Aortic valve bypass surgery in severe aortic valve stenosis: Insights from cardiac and brain magnetic resonance imaging

Cesare Mantini, MD, PhD,^a Massimo Caulo, MD, PhD,^a Daniele Marinelli, MD,^b Piero Chiacchiaretta, PhD,^c Armando Tartaro, MD,^c Antonio Raffaele Cotroneo, MD,^a and Gabriele Di Giammarco, MD^b

ABSTRACT

Objective: To investigate and describe the distribution of aortic and cerebral blood flow (CBF) in patients with severe valvular aortic stenosis (AS) before and after aortic valve bypass (AVB) surgery.

Methods: We enrolled 10 consecutive patients who underwent AVB surgery for severe AS. Cardiovascular magnetic resonance imaging (CMR) and brain magnetic resonance imaging were performed as baseline before surgery and twice after surgery. Quantitative flow measurements were obtained using 1.5-T magnetic resonance imaging (MRI) scanner phase-contrast images of the ascending aorta, descending thoracic aorta (3 cm proximally and distally from the conduit-to-aorta anastomosis), and ventricular outflow portion of the conduit. The evaluation of CBF was performed using 3.0-T MRI scanner arterial spin labeling (ASL) through sequences acquired at the gray matter, dorsal default-mode network, and sensorimotor levels.

Results: Conduit flow, expressed as the percentage of total antegrade flow through the conduit, was $63.5 \pm 8\%$ and $67.8 \pm 7\%$ on early and mid-term postoperative CMR, respectively ($P < .05$). Retrograde perfusion from the level of the conduit insertion in the descending thoracic aorta toward the aortic arch accounted for 6.9% of total cardiac output and 11% of total conduit flow. We did not observe any significant reduction in left ventricular stroke volume at postoperative evaluation compared with preoperative evaluation ($P = .435$). No differences were observed between preoperative and postoperative CBF at the gray matter, dorsal default-mode network, and sensorimotor levels ($P = .394$).

Conclusions: After AVB surgery in patients with severe AS, cardiac output is split between the native left ventricular outflow tract and the apico-aortic bypass, with two-thirds of the total antegrade flow passing through the latter and one-third passing through the former. In our experience, CBF assessment confirms that the flow redistribution does not jeopardize cerebral blood supply. (J Thorac Cardiovasc Surg 2018;156:1005-12)

Cerebral blood flow before and after AVB implantation.

Central Message

After AVB cardiac output is split between native aortic valve and the conduit with two-thirds of the antegrade flow through the conduit without any cerebral perfusion impairment.

Perspective

The use of AVB in the current era could be considered complementary to TAVI for the treatment severe aortic stenosis in high risk patients. Herein we report the result of our experience in the use of AVB and the hemodynamic changing induced by the creation of a second left ventricular outflow and the potential influence on cerebral blood flow.

See Editorial Commentary page 1013.

From the ^aDepartment of Neuroscience, Imaging and Clinical Science, Institute of Radiology, ^bDepartment of Neuroscience, Imaging and Clinical Sciences, Institute of Cardiac Surgery, Department of Cardiac Surgery, and ^cDepartment of Neuroscience, Imaging, and Clinical Science, Università "G.D'Annunzio" Chieti e Pescara, Chieti, Italy.

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Address for reprints: Gabriele Di Giammarco, Clinica Cardiochirurgica Ospedale, S.S. Annunziata via dei Vestini s.n.c., Chieti 66100, Italy (E-mail: gabriele.digiammarco@unich.it).

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Aortic stenosis (AS) is by far the most frequent valve disease, and its prevalence increases progressively with age. The introduction of aortic valve replacement (AVR) in the early 1960s improved the prognosis of patients with symptomatic severe

Scanning this QR code will take you to a supplemental video for the article.

Abbreviations and Acronyms

AS	= aortic stenosis
AAC	= apico-aortic conduit
AVB	= aortic valve bypass
AVR	= aortic valve replacement
BMR	= brain magnetic resonance imaging
CBF	= cerebral blood flow
CCC	= concordance correlation coefficient
CMR	= cardiovascular magnetic resonance imaging
CPB	= cardiopulmonary bypass
DMN	= default mode network
LV	= left ventricular
MRI	= magnetic resonance imaging
NAV	= native aortic valve
PC	= phase-contrast
TAVI	= transcatheter aortic valve implantation

AS whose life expectancy was <3 years in the absence of surgery.¹⁻³ Recent surveys and meta-analyses have reported a 30-day all-cause mortality of 2% after AVR with 80% survival after 1 year.⁴⁻⁶ AVR requires median partial sternotomy or small right thoracotomy, the use of cardiopulmonary bypass (CPB), ascending aorta manipulation (cannulation and cross-clamping), cardioplegic arrest, debridement of the native aortic valve (NAV), and aortic suture after prosthesis implantation. Each of these surgical steps may represent a risk factor for additional morbidity and/or mortality, particularly in cases of calcified or porcelain aorta and cases with severe systemic comorbidities.^{5,7-9} Several studies have reported that nearly 60% of patients with symptomatic AS are never referred to surgery, owing to the perioperative risks. This has sparked researchers' interest in alternative treatment approaches.^{3,10-12}

Transcatheter aortic valve implantation (TAVI) is indicated in patients with severe symptomatic AS who are deemed by the heart team to be unsuitable for surgery but have a sufficient life expectancy.¹³ However, anatomic issues (eg, peripheral vascular disease, coronary ostia position, annulus size, presence of severe left ventricular (LV) dysfunction, associated severe mitral valve disease) or severe renal dysfunction may be a contraindication to TAVI.¹⁴ Furthermore, hospital charges for TAVI are nearly twice those of aortic valve bypass (AVB).¹⁵ The insertion of a valved conduit into the apex of the left ventricle and directed to the thoracic descending aorta represents an alternative unconventional alternative surgical option proposed by Sarnoff and colleagues in 1955.¹⁶ It can be performed on the beating heart, with or without CPB support, through a small left thoracotomy. A second LV output is created to bypass aortic valve stenosis by means of a valved 2-piece conduit (Figure 1 and Video 1).¹⁷

AVB has been proposed as an alternative to AVR and recently as complementary to TAVI in selected patients, including those with (1) a heavily calcified ascending aorta (porcelain aorta); (2) a coronary ostia too close to the aortic annulus, at risk of obstruction after TAVI deployment; (3) a very narrow and calcified native aortic annulus in which the enlargement is at increased risk of complications; (4) a patent coronary artery bypass graft crossing the sternal midline at risk of damage in sternal reentry; (5) a contraindication to CPB; and (6) aortic valve calcifications involving a mitral anterior leaflet below 1 cm. Importantly, moderate or severe aortic valve regurgitation and/or the presence of severe calcifications or diffuse atheroma of the thoracic descending aorta are common contraindications to the creation of an apico-aortic conduit (AAC).¹⁷

The site of distal anastomosis on the midportion of the descending thoracic aorta is considered an issue as terms of flow distribution toward aortic territories both proximal and distal to the landing site.¹⁸ Even though it has been reported that antegrade flow through the AVB and NAV would account for 60% to 70% and 30% to 40% of total cardiac output, respectively,¹⁹ little is known about cerebral and aortic perfusion in this setting.

In this study, we sought to evaluate and describe the distribution of aortic blood flow and to assess the pattern of cerebral perfusion before and after AVB with cardiovascular magnetic resonance imaging (CMR) and brain magnetic resonance imaging (BMR).

METHODS

Study Population and Operative Technique

Between March 2012 and January 2013, 10 high-risk patients¹⁹ with severe aortic valve stenosis underwent AAC using a Correx AVB (Correx, Boston, Mass). The device includes a rigid preformed part that is released through an automatic delivery system into the LV apex and a valved conduit that is implanted onto the descending thoracic aorta below the level of the hilum of the lung (Figure 2). The porcine valve prosthesis used in the Correx AVB is a 21-mm stentless Elan valve (Vascutek Ltd, Inchinnan, United Kingdom).

The valved conduit on the descending thoracic aorta is installed first under side clamping. Then the ventricular conduit is released using an automated delivery system that retains the muscle core by means of an inflated balloon. The system was designed for implantation without the need for cardiac arrest and thus without the use of CPB. The diameter of the ventricular connector is 22 mm, and that of the valved conduit is 18 mm. Under general anesthesia, a left anterior thoracotomy (12-14 cm) at the fifth or sixth intercostal space is performed to provide access to both the LV apex and the descending thoracic aorta. After heparin administration at a dose of 1 mg/kg, an end-to-side anastomosis between the valved conduit and the side-clamped descending aorta was performed using a series of 2-0 braided U stitches reinforced with small pledgets on the aortic external surface. At this point, the clamp was moved on the valved conduit to prevent any bleeding even in the presence of a fully competent bioprosthesis. The ventricular arm of the AVB was then deployed, introducing the tip of the deflated balloon of the automated coring system through a stab wound at the LV apex in the center of a crown of braided 2-0 U stitches. Finally, the U stitches were knotted on the sewing ring of the AVB ventricular portion. The next step was the connection of the 2 portions by means of a cup system with a male end (LV connector side) and a female end (valved conduit

FIGURE 1. Cardiovascular magnetic resonance imaging (A) and computed tomography (B-F) images showing the aortic valve bypass that connects the left ventricular apex (white arrows) to the descending thoracic aorta (white arrowheads) by a valved conduit (black arrows).

VIDEO 1. Cine balanced steady-state free-precession magnetic resonance imaging cine sequence showing the aortic valve bypass connecting the left ventricular apex to the descending thoracic aorta by a valved conduit. Video available at: [https://www.jtcvs.org/article/S0022-5223\(18\)30981-4/fulltext](https://www.jtcvs.org/article/S0022-5223(18)30981-4/fulltext).

side). Finally, the connection of the 2 parts was secured with a fabric sling tightened over it, and the LV connector curvature was adjusted using a drawstring premounted in the conduit (Figure 2).

MRI Protocols

CMR and BMR were performed once before surgery and twice after surgery for first and second postoperative control.

CMR Protocol

CMR imaging was performed with a 1.5-T MR scanner (Achieva; Philips Medical Systems, Best, The Netherlands) using a dedicated phased-array cardiac synergy coil for signal reception. After localization of the heart using 3-plane and oblique survey images, cine imaging of cardiac function was performed with a balanced, steady-state free-precession technique at 30 phases per cardiac cycle (by vectorcardiographic gating) in 8 to 14 parallel short-axis, 2-chamber, and 4-chamber (8-mm thick, 0-mm gap). A 3-chamber view (for the LV ventricular outflow tract) and an oblique coronal view cine image (for the LV aortic outflow tract) were acquired. These images were used as localizers to prescribe cross-sectional flow-sensitive sequences. For the quantitative flow measurements, we obtained 4 through-plane phase-contrast (PC) sequences: in the ascending aorta (10 mm above the sinotubular junction), at the level of the conduit ventricular anastomosis and at the level of the descending thoracic aorta, 3 cm proximal and distal to the conduit–aortic anastomosis (Figure 3). PC

FIGURE 2. Correx aortic valve bypass system: left ventricular connector, valved conduit, and automated coring and delivery system. LV, Left ventricular.

imaging parameters included a repetition time of 4.60 to 4.92 ms, an echo time of 2.76 to 3.05 ms, a flip angle of 15°, 24 phases, pixel spacing of 1.32 to 2.07 mm, a slice thickness of 10 mm, and a 256×208 acquisition matrix. Each PC velocity mapping acquisition produced 2 images, including 1 magnitude image and 1 phase image. Typically, the MRI encoding velocity was 3.5 m/s in the ascending aorta, 1.5 m/s in the ventricular portion of the conduit, and 1 m/s in the descending thoracic aorta. Phase-contrast images were repeated with correct encoding velocity if aliasing occurred. CMR

images were transmitted to a dedicated workstation (MR WorkSpace 2.6.3.2; Philips Medical Systems, Best, The Netherlands), and the quantitative flow images were analyzed. Quantitative blood flow measurements (Figure 3, B' and C') were generated for each 2-dimensional cut plane by tracing the endothelial border of the corresponding vessel to create a region of interest encompassing the lumen. Biventricular function was measured using the short-axis stack with balanced steady-state free precession sequences. Regions of interest were created by tracing the endocardial and epicardial LV borders for each patient at end-systole and end-diastole. Quantitative data analysis inherent to blood flow, end-diastolic and end-systolic volumes, stroke volume, and ejection fraction was performed using MR WorkSpace 2.6.3.5 (Philips Medical Systems).

BMR Imaging Protocol

BMR was performed using a 3.0-T magnetic resonance imaging (MRI) scanner (Achieva; Philips Medical Systems) with an 8-channel head-array receiving coil for sensitivity encoding parallel imaging. Arterial spin labeling (ASL) sequences were acquired to evaluate cerebral blood flow (CBF). T1-weighted, fluid-attenuated inversion recovery, and diffusion-weighted MRI sequences were acquired to exclude brain tumors and previous and/or acute ischemic lesions. A radiologist with a 15 years of experience in neurologic imaging evaluated these sequences of each examination.

ASL Perfusion MRI

The MRI parameters for the ASL sequence were as follows: repetition time, 4000 ms; echo time, 20 ms; flip angle, 40°; sensitivity encoding

FIGURE 3. A, Volume rendering technique reconstructions of a contrast-enhanced magnetic resonance angiography study. B-E, Through-plane phase-contrast images acquired in a short-axis plane in the ascending aorta (1 cm above the sinotubular junction; yellow line) (B), at the ventricular portion of the conduit (blue line) (C), and at the descending thoracic aorta (3 cm proximal and distal to the conduit-aortic anastomosis; white lines) (D and E). B' and C', Flow-time graphs (mL/s) obtained from the phase-contrast image analysis showing that approximately two-thirds of ventricular outflow was directed through the valved conduit (C') and one-third was directed through the native aortic valve (B').

FIGURE 4. Arterial spin labeling maps obtained in the same patient before and after surgery describe the evolution of cerebral blood flow values calculated at the gray matter, dorsal default mode network, and sensorimotor levels. As shown on the color maps, there were no significant differences between the preoperative and I^2_{PC} cerebral blood flow in any of the 3 evaluated districts. CBF, Cerebral blood flow.

factor, 2.3; field of view, 24×24 cm; matrix, 68×68 ; in-plane resolution, 3.53×3.53 mm; and section thickness/gap, 6/2 mm. The first labeling delay time (TI_1) of the multiple time point ASL sequence was 40 ms; 7 different phase data were acquired at 7 different labeling delay time points every 250 ms ($\Delta TI = 250$ ms). A total of 49 images, labeled subtracted from nonlabeled images, were obtained. The labeling slab thickness was 150 mm; it was positioned at the level of the upper cervical region. The total acquisition time was 5 minutes and 52 seconds. The ASL imaging data were transferred to an off-line workstation and analyzed with software for analysis and visualization of MRI used for constructing CBF maps (Figure 4). CBF values were calculated at the gray matter, dorsal default mode network (DMN), and sensorimotor levels.

TABLE 1. Baseline characteristics

Parameter	Value
Number of patients	8*
Age, y, mean $\pm$ SD	78.4 ± 4.4
BSA, m^2 , mean $\pm$ SD	1.74 ± 0.15
Preoperative cardiac index, L/min/ m^2 , mean $\pm$ SD	2.6 ± 0.89

SD, Standard deviation; BSA, body surface area. *Eight patients underwent preoperative and early brain magnetic resonance imaging (BMR) control. Only 6 patients also underwent mid-term BMR control, because 2 patients died in the meantime.

Statistical Analysis

Continuous variables are expressed as mean $\pm$ standard deviation or median and interquartile range, and categorical variables are expressed as frequency and percentage. Continuous variables were compared using the Student independent t test or Mann–Whitney nonparametric test, and categorical variables were compared using the χ^2 or Fisher exact test. Agreements between measurements were explored using the Lin concordance correlation coefficient (CCC). The significance level of all analyses was set at $P < .05$. Statistical analyses were performed with MedCalc statistical software.

CBF values were compared using a 2-tailed t test to calculate statistical significance.

RESULTS

Patient Characteristics and Study Protocol

Ten high-risk patients (6 men, mean age, 79.4 ± 5.8 years) with symptomatic severe AS candidates to surgical treatment of AS with beating-heart AVB insertion without CPB underwent imaging with CMR and BMR. CMR and BMR were performed before surgery (mean, 2.5 days) and then after surgery for the early (mean, 11.7 ± 2.14 days) and mid-term (mean, 253.5 ± 31.4 days) postoperative control. Two patients were excluded from BMR owing to the presence of devices noncompatible with a 3.0-T MRI scanner. Consequently, 8 of 10 patients underwent preoperative and early BMR. After 8.4 months, 8 patients underwent mid-term CMR, and only 6 patients underwent mid-term BMR. Two patients died from non–cardiac-related causes, 1 due to cholangiocarcinoma at 195 days after surgery and the other from necrotic hemorrhagic pancreatitis at 193 days after surgery. Demographic and hemodynamic data, as well as the clinical presentation, of the cohort are summarized in Table 1. According to CMR direct preoperative planimetry, the average aortic valve area was 0.71 ± 0.19 cm^2 ; no aortic planimetry assessments were performed in postoperative controls. According to CMR quantitative blood flow measurements, peak velocity across the NAV was 4.2 ± 0.4 m/s in the preoperative evaluation, 1.6 ± 0.5 m/s in the early postoperative control evaluation, and 1.5 ± 0.3 m/s in the mid-term postoperative control evaluation.

TABLE 2. Comparison of left ventricular function before and after surgery

Parameter	Preoperative	Early postoperative control (mean, 11.7 ± 2.14 d)	Mid-term postoperative control (mean, 253.5 ± 31.4 d)
Number of patients	10	10	8*
LVSF, mL, mean ± SD	64.2 ± 18.3	56.25 ± 13.2	66.8 ± 18.6
LVEF, %, mean ± SD	54.6 ± 14.1	51.8 ± 8.4	55.5 ± 12.9
LVEDV, mL, mean ± SD	121.1 ± 28.9	106.5 ± 23	135 ± 55
LVESV, mL, mean ± SD	56.8 ± 26.9	51.1 ± 15.2	60.6 ± 36

LVSF, Left ventricular stroke volume; SD, standard deviation; LVEF, left ventricular ejection fraction; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume. *Two deaths occurred from noncardiac causes, cholangiocarcinoma and necrotic hemorrhagic pancreatitis, at 145 and 193 days after surgery, respectively.

Comparison of LV Morphology and Function Before and After Surgery

Biventricular stroke volume, ejection fraction, end-diastolic volume, and end-systolic volume before and after AVB are summarized in Table 2. No variations in LV stroke volume and LV ejection fraction were identified using a modified Simpson method ($P = .435$) on postoperative CMR.

Relative Blood Flow Distribution After AAC Insertion

LV outflow was distributed between the AAC (early, 39.2 ± 5 mL; mid-term, 45.8 ± 4 mL) and the NAV (early, 22.5 ± 3 mL; mid-term, 21.7 ± 2 mL). Cardiac output through the NAV was significantly reduced after AVB (preoperative NAV flow 58 ± 18 mL; $P < .0001$). Conduit flow (expressed as percentage of total anterograde flow passing through the conduit) was $63.5 \pm 8\%$ in the early and $67.8 \pm 7\%$ in the mid-term control.

There was no evidence of significant conduit valve regurgitation. Retrograde perfusion from the level of the conduit insertion in the descending aorta backward to the aortic arch accounted for 6.9% of total cardiac output and 11% of total conduit flow.

Cerebral Perfusion (ASL)

There were no statistically significant differences between the preoperative and I°_{PC} CBF at the gray matter ($P = .20$), dorsal DMN ($P = .60$), and sensorimotor ($P = .28$) levels (Figure 4). No significant difference was

found between preoperative and the mid-term CBF at the gray matter ($P = .32$), dorsal DMN ($P = .31$), and sensorimotor ($P = .74$) levels and between early and mid-term CBF at the gray matter ($P = .92$), dorsal DMN ($P = .36$), and sensorimotor ($P = .30$) levels (Table 3 and Figure 4). No patient had a clinical stroke. There were no changes between the preoperative and first/second postoperative FLAIR and DWI sequences.

Reproducibility

The CMR and BMR measurements were repeated twice after the examination by the same radiologist. Each method had similarly high intraobserver reproducibility (CCC = 0.91 and 0.90, respectively). To investigate the interobserver variability of CMR and BMR measurements, a second observer performed the measurements blinded to the results reported by the first observer. Interobserver reproducibility was excellent (CCC = 0.92 for the CMR and 0.94 for the BMR).

DISCUSSION

An analysis of our present findings suggests that CBF after insertion of an AVB conduit derives mostly from antegrade blood flow across the NAV rather than retrograde flow through the conduit. We observed that 63.5% of ventricular outflow was directed through the valved conduit, compared with 36.5% through the NAV. We also found that retrograde blood flow in the descending aorta above the level of the conduit insertion accounted for <7% of total cardiac output

TABLE 3. CBF values

Evaluated districts	Preoperative, (mL/100 mL)/min	Early postoperative control (8 patients*; mean, 11.7 ± 2.14 d), (mL/100 mL)/min	Mid-term postoperative control (6 patients; *mean, 253.5 ± 31.4 d), (mL/100 mL)/min
Gray matter, mean ± SD	35.7 ± 11.3	39.5 ± 9.9	39.7 ± 4.6
Dorsal DMN, mean ± SD	47.2 ± 11.4	49.5 ± 14.5	54.3 ± 10.6
Sensorimotor, mean ± SD	59.3 ± 26.3	64.9 ± 22.6	56.7 ± 10.9

SD, Standard deviation; CBF, cerebral blood flow; DMN, default mode network. *8 patients underwent preoperative and early BMR control; only 6 patients also underwent mid-term BMR control, because 2 patients died in the meantime.

and approximately 11% of total conduit flow. Our results are consistent with those of Stauffer and colleagues,¹⁸ who reported that 65% of ventricular outflow is directed through the valved conduit and 35% is directed through the NAV with an 8% of total cardiac output as retrograde flow in the descending thoracic aorta. These findings are in line with previous results of computational modeling of AVB fluid dynamics, which demonstrated the presence of a small degree of retrograde flow in the descending aorta above the insertion of the valved conduit for larger-diameter conduits.²⁰ In that study, all CBF derived from antegrade flow in the ascending aorta. Importantly, the magnitude of CBF was unchanged before and after insertion of the AVB conduit. We also confirmed a postoperative preserved LV function despite removal of the apical myocardial plug and insertion of a rigid connector into the LV apex.

One of the most widely debated issues in AVB surgery is the possibility of flow subtraction with cerebral hypoperfusion due to a possible flow competition between the 2 ventricular outputs.²¹ Concerning this, we did not find any significant difference between preoperative and postoperative CBF at the gray matter ($P = .20$), dorsal DMN ($P = .60$), and sensorimotor ($P = .28$). This represents an important point in favor of the safety of this surgical choice.

Another concern expressed in the literature is the possible development of thrombosis at the level of flow collision.²² Takeda and colleagues²³ reported thrombus formation in patient submitted to AVB whose degree of AS was very severe; the authors hypothesize that a very low cardiac output through a very narrowed LV outflow tract or a very severe AS might mimic what usually happens in patients with an LV assist device in which the mechanical circulatory support takes over the whole cardiac output. In our experience, notwithstanding the severity of NAV stenosis, we did not observe any thrombus formation either at the level of Valsalva sinuses or inside the LV. Ultimately, a possible risk of aortic thrombosis at the level of flow collision at the proximal portion of the descending thoracic aorta has been reported. Even if we do not have continuous recordings that can demonstrate the time course of the flow partition, we hypothesize that that collision point is not fixed, but moves toward and backward related to the physiological increases in cardiac output and peripheral demand. Regarding such a complication, we agree with the comment published by Yavuz and colleagues²⁴ confirming that the probability of developing it may be inversely related to preexisting cardiac function.

CONCLUSIONS

The evidence reported here demonstrates that LV function is preserved after the implantation of the AAC at the level of the LV apex, and that cardiac output is distributed between the 2 LV outputs as described previously.¹⁸ Assessment of cerebral perfusion confirms that the blood

flow distribution between the 2 LV outlets did not impair blood supply to the brain. These observations confirm that AVB surgery represents a valuable complementary approach to conventional AVR or TAVI in selected patients.

Conflict of Interest Statement

Authors have nothing to disclose with regard to commercial support.

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Key Words: aortic valve bypass surgery, apico-aortic conduit, severe aortic stenosis, cardiovascular magnetic resonance, cerebral perfusion

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