Valve adaptation as a cause of disc opening reduction in mechanical heart valves: the case of the Lillehei-Kaster valve

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At fluoroscopy a decreased disc motion in mechanical heart prostheses is often a sign of valve thrombosis. On occasion, however, despite an exhaustive diagnostic work-up, common causes of prosthetic valve thrombosis are not found. In these cases the valve disc abnormalities are thought to be due to functional changes. We here report our experience with 5 consecutive patients carrying the Lillehei-Kaster prosthesis who had this fluoroscopic finding that was lately attributed to "valve physiologic adaptation". The time of onset, differential diagnosis and clinical/hemodynamic impact of valve adaptation are discussed.

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Introduction

A decreased disc motion in mechanical heart prosthesis is usually a sign of valve obstruction. Acute or subacute thrombosis, progressive pannus formation or their combination are the most frequent causes of obstruction[1]. Less frequently, disc impingement against intracardiac structures, unraveled or excessively long ends of sutures and retained valve remnants represent extrinsic factors that can interfere with the normal valve function[2-4]. In a few instances, however, a plausible cause of valve dysfunction cannot be found. In such cases, the abnormal valve disc opening may be due to specific hemodynamic conditions (i.e. low output state, suboptimal valve orientation, paravalvular regurgitation)[5,6] or to the so-called "valve physiologic adaptation"[7]. It is crucial to differentiate an organic from a functional disc motion restriction since the treatment strategy and prognosis are totally different. We here describe the clinical, non-invasive and intraoperative findings of 5 consecutive patients with the tilting disc Lillehei-Kaster (L-K) prosthesis (Fig. 1) presenting with clinical and hemodynamic findings suggestive of obstructive valve thrombosis that was lately attributed to functional factors.

Description of cases

During a period of 3 years 5 patients (3 males, 2 females, aged 58-66 years) with the L-K mechanical prosthesis (3 mitral and 2 aortic) were seen at our Institution. Table I lists the clinical, non-invasive and surgical findings. The mean time interval from surgery was 18 years (range 15-21 years). Patients were admitted to the hospital because of heart failure in 4 and cerebral ischemia in 1. Two patients were in sinus rhythm and 3 in atrial fibrillation. At the time of presentation the anticoagulation status was optimal in all cases (INR > 3).

Cinefluoroscopy, transthoracic and transesophageal echocardiography (TEE) were carried out in each patient as previously described[8]. At cinefluoroscopy the valve opening angle was definitively reduced as compared to reference values (58 ± 2 vs 80°). Color Doppler identified normal diastolic/systolic flow jets in all cases. The mean Doppler gradient (10.6 ± 1.1 mmHg) in the 3 patients with mitral prostheses (Fig. 2) was higher than the mean (+2 SD) value obtained in a reference patient population with normally functioning tilting disc mitral prostheses (any value > 8 mmHg being considered abnormal)[8]. Besides, with regard to the 2 patients with aortic prostheses, one had very high pressure gradients (64/42 mmHg) (Fig. 3) and a reduced valve area (0.9 cm^2) while the other had normal values across the prosthesis with moderate periprosthetic regurgitation. TEE failed to demonstrate any masses (thrombus and/or pannus formation) in proximity of the prostheses. Cardiac catheterization showed moderate pul-

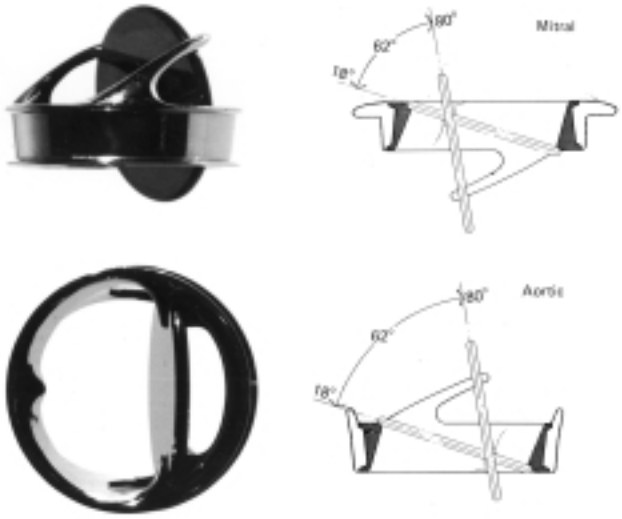

Figure 1. *The Lillehei-Kaster prosthesis.*

monary hypertension with a normal cardiac index in all cases. No significant coronary artery stenosis was detected. In order to evaluate whether an increase in cardiac output could modify the valve disc opening, atrial (patients no. 3 and 4) or ventricular (patients no. 1, 2 and 5) pacing at a maximal heart rate of 130 b/min was performed. The cardiac output and cardiac index increased from 5.49 ± 0.61 to 6.59 ± 0.70 l/min and from 3.36 ± 0.41 to 4.08 ± 0.48 l/min/m^2, respectively. The valve disc opening angle, however, did not change (58 ± 2 vs 60 ± 1°) in any patients.

On the basis of these data reasons for heart failure were thought to be progressive pulmonary hypertension with right heart failure in the 3 patients with mitral prostheses, and patient-prosthesis mismatch in the patient with an aortic valve and a high pressure gradient. In the remaining patient with an aortic prosthesis, cerebral ischemia was found to be due to carotid artery stenosis. Although TEE did not show any true evidence of prosthetic thrombosis, all patients underwent reoperation, in view of their prosthesis-related clinical symptoms (patients no. 1, 2, 4 and 5) or of concomitant significant aortic regurgitation (patient no. 3) and considering the long time elapsed from surgery in all. At surgery, visual and manual inspection confirmed the TEE findings (Fig. 4). A restricted pannus not encroaching on the sewing ring was found in all cases. Valve disc excursion was unimpeded. The explanted prostheses were manually inspected and checked with the aid of an optical microscope. No macroscopic abnormality was detected on both the valve housing and the disc. The occluder showed a worn circular band that

Table I. Clinical, non-invasive and surgical findings.

	Patients				
	1	2	3	4	5
Age (years)	60	58	64	66	61
Sex	F	M	M	F	M
BSA (m^2)	1.49	1.67	1.68	1.58	1.70
Prosthesis	L-K, mitral, Ø 20	L-K, mitral, Ø 20	L-K, aortic, Ø 18	L-K, aortic, Ø 18	L-K, mitral, Ø 20
Rhythm	AF	AF	SR	SR	AF
Time from surgery (years)	21	19	19	16	15
Symptoms	Heart failure NYHA III	Heart failure NYHA III	Cerebral ischemia	Heart failure NYHA III	Heart failure NYHA II-III
Cinefluoroscopic findings					
Opening angle (°)	62	56	56	56	58
Closure angle (°)	18	18	18	18	18
TTE findings					
PG (mmHg)	22/10	22/10	36/24	64/42	26/12
VA (cm^2)	1.4	1.38	1.3	0.9	1.34
EF (%)	58	62	57	41	66
TEE findings	Moderate periprosthetic leak Absence of thrombus and/or pannus formation interfering with disc motion				
Hemodynamic findings					
CI (l/min/m^2)	3.9	3.6	3.0	2.9	3.4
PAP (mmHg)	65/25 (38)	46/19 (30)	80/33 (48)	45/26 (32)	58/26 (37)
PWP (mmHg)	28	25	29	25	24
Surgical findings	No prosthetic thrombosis. In all cases only mild pannus formation not encroaching on the sewing ring				
Explanted prosthesis evaluation	*Housing:* no structural defect. *Occluder:* worn circular band uniformly distributed on both sides. The depth of this band was the same as that found in normally functioning prostheses. The disc was moving freely within the struts				

AF = atrial fibrillation; BSA = body surface area; CI = cardiac index; EF = ejection fraction; L-K = Lillehei-Kaster; PAP = pulmonary artery pressure; PG = pressure gradient; PWP = pulmonary wedge pressure; SR = sinus rhythm; TEE = transesophageal echocardiography; TTE = transthoracic echocardiography; VA = valve area.

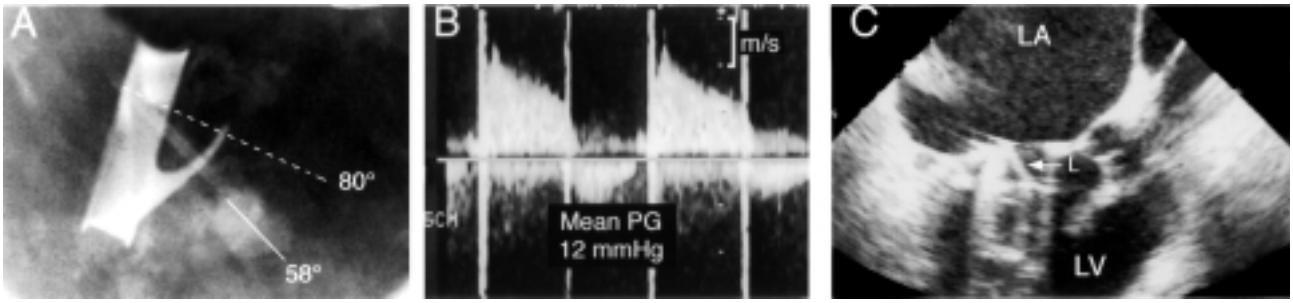

Figure 2. *Cinefluoroscopy* (A)*, Doppler* (B) *and transesophageal echocardiography* (C) *in a patient with a mitral Lillehei-Kaster prosthesis. In comparison to normal values (80°,* dotted line*), the opening angle is decreased (58°). L = leaflet; LA = left atrium; LV = left ventricle; PG = pressure gradient.*

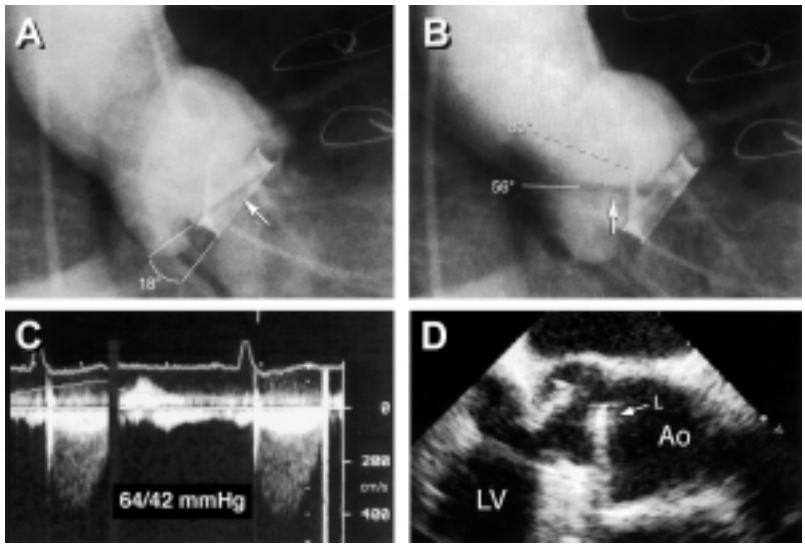

Figure 3. *Cinefluoroscopy* (A *and* B)*, Doppler* (C) *and transesophageal echocardiography* (D) *in a patient with an aortic Lillehei-Kaster prosthesis.* Frames A *and* B *were taken during aortography. In* frame A*, it may be seen that the valve is closed and the disc is correctly seated* (arrow) *with a closure angle of 18°. In* frame B*, it may be seen that the valve opens with an angle of 56° that is smaller than normal values (80°,* dotted line*). Ao = aorta; L = leaflet; LV = left ventricle.*

was uniformly distributed on both sides suggesting a correct rotation of the disc. The depth of this circular band was similar to that usually found in normally functioning tilting disc prostheses.

Table II shows clinical and echocardiographic data after prosthesis replacement.

Discussion

The L-K valve is a monocuspid disc prosthesis that was introduced into clinical practice in 1969 (Fig. 4). The valve housing is made of titanium and the occluder consists of a thin pyrolite-carbon disc coating a graphite substrate. Two disc retaining struts arise diagonally from the outflow edges of the valve housing. The angles of disc opening and closure are 80 and 18° respectively. Despite favorable results *in vitro*, the *in vivo* hemodynamic performance was somewhat less satisfactory and the prosthesis was withdrawn from the market in the late '80s[9-14]. During the early phases of the clinical experience it was incidentally detected at fluoroscopy that the opening angle of the valve disc was frequently smaller than that observed in *in vitro* tests[10,15]. This finding was thought to be the result of non-specific hemodynamic factors although in view of the lack, at that time, of TEE evaluation, it was not possible to definitely rule out common causes of valve dysfunction. The 5 patients included in this case report study, on the contrary, were submitted to an extensive diagnostic work-up that failed to reveal any of the common intrinsic or extrinsic causes of valve obstruction. Although functional (i.e. atrial fibrillation-induced flow variability) or geometrical mechanisms (i.e. right ventricular overload with paradoxical motion of the septum) interfering with valve opening cannot be excluded, "valve physiologic adaptation" was thought to be the cause for the decreased disc motion.

Actually, the prevalence of this finding is unknown. Since cinefluoroscopy is the best diagnostic method available for the identification of this problem its recognition strictly depends on how often this tech-

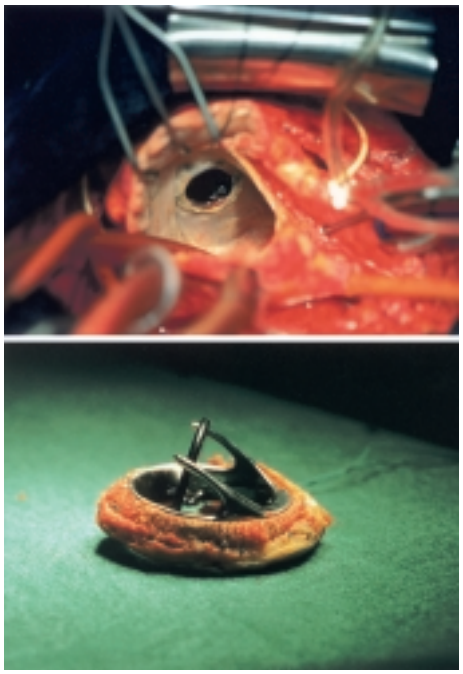

Figure 4. *Intraoperative findings.* Upper panel: *mitral Lillehei-Kaster prosthesis seen by the atrial side showing no significant pannus encroaching on the sewing ring.* Lower panel: *explanted prosthesis showing free maximal disc opening.*

nique is currently used. The lack of systematic data suggests either that the occurrence of this phenomenon is actually low or that it has largely passed undetected because of the limited use of fluoroscopy. The latter hypothesis seems more likely considering the ever increasing role of echocardiography as the first-choice diagnostic method for the evaluation of heart valve prostheses. The fact that none of our patients underwent a cinefluoroscopic study despite a very long follow-up supports this concept. Unfortunately, transthoracic echocardiography is less sensitive than fluoroscopy in the evaluation of valve disc opening and Doppler study may be normal even in case of proved prosthetic thrombosis leading to a potential underestimation of this heart valve complication[8,16-20].

The extent of valve adaptation varies among different prostheses. Ishimaru et al.[7] reported, for the Björk-Shiley tilting disc valve, a mean decrease in the opening angle of 4° (56 ± 2°) that corresponds to 6% of the maximal opening value (60°). Other prostheses, such as L-K, Bicer[8], Omniscience[21,22], Omnicarbon and Medtronic-Hall showed a 15-20° decrease in the opening angle that corresponds to 20-30% of their maximal opening value (75-80°). Since the opening angle during adaptation is roughly the same (i.e. 60°), the greater reduction in disc motion is simply the result of the larger disc opening. Thus, valve disc adaptation is rarely detectable in heart prostheses with a maximal opening angle of 60°. Changes in these types of valves are largely explained by the interobserver variability at fluoroscopy (approximately 5%)[8,23] rather than by true valve adaptive changes.

A decrease in disc motion due to adaptation should be differentiated from that due to prosthetic valve thrombosis. An intermittent full opening of the disc at rest as well as an opening normalization in case of an increased cardiac output may suggest functional changes[10]. In our 5 patients we did not find any significant variation in the disc opening angle during pacing. This could be the result of a modest increase in cardiac output (20%) elicited by pacing likely to be due to the unfavorable (ventricular) mode of pacing in 3 patients or to the underlying heart failure in most. This suggests that the valve capacity to improve its performance by resorting to a larger opening angle and to a larger functional area ("reserve opening capacity") comes into play for a larger cardiac output increase. If the patient's functional class is severely reduced, as in our cases, this flow threshold could never be reached and this valve capacity would thus be hampered. A further consideration favoring functional changes is that in our patients the decrease in disc motion was quite stable (i.e. 58-60°) and similar to that reported by Sigwart et al.[10] (i.e. 63°) and by Paquet et al.[15] (i.e. 60°). It seems unlikely that a thrombotic process can modify disc excursion to the same extent in all patients.

A decrease in valve disc opening due to physiologic adaptation should have negligible hemodynamic and clinical effects. *In vitro* evaluation with the Omniscience prosthesis as well as historical series with other types of tilting disc prostheses have shown that an

Table II. Clinical and echocardiographic data after prosthesis replacement.

	Patients				
	1	2	3	4	5
Type of prosthesis	CPHV, Ø 25	CPHV, Ø 27	CPHV, Ø 21	SB, Ø 21	SB, Ø 27
NYHA class	III→I	III→I	I→I	III→I	II-III→II
Pressure gradient (mmHg)*	3.4	3.8	28/14	31/24	2.8
Ejection fraction (%)	56	65	55	58	60

CPHV = CarboMedics prosthetic heart valve; SB = Sorin Bicarbon. * mean pressure gradient for mitral prostheses measured at 6 months of follow-up.

opening angle of 60° is hemodynamically successful[21,24]. However, in the 3 patients with mitral prostheses, the "mildly" stenotic prosthesis could have led to the development of a chronic and progressively increasing pressure gradient and ultimately to heart failure. This, in turn, could have hampered the valve capacity to recruit a larger valve orifice by limiting the increase in cardiac output during exercise. In the patients with an aortic prosthesis and a high pressure gradient the progressive deterioration of the NYHA functional class may have been due to a moderate valve prosthesis-patient mismatch[25,26]. The effective to anatomic orifice area ratio was in fact < 0.9 cm^2/m^2. The improved clinical conditions (i.e. NYHA classification) and pressure gradient observed in all but one patient after prosthesis replacement support this hypothesis.

The major limitation of this report is that the L-K device is a prosthesis that has been withdrawn from the market in the late '80s. However, many patients may still be carrying this type of valve. Moreover, since similar prostheses are currently available on the market, it is relevant to know if these results and considerations may be extrapolated to these valves. The lack of changes in pressure gradient during pacing in the 5 patients is another limitation of the study. Besides, no personal data are available on the variations in disc opening during pacing in patients with the L-K (or similar) prosthesis and a normal ventricular function.

In conclusion, some patients with the L-K prosthesis show, at fluoroscopy, a reduced disc opening mimicking valve thrombosis that is actually due to "physiological valve adaptation". This finding appears to be a prerogative of large opening angle prostheses. An increased cardiac output is the trigger to recruit the "valve reserve capacity". However, the inability to effectively increase cardiac output during exercise in patients with heart failure may limit the hemodynamic advantage of this valve and may be, in some cases, even detrimental.

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