraction of 55%. The RV/RA gradient was 31 mm Hg.

Patient 4

A 56 yrsold female (BMI 30.5) had been operated at the age of 39 years because of a valvular aortic stenosis. At initial operation, a limited subvalvular resection had been performed and a 19 mm St. Jude Medical prosthetic valve had been implanted. 27 years later, at the age of 66 years, she presented with a severe PPM and a mean gradient of 40 mm Hg across the 19 mm valve. A subvalvular pannus formation could not be completely excluded in the echocardiography. In addition, she suffered from combined mitral valve disease with severe stenosis and moderate insufficiency and from severe tricupidal regurgitation. The patient's weight increased from 50 to 68 kg since the first operation and she suffered from supraventricular arrhythmias and exercise dyspnea NYHA functional class III. A re-operation was scheduled and a triple valve procedure was planned. The mitral valve anulus was extremely calcified and narrowed; a 25 mm mechanical valve was implanted.

For this reason, a Manougian enlargement was not considered since enlargement would have been technically difficult due to the mitral anulus calcifications. Therefore, an anterior Konno aorto-ventriculoplasty was necessary to allow the implantation of a 22 mm Medtronic AP 360 mechanical valve. Finally, tricupidal reconstruction was performed using a 28 mm Physio® anuloplasty ring. The patient recovered well and did not suffer from any symptoms.

At last echocardiography 2 years after the operation, mean gradient across the aortic prosthesis was 8 mm Hg, the left ventricular function was normal (EF 60%) and the gradient across the tricupid valve was estimated at 30 mm Hg, indicating only a mild elevation of pulmonary pressure.

Patient 5

The 61 yrs old female had been first operated at the age of 5 (in 1964), when a ventricular septal defect was closed with a Dacron patch. She did very well until the age of 60; she then developed dyspnea and was very intolerant for mild exercises (functional class NYHA III). ECG showed an atrio-ventricular block II from Wenckebach type. Echocardiographic examination showed a severe aortic valve stenosis with a mean gradient of 42 mm Hg and a severe mitral valve stenosis with a mean gradient of 10 mm Hg. The left ventricular ejection fraction was 33%. Mitral valve was excessively calcified as well as the mitral annulus. A double valve replacement was decided. Following resection of the leaflets and decalcification of the annulus, a 27 mm St-Jude Medical mechanical valve was inserted. After resection of the native aortic valve and decalcification of the annulus, a sizer of only 17 mm could be introduced through the aortic annulus. Since a Manougian enlargement would have had only a limited effect, a Konno aorto-ventriculoplasty using a large xenopericardial patch was performed and a 21 mm St-Jude Medical valve was implanted. Intraoperative echocardiography showed a mean/maximal gradient of 7/12 across the aortic valve prosthesis. The patient recovered well; however, because of an atrio-ventricular block III, a DDD-pacemaker was implanted on postoperative day 5. The further recovery was excellent. At the last follow-up 18 months after the operation, the patient was doing well without any symptoms at moderate exercises. Echocardiography showed a slightly improved left ventricular function (EF 40–45%) and the gradient across the aortic prosthesis was estimated at 5/8 mm Hg. The mean diastolic gradient across the mitral valve was 5 mm Hg.

Patient 6

The 20 yrs old male had undergone a balloon valvuloplasty in the neonatal period because of a critical congenital aortic stenosis. At the age of 13 years, he had received an aortic valve replacement with a 16 mm ATS (labelled today Medtronic AP 360) mechanical prosthesis after Nicks' anulus enlargement. Due to somatic growth, he developed a severe PPM with a mean gradient of 40–50 mm Hg but presented also a subvalvular obstruction due to hypertrophic septal musculature. The previously implanted 16 mm mechanical valve was excised, a subvalvular resection performed and a Konno procedure was done with a patch enlargement of the LVOT. A Medtronic mechanical aortic valve 22 mm could be implanted. The intraoperative mean gradient after weaning from cardiopulmonary bypass varied between 8–12 mm Hg. The patient was discharged on postoperative day 6 following an absolutely normal recovery. At 6 months follow-up, the physical performance was completely normal and the patient did not complain about any symptoms (functional class NYHA I). The transprothetic gradient was 10/20 mm Hg with a calculated aortic valve area of 4,0 cm^2. LV mass index showed a regression from 110 to 100 g/m^2. Ejection fraction remained stable with 53%.

Patient 7

This 33 yrs old female suffered from progeria syndrome type Hutchinson . At the age of 26 years (body weight 35 kg), she presented with a degenerative aortic stenosis with heavily calcified aortic anulus. A biological valve prosthesis of 19 mm diameter was implanted according to the wish of the patient who was reluctant to take oral anticoagulation. Four years after surgery, she already presented with elevated pressure gradients across the tissue valve. Seven years later, she suffered from dyspnea at rest (functional class IV) and the echocardiography did not only show a severe degeneration of the prosthesis but also a very severly calcified mitral

valve stenosis. Left ventricular function was normal with an ejection fraction of 74% but the NT-pro BNP value was high with 2200 ng/L. Systolic pulmonary pressure was very high with 100 mm Hg. Despite the severe symptoms, the patient and her family were sceptical against a redo-procedure. Following several discussions with the patient who became a MD in the meanwhile, re-operation was decided.
Unfortunately, the patient developed lung edema just before induction of the anaesthesia. Therefore, re-opening of the chest with the oscillating saw had to be quickly performed since a peripheral cannulation was not possible because of the very small size of the iliac vessels. After crossclamping and opening of the aorta, the aortic prosthesis was inspected. The leaflets were very stiff but not that much calcified. The mitral valve was approached through a transseptal incision. The mitral anulus was heavily calcified, the leaflets of the native mitral valve could not be recognized anymore. After resection of calcium amounts inside the anulus (the leaflets), a 23 mm sizer could be passed through the circumferentially calcified atrio-ventricular connection and a 23 mm mechanical Medtronic valve was implanted. Following this step, a sizer of 15 mm could no more be passed through the aortic annulus. A Konno enlargement was necessary to implant an 18 mm Medtronic mechanical aortic prosthesis. The right ventricular incision was closed with a second xenopericardial patch as usual. Because of a severe diastolic dysfunction and severe pulmonary artery hypertension, a veno-arterial (VA)-ECMO was used to stabilize the hemodynamics and could be weaned after 48 hours after some thrombotic material on the mitral valve was removed. Delayed sternal closure was performed on postoperative day 7. The patient required tracheostomy because of a critical illness polyneuropathy but the tracheal cannula could be removed after 3 weeks. During the hospitalization, the patient developed a small subdural hematoma under therapeutical anticoagulation (INR 3,2). At this stage oral anticoagulantion was stopped and heparin at a stable dosage of 20'000 IE started. Under this regimen, the patient unfortunately developed a thrombosis of the mitral valve. Because of the prolonged postoperative course and the overall evaluation of the situation, a surgical re-exploration to deblock the mitral valve was denied and the patient unfortunately died at postoperative day 45.

Patient 8

The last patient was a 49 yrs old male that had received mitral valve reconstruction (xeno-pericardial patch repair of A3) and aortic valve replacement using a 23 mm tissue valve (Perimount Magna®, from Edwards) because of double valve endocarditis caused by haemolytic streptococci. Because of persisting endocarditis and suspicion of abscess formation and ventricular septal defect, a re-exploration was performed 2 months later.
At reoperation, a large paravalvular regurgitation between the left and right aortic leaflets was found together with an abscess of the interventricular septum leading to a small muscular defect at the mid-ventricular level of the septum. The aortic valve prosthesis was excised and the septum was incised according to the Konno septal incision up to the level of the ventricular septal defect (VSD). To allow the insertion of a larger prosthesis, the whole defect was closed with a large xenopericardial patch. The 23 mm aortic tissue valve prosthesis was replaced with a new 27 mm Perimount® tissue valve and the patch used for closure of the aortotomy. The mitral valve was reconstructed with a Physio II anuloplasty ring 32 mm. During weaning from cardiopulmonary bypass, a residual VSD was suspected at the base of the patch. A second pump run was decided and an additional xeno-pericardial patch was inserted to cover the residual defect located at the lowest part of the septal incision. The patient recovered well from this prolonged intervention but suffered from transient renal failure and had to be converted several times because of supraventricular tachycardia.
At first follow-up examination 8 months postoperatively, echocardiography demonstrated a normal left ventricular function (EF 55–60%), a normal functioning aortic valve prosthesis with a transvalvular gradient of 15/22 mm Hg and a calculated aortic valve area of 2,3 cm^2. The left ventricular outflow appeared optically still somewhat narrowed with a diameter of 18 mm but without significant gradient nor turbulences. The patient was lost to follow-up 6 years postoperatively.

Operative technique

The procedure is performed through a median sternotomy. Cardiopulmonary bypass is instituted following aortic and bi-caval cannulation and conducted in mild hypothermia (32–34°C). Myocardial protection is performed with single-dose low volume (100 ml) cristalloid cardioplegia (Cardioplexol®, Bichsel, Interlaken, Switzerland) and blood cardioplegia is repeated at 30–45 minutes intervals thereafter through instillation into the coronary ostia. Alternatively, retrograde cardioplegia may be administred.
If the anticipated annular size would be considered to be too small for an adequate-sized prosthetic valve af-

ter the Manougian procedure, an anterior anular enlargement according to the original Konno-Rastan description is considered the best option.

A longitudinal aortotomy is performed slightly above the level of the sinotubular junction and the aortic root and aortic valve are inspected; this includes a gross evaluation of the size of the aortic annulus. Once the indication to proceed with a Konno aorto-ventriculoplasty is confirmed, the longitudinal incision is prolonged into the aortic root just to the right of the anterior commissure between the left and right aortic leaflets, this means well left of the right coronary artery ostium. Then a small oblique/transverse incision is made into the right ventricular outflow tract, just beneath the pulmonary valve. The pulmonary valve cusps have to be visualized since they may lie very close to the point of entry into the right ventricle (fig. 1).

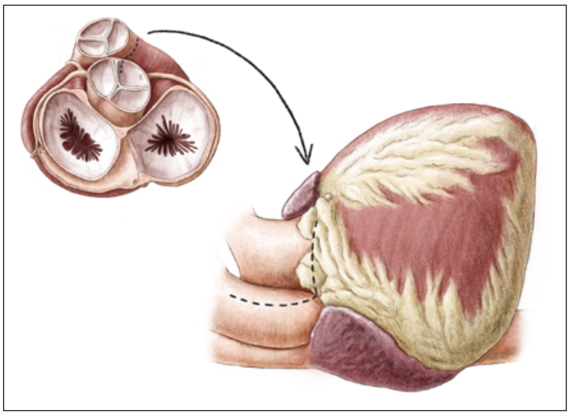

Figure 1: Overview of the anatomical relationship between the four different valves and schematic representation of the opening 1./ of the ascending aorta and 2./ of the right ventricle. At this place, a special care should be directed to the pulmonary valve leaflets, which lies sometimes very close to the incision on the right side.

After the right ventricle has been opened, the previously inserted valve prosthesis is resected free from the aortic annulus. Thereafter, the scissors are positioned with one blade in the left ventricle through the aortotomy and the other blade in the right ventricle through the ventriculotomy. The incision is carried far enough into both ventricles to allow a perfect view into the left ventricle. The interventricular septum is incised deep enough (usually in front of the tip of the papillary muscles to allow a sufficient enlargement) (fig. 2). Hypertrophic septal musculature may be optionally resected. Then a large oval xeno-pericardial patch is sutured to enlarge the septum, beginning at the inferior border of the incision in the ventricular septum (fig. 3). The first suture is placed transmurally through the thick septum from the left to the right. To determine the broadness of the patch, a large enough valve sizer can be used to assess the maximal size of the aortic valve prosthesis to be implanted. The patch is then sewn 1 to 2 cm cranially to the level of the aortic annulus. The newly created and enlarged annulus is sized and an appropriate aortic valve prosthesis is chosen.

Thereafter, the valve is implanted in a typical way with pledgeted sutures placed through the aortic anulus and stitched in a ventriculo-aortic fashion. At the level of the patch (that represents approximatively 1/3 of the circumference), the stitches are placed from outside to inside (fig. 4).

The coronary arteries are usually well aside from the plane of the sewing cuff of the prosthesis. After tying these pledgeted sutures, the patch is used to close the

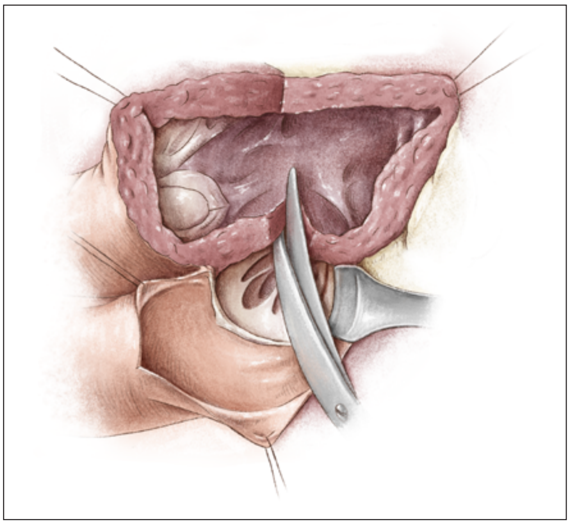

Figure 2: Extension of the incision into the interventricular septum as deep as possible to allow the most complete enlargement of the left-ventricular outflow tract.

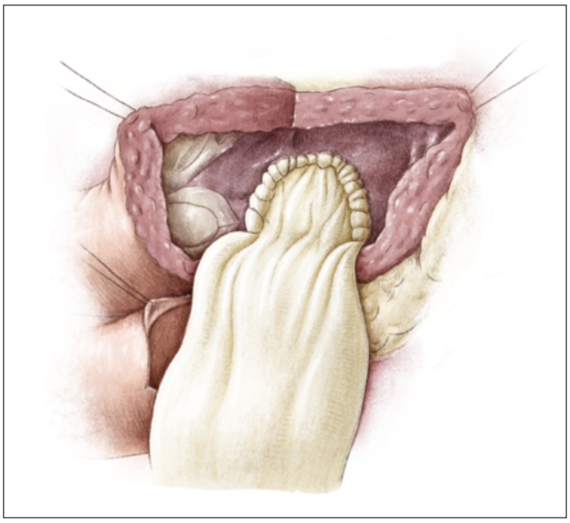

Figure 3: The first xeno-pericardial patch is inserted to enlarge the left-ventricular outflow tract and sutured in a first step up to a plane just above the former aortic anulus.

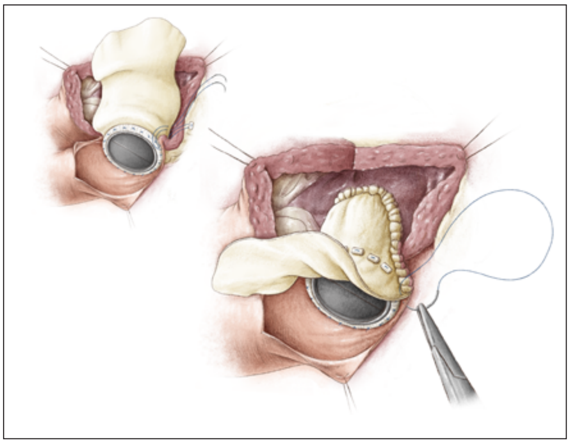

Figure 4: In the meanwhile, the new aortic valve prosthesis (here a two-leaflet mechanical valve) is fixed to the native aortic anulus in the posterior part of the sewing cuff and in the anterior portion to the xeno-pericardial patch (with teflon-felt pledgets on the outside of the patch).

aortotomy; usually it bulges because of the large mechanical prosthesis.

A second patch is then attached at the right ventricular side of the septum and then runs along the free wall of the right ventricle to close the latter. Care is there taken that this second patch is large enough to avoid narrowing of the right ventricular outflow tract (fig. 5). This part of the operation can be at least partially performed on beating heart during reperfusion.

Results

All patients survived the operation but one patient died in-hospital from irreversible cardiogenic schock due to fixed pulmonary hypertension after 7 weeks. This patient, suffering from progeria Hutchinson was supported early postoperatively with a VA-ECMO that could be weaned after 48 hours but she developed several complications (respiratory and renal failure, thrombosis of the mechanical mitral valve). All other patients survived the procedure and the hospitalization and did well: their actual follow-up extend from 14 months to 16 years. All patients were in NYHA functional class I to II, for dyspnea and angina at last examination, excepted one patient who presented with mild dyspnea at moderate to strong exercises (NYHA functional class II). One patient was lost to follow-up after 13 years.

The following sizes of aortic valve were implanted; 7 mechanical valves: 18 mm, 21 mm, 2 × 22 mm, 23 mm, 24 mm, 26 mm and one 27 mm biological prosthesis. The sizes of mitral mechanical valves were 23 and 27 mm. One mitral ring 32 mm and two tricuspid rings (28 and 32 mm) were used. The median duration of cardiopulmonary bypass was 226 min (from 105 to 354 min) and duration of the aortic cross-clamping was 164 min (from 98 to 221 min). Postoperatively, definitive pacemaker implantation was necessary in 2 patients. Mean duration of hospitalization of the 7 patients who were discharged was 11 days (6 to 20 days).

Discussion

The Konno procedure is a very efficient but also complex procedure to enlarge the aortic annulus. It allows aortic surgical valve replacement with an adequate-sized mechanical or biological prosthesis even though in the most narrowed aortic annulus. This operation seems particularly indicated for a small group of patients who had prior aortic valve surgery (with or without a previous Nicks or Manougian enlargement) but demonstrates a PPM cause by somatic outgrowth, obesity or by the choice of a too small valve at the initial operation.

The results observed in this small series of 8 patients with a complex history are encouraging. All survivors had a very satisfying clinical evolution with absence of symptoms in the daily life. The hemodynamic results were very encouraging and all patients had either improvement of the left ventricular function and regression of the left ventricular hypertrophy.

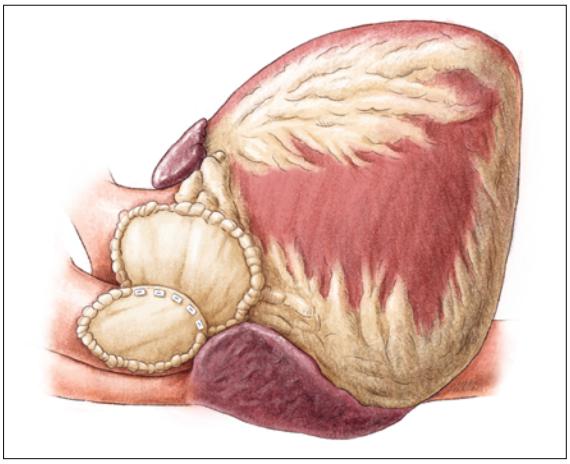

Figure 5: Finally, the patch to close the ventricular septum is extended cranially in order to close the aortotomy. A second patch is inserted on the right side of the septum and starts as deep as the patch on the left side. This patch is used to create a double closure of the ventricular septum and then to close to the right ventricular outflow tract without obstruction. Care should be taken to the integrity of the pulmonary valve. View of the operative site at the end of the procedure.

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Prosthesis-patient mismatch and early and late outcome after aortic valve replacement

PPM is a typical condition observed after surgical aortic valve replacement that was first described by Rahimtoola in 1978. It is defined by a too small aortic valve prosthesis (aortic valve area or/and EOA) according to the size of the patient defined by the body surface area (BSA) [1]. Moderate PPM occurs when indexed EOA is lower than 0.85 cm^2/m^2 while severe PPM is defined by EOA lower than 0,65 cm^2/m^2. The relationship between PPM and results following aortic valve replacement has been discussed controversally so far. While some authors found PPM to be associated with adverse early and/or late outcome, others did not find any negative influence [3–12]. In addition, PPM may also preclude regression of left ventricular hypertrophy [13, 14]. More recently, some reports about PPM following transcatheter aortic valve implantation and surgical implantation of sutureless (rapid-deployment) valves, that have a similar design to transcatheter devices have been published as well [4, 7, 12–16]. In the early postoperative period, PPM may have a particular negative impact on outcome because it may affect patients with impaired left ventricular function prior to surgery because the preoperatively damaged left ventricle is particularly vulnerable when a residual gradient increases the hemodynamic burden. Mortality is reported between 4 and 8% in patients with PPM [10, 14, 17].

In a particularly large series of 2154 patients, Rao and co-authors calculated the ratio between the prosthetic valve EOA and the patient's BSA to account for differences between manufacturers' labeled valve sizes (17). The lowest decile in this cohort had a calculated EOA/BSA of <0,75 cm^2/m^2 (Small group, n = 227) compared with the control group (n = 1927), in whom the EOA/BSA ratio was >0,75 cm^2/m^2. The authors found that 30-day mortality was significantly higher (8% versus 5%, p = 0.03) in patients with PPM. Actuarial survival at 12 years was 50+/-5% in the small group compared with 49±2% in the control group (P = 0,27). However, freedom from valve-related mortality was significantly lower in the small group (75+/-5% versus 84±2%, P = 0.004). Cox regression analysis determined age and NYHA functional class to be the multivariate predictors of overall mortality, whereas advanced age and EOA/BSA <0,75 cm^2/m^2 were found to be the predictors of valve-related mortality.

In a recent series of 1707 patients, there was a significant difference in early mortality (0,6% in patients without PPM and 4,2% in those with PPM (p <0,005). PPM was more frequently observed in female, obese and older patients as well as in patients with tissue valves and/or smaller sizes 19 and 21 mm [18].

Moreover, the patient's age and indexed valve EOA were found to be independent predictors of long-term valve-related death. This relationship was even more pronounced in patients suffering from a reduced left ventricular function since EF below 40% was associated with a late mortality of 67% [18]. Similarly, PPM was associated with long-term all cause mortality and remained a strong independent factor even after adjustment for other risk factors [10]. Some of these studies emphasize the fact that a failing ventricle is very sensitive to an increased afterload, not only in the early postoperative period and that every surgical technique that may help to avoid PPM should be considered particularly in patients with additional workload or concomitant diseases such as hypertension, coronary artery disease, and mitral insufficiency [5, 6, 9, 10, 14, 18].

Strategies to avoid PPM

One of the main goals when the surgeon plans a surgical aortic valve replacement (SAVR) should address the question how to avoid PPM? To fulfill this purpose, estimation of the potential risk of PPM prior to SAVR (but also TAVR) and calculation of the indexed EOA using reference values is mandatory. In addition, precise assessment of the annulus size during preoperative or at least intraoperative echocardiography may help to identify those patients at risk for the implantation of a too small valve. This may be particularly important in patients with a large BSA and in very thin and small patients (e.g. women) in whom the aortic annulus is expected to be small. Based on this assessment, an appropriate surgical/interventional strategy may be selected, such as planned annulus enlargement, the implantation of a sutureless valve or the option for transcatheter valve replacement in order to implant a large enough valve at the initial operation.

One simple method is to assess the patient's BSA, then to multiply it by 0,85 cm^2/m^2. The result gives the smallest EOA that the prosthetic valve should have to avoid PPM. As an example: if the patient's BSA is 1,9 m^2, the minimal EOA to avoid PPM would be 1,9 × 0,85 = 1,61 cm^2. The surgeon should choose the prosthesis according to this EOA and the reference values known for the different types and sizes of implants.

In the case depicted above, the EOA of the prosthetic valve should be larger than 1,61 cm^2 to avoid PPM. If a mechanical valve would be considered, a 21 mm St-Jude Medical Regent valve (EOA 2,0 ± 0,7 cm^2) or a 23 mm Medtronic AP360 (EOA 2,0 ± 0,6 cm^2) would be the proper choice.

In case of SAVR with a tissue valve, a 21 mm Carpentier Perimount Magna (EOA 1,7 ± 0,3 cm^2) or a 27 mm Hancock II (EOA 1,6 ± 0,2 cm^2) should be selected. However,

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looking at the relatively large confidence intervals of these EOAs, implantation of the next larger valve should be sought whenever possible. Most companies now provide with tables that precisely describe the projected indexed EOA for a specific patient's BSA and prosthesis size. When using such values, it is important to remember that some important discrepancies still exist between the sizes of the different types of prostheses and the size for a given patient's annulus. Especially in vitro data seem to be usually too optimistic.

The consensus document of the EACTS recommends comparison and selection of surgical heart valves (SHV) using the Valve Chart which can be used when comparing SHVs from different manufacturers (table 2) [19]. Basically, there are four potential scenarios when the surgeon faces a potential PPM issue:

- To select the implant with a larger EOA such as a stentless bioprosthesis, a sutureless bioprosthesis, a new generation mechanical prosthesis or exceptionally an aortic homograft
- To enlarge the aortic annulus to accommodate for a larger prosthesis
- To replace the aortic valve with a pulmonary autograft (Ross procedure)
- To switch the patient (if reasonable according to his age) for a TAVR

To accept a moderate PPM, especially in patients with a normal LV function is a suboptimal option. Several techniques of aortic annulus enlargement have been described (table 3): the first one was the Nicks' procedure (20). It is the easiest way to enlarge the aortic annulus; it is safe and can be performed after a short learning curve. However, it is the technique with the lowest degree of annular enlargement. In 1979 the Manougian technique was described: it differs from the Nicks technique as the former involves an incision through the aorta and the aortic annulus as low as into the anterior mitral leaflet, thus creating a larger diameter in the annulus and facilitating the insertion of a larger valve [21].

The Konno-Rastan procedure, described in this report is an anterior annulus enlargement that also allows an unobstructed LVOT [22, 23]. In comparison with both techniques presented above, the Konno procedure is the most challenging one but provides the greatest possible enlargement, It is the operation with the highest risk of atrioventricular block. In patients with a prior or a concomitant mitral valve procedure, (e.g. replacement), the Konno enlargement is ideal since it does not interfer with aorto-mitral continuity.

Nevertheless, when handling with a narrow aortic annulus in children, adolescents and younger adults, the original Ross procedure is still one of the best options – if not the best – to avoid PPM already during the initial operation. However, this intervention is not frequently performed since there are probably only few centres (and thereby also surgeons) familiar with the whole spectrum of LVOT enlargment procedures.

For adult patients having received prior aortic valve replacement and suffering from a severe PPM with corresponding symptoms, the Konno enlargement procedure is a valuable option [24, 25]. Comparing the different methods in a cadaveric model, Losenno found that all aortic enlargement methods allow for insertion of a larger valve [26]. The Nicks, Manougian, modified Bentall and aortoventriculoplasty procedures enlarged the annulus by 0,43 ± 0,45 mm, 3,63 ± 0,95 mm, 0,78 ± 0,65 mm, and 6.08 ± 1,19 mm, respectively (P <0,001). The larger change in prosthesis size

	EOAi by Prosthesis size (mm)					
Prosthesis size (mm)	19	21	23	25	27	29
Average EOA (cm^2)	1.1	1.3	1.5	1.8	2.3	2.7
BSA (m^2)						
0.6	1.83	2.17	2.50	3.00	3.83	4.50
0.7	1.57	1.86	2.14	2.57	3.29	3.86
0.8	1.38	1.63	1.88	2.25	2.88	3.38
0.9	1.22	1.44	1.67	2.00	2.56	3.00
1	1.10	1.30	1.50	1.80	2.30	2.70
1.1	1.00	1.18	1.36	1.64	2.09	2.45
1.2	0.92	1.08	1.25	1.50	1.92	2.25
1.3	0.85	1.00	1.15	1.38	1.77	2.08
1.4	0.79	0.93	1.07	1.29	1.64	1.93
1.5	0.73	0.87	1.00	1.20	1.53	1.80
1.6	0.49	0.88	0.88	0.88	0.88	1.69
1.7	0.65	0.76	0.88	1.06	1.35	1.59
1.8	0.61	0.72	0.83	1.00	1.28	1.50
1.9	0.58	0.68	0.79	0.95	1.21	1.42
2	0.55	0.65	0.75	0.90	1.15	1.35
2.1	0.52	0.62	0.71	0.86	1.10	1.29
2.2	0.50	0.59	0.68	0.82	1.05	1.23
2.3	0.48	0.57	0.65	0.78	1.00	1.17
2.4	0.46	0.54	0.63	0.75	0.96	1.13
2.5	0.44	0.52	0.60	0.72	0.92	1.08

Table 2: From Pibarot, Heart 2006 (with permission) Ref 2.

Table 3: Types of surgical enlargement technique of the aortic root, and how they are carried out. The order corresponds to the degree of enlargement that can expected with the respective technique.

Nicks Posterior	Through the noncoronary sinus, ends where the mitral valve originates Single patch repair of the aortotomy
Manouguian Posterior	Through the left noncoronary commissure, ends within the anterior leaflet of the mitral valve Double patch repair of aortotomy and the left atrial roof
Nunez Posterior	Through the left noncoronary commissure, ends where the mitral valve originates Single patch repair of the aortotomy
Konno-Rastan Anterior	[1] Through the right coronary sinus, ends within the interventricular septum (2) Into the right ventricular free wall, inferior to the pulmonary valve Double patch repair of interventricular septum and the right ventricle

was observed after aortoventriculoplasty techniques (P <0,001).

In a comparative review of all enlargement techniques, Massias concluded that the choice of operative technique largely depends on the preference of the surgeon since all techniques may effectively help to reduce or avoid PPM. However, there is still absence of significant data investigating the impact of the surgical technique on patient mortality and morbidity.

In one recent retrospective analysis of 4120 patients receiving isolated SAVR, 171 (4%) underwent a concomitant aortic annulus enlargement. Early mortality was not statistically significantly different: 1,4% after isolated SAVR and 1,8% in case of additional annulus enlargement [28]. Postoperative complications were <5% in all matched 338 patients: bleeding (3% vs 3%), pericardial effusion (3,0% vs 4,2%), sternal instability (1,8% vs 0%) and sternal wound infection (3,0% vs 1,2%). Factors independently associated with overall mortality were age [hazard ratio (HR) 1,71], chronic obstructive pulmonary disease (HR 1,47), diabetes (HR 1, 82), atrial fibrillation (HR 2,14) and postoperative respiratory failure (HR 2,84). The authors concluded that annulus enlargement can be performed safely in experienced centres with minimal significant increase in the risk of adverse outcome.

In our series, the majority of patients received a mechanical valve prosthesis: reasons for that were the younger age of the patients, the wish of the patients to avoid a further procedure and/or the fact that they were already on oral anticoagulation because of atrial fibrillation or because of a mechanical mitral prosthesis.

Conclusion

The Konno anterior aorto-ventriculoplasty is a very efficient method to sufficiently enlarge the left ventricular outflow tract, independently from the initial pathology. This technique is only rarely performed because it is a more complex intervention; this is reflected by the prolonged duration of cardiopulmonary bypass. However, this type of annular and subvalvular enlargement usually allows the insertion of a larger prosthesis than after a simpler Manougian or Nicks posterior annulus enlargement. For adult patients with severe PPM or another form of complex LVOT obstruction, the Konno intervention is a valuable option that should be offered to these patients.

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References

1 Rahimtoola SH. The problem of valve prosthesis-patient mismatch. Circulation. 1978;58:20 4.
2 Pibarot P, Dumesnil JG. Prosthesis-patient mismatch: definition, clinical impact, and prevention. Heart. 2006;92:1022–9.
3 Swinkels B, Ten Berg J, Kelder J, Vermeulen F, van Boven WJ, de Mol B. What can we learn from the past by means of very long-term follow-up after aortic valve replacement? J Clin Med 2021;10:3925.
4 Tenacle J, Pibarot P, Herrmann HC, Kodali S, Leipsic J, Blanke P. Prosthesis-patient mismatch after aortic valve replacement in the PARTNER 2 trial and registry. JACC Cardiovasc Interv 2021;14:1466–77.
5 Dahlbacka S, Laakso T, Kinnunen EM, Moriyama N, Laine M, Virtanen M, et al. Patient-prosthesis mismatch worsens long-term survival: insights from the FinnValve registry. Ann Thorac Surg 2021;111:1284–90.
6 Kim HJ, Kim HJ, Kim JB, Jung SH, Choo SJ, Chung CH, et al. Prosthesis-patient mismatch after surgical aortic valve replacement in patients with aortic stenosis. Interact Cardiovasc Thorac Surg 2020;31:152–7.
7 He S, Fang Z. Incidence, predictors and outcome of prosthesis-patient mismatch after transcatheter aortic valve replacement: a meta-analysis. Medicine 2020;99:e20717.
8 Tasca G. Brunelli F, Cirillo M, et al. Impact of valve prosthesis-patient mismatch on left ventricular mass regression following aortic valve replacement. Ann Thorac Surg. 2005;79:505–10.
9 Tasca G, Mhagna Z, Perotti S, et al. Impact of prosthesis-patient mismatch on cardiac events and mid-term mortality after aortic valve replacement in patient with pure aortic stenosis. Circulation 2006;113:570-6.
10 Elmahdy W, Osman M, Farag M, Shoaib A, Saad H, Sullivan K, et al. Prosthesis-patient mismatch increases early and late mortality in low risk aortic valve replacement. Semin Thoracic Surg 2020;33:23–30.
11 Howell NJ, Keogh BE, Ray D, et al. Patient-prosthetic mismatch in patient with aortic stenosis undergoing aortic valve replacement does not affect survival. Ann Thorac Surg 2010;89:6064.
12 Pibarot P, Weissman NJ, Stewart WJ et al. Incidence and sequelae of prosthesis-patient mismatch in transcatheter versus surgical valve replacement in high-risk patients with severe aortic stenosis. J Am Coll Cardiol 2014;64:1323–34.
13 Tasca G, Brunelli F, Cirillo M, Dalla Tomba M, Mhagna Z, Troise G, Quaini E. Impact of the improvement of valve area achieved with aortic valve replacement on the regression of left ventricular hypertrophy in patients with pure aortic stenosis. Ann Thorac Surg. 2005;79:1291–6
14 Ruel M, Al-Faleh H, Kulik Chan KL. Prothesis-patient mismatch after aortic valve replacement predominantly affects patients with preexisting left ventricular dysfunction: effects on survival, freedom from heart failure and left ventricular mass regression. J Thorac Cardiovasc Surg 2006;131:1036–44.
15 Leone PP, Fazzari F, Cannata F, Sanz-Sanchez J, Mangieri A, Monti L et al. Clinical and technical challenges of prosthesis-patient mismatch after transcatheter aortic valve implantation. Front Cardiovasc Med 2021;8:670457. doi 10.3389/fcvm.2021.670457.
16 D'Onofrio A, Tessari C, Cibin G, Lorenzoni G, Martinelli GL, Solinas M, et al. Clinical and hemodynamic outcomes of rapid-deployment aortic bioprostheses. Semin Thorac Cardiovasc Surg 2021; May 9:S1043-0679(21)00200-8. doi:10.1053/j.semtcvs. 2021.04.006.
17 Rao V, Jamieson WE, Ivanov J, Armstrong S, David TE. Prosthesis-patient mismatch affects survival after aortic valve replacement. Circulation. 2000;102(Suppl 3):5–9.
18 Mohty D, Dumesnil JG, Echahidi N, et al. Impact of prosthesis-patient mismatch on long-term survival after aortic valve replacement: influence of age, obesity and left ventricular dysfunction. J Am Coll Cardiol 2009;53:39–47.
19 Durko AP, Pibarot P, Atluri P, Bapat V, Cameron DE, Casselman FPA et al. Essential information on surgical heart valve characteristics

for optimal valvew prosthesis selection: expert consensus document from the European Association for Cardio-throacic Surgery, the Society of Thoracic Surgeons and the American Association for Thoracic Surgery valve labelling task force. Eur J Cardiothoracic Surg 2021;59:54–64.
20 Nicks R, Cartmill T, Bernstein L. Hypoplasia of the aortic root: the problem of aortic valve replacement. Thorax 1970;25:339–46.
21 Manougian S, Seybold-Epting W. Patch enlargement of the aortic valve ring by extending the aortic incision into the anterior mitral leaflet. J Thorac Cardiovasc Surg. 1979;78:402–12.
22 Konno S, Imai Y, Lida Y, Nakajima M, Tatsuno K. A new Method for prosthetic valve replacement in congenital aortic stenosis associated with hypoplasia of the aortic valve ring. J Thorac Cardiovasc Surg. 1975;70:909–17.
23 Rastan H, Koncz J. Plastic enlargement of the left ventricular outflow tract. A new operative method. Thoraxchir Vask Chir. 1975;23:169–75.
24 Erez E, Tam VK, Williams WH, Kanter KR. The Konno aortoventriculoplasty for repeat aortic valve replacement. Eur J Cardiothorac Surg. 2001 Jun;19(6):793–6.
25 Urganci E, Aliabadi-Zuckermann A, Sandner S, Herbst C, Gökler J, Laufer G, et al. The Konno-Rastan procedure. Multimed Man Cardiothorac Surg. 2019 Jul 25;2019
26 Losenno KL, Gelinas JJ, Johnson M, Chu MW. Defining the efficacy of aortic root enlargement procedures: a comparative analysis of surgical techniques. Can J Cardiol. 2013;4:434–40.
27 Massias SA, Pittams A, Mohamed M, Ahmed S, Younas H, Harky A. Aortic root enlargement: When and how. J Card Surg. 2020;1-7.
28 Haunschild J. Scharnowski S, Mende M, et al. Aortic root enlargement to mitigate patient-prosthesis mismatch: do early adverse events justify reluctance? Eur J Cardiothorac Surg. 2019;56:335-342.

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