evolution of the autograft (HR: 1.70; 95% CI: 0.97 to 2.98; p = 0.06).

The year of the surgery (accounting for changes in surgical techniques and medical therapies) was significantly associated with worse graft evolution (p = 0.001) in both models; however, it did not change the results. Considering reoperations and deaths in competitive-risks regression models also gave similar results.

In this series of patients who underwent a Ross operation, an increase in both systemic and pulmonary arterial pressures were found to be independent risk factors for autograft and homograft degeneration, respectively. These results suggested that aggressive blood pressure control should be initiated post-operatively and prolonged throughout the life of the patient to reduce the risk of potential reoperations. We hypothesized that the mechanism through which the degeneration occurs was linked to the previously demonstrated autograft dilation. The association between ascending aorta prosthesis and fast autograft degeneration could be related to an active process through which Dacron prostheses could alter the arterial tension–compliance relationship or be a passive marker of structural collagen fiber impairment. Further studies will be required to evaluate these hypotheses. Nonetheless, Ross procedure patients showed excellent and highly encouraging results with both grafts even after >20 years of follow-up. With increasing knowledge regarding the evolution of autografts and homografts, we hope to better target patients who will benefit the most from the procedure and to improve post-operative management of patients.

Louis Simard, BSc
Jean Perron, MD
Mylène Shen, MSc
Lionel Tastet, MSc
Siamak Mohammadi, MD
Marine Clisson, BSc
Anthony Poulin, MD
***Marie-Annick Clavel, DVM, PhD**

*Institut Universitaire de Cardiologie et de Pneumologie de Québec
Quebec Heart and Lung Institute
2725, Chemin Sainte-Foy, #A-2047
Québec, QC G1V 4G5
Canada
E-mail: Marie-Annick.Clavel@criucpq.ulaval.ca

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REFERENCES

1. Nishimura RA, Otto CM, Bonow RO, et al. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2014;63:e57–185.

2. David TE. Reoperations after the Ross procedure. Circulation 2010;122:1139–40.

3. Ryan WH, Prince SL, Culica D, Herbert MA. The Ross procedure performed for aortic insufficiency is associated with increased autograft reoperation. Ann Thorac Surg 2011;91:64–9.

4. Lancellotti P, Tribouilloy C, Hagendorff A, et al. European Association of Echocardiography recommendations for the assessment of valvular regurgitation. Part 1: aortic and pulmonary regurgitation (native valve disease). Eur J Echocardiogr 2010;11:223–44.

The Time-Varying Risk of Cardiovascular and Noncardiovascular Readmissions Early After Acute Myocardial Infarction

Twenty percent of patients who experience a myocardial infarction (MI) will be readmitted within 30 days of discharge (1,2). Previous studies have suggested that the timing of readmission after MI by cause is relatively uniform (2). Because hospital reimbursement and payment structures continue to change and move toward bundled payments (3), understanding when and why patients are at greatest risk for readmission is critical to help provide quality care and manage costs. We sought to further evaluate the risk of readmission over time by cause after MI.

We retrospectively identified all patients discharged alive from our main hospital with a principal diagnosis of MI from April 2008 until June 2012 using discharge International Classification of Disease-9 (ICD-9) diagnosis codes (ICD 410 to 410.9). All readmissions to our health system within 3 months were identified using our institutional billing system. Readmissions outside of our health system were not available and not included in the analysis. The primary cause of readmission was identified using principal diagnosis billing codes and categorized into 4 groups: MI-related, other cardiovascular (CV)–related, non–CV-related, or planned. Time-varying instantaneous risk of readmission was estimated for all readmissions and readmissions by cause using a nonlinear parametric temporal decomposition model (4).

The study cohort consisted of 3,069 patients discharged alive after an index MI. Within 3 months of the index MI, there were 494 readmissions. Forty-seven percent (232 of 494 patients) were either

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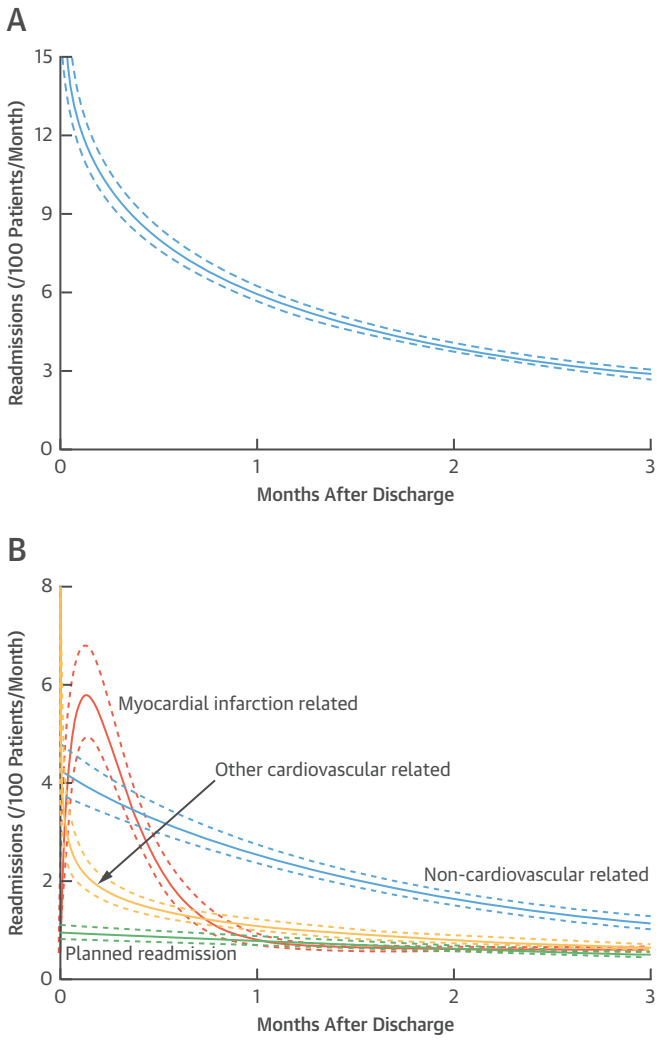
FIGURE 1 Risk of Readmission After Myocardial Infarction Over Time



(A) Instantaneous risk of all-cause readmission after myocardial infarction (MI) and readmission after MI by **(B)** cause enclosed within **dashed** 68% confidence bands equivalent to 1 SE. MI-related **(orange lines)**; noncardiovascular-related **(blue lines)**; other cardiovascular-related **(yellow lines)**; and planned readmission **(green lines)**.

MI- or other CV-related. Thirty-nine percent (191 of 494 patients) occurred within the first 15 days. The instantaneous risk of readmission after MI was highest immediately after discharge and then dropped rapidly early in the post-discharge period (Figure 1A). The risk of MI-related and other CV-related readmissions was highest immediately after discharge, and after approximately 15 days, non–CV-related causes posed the highest risk for readmission (Figure 1B). During the second and third months after discharge, the risk of readmission was substantially lower and remained relatively constant.

Our findings contrasted with previous studies that suggested that the causes of readmission after MI did not vary substantially over time (1,2). Our results demonstrated that both the risk and primary cause of readmission changed dynamically over time. Nearly 40% of readmissions within 90 days occurred within the first 15 days, and these readmissions were predominantly CV-related. This finding suggested that the factors that led to these readmissions were likely embedded within the index hospitalization. Readmission prevention strategies should thus begin on the day of admission and not on the day of discharge. As pressure builds to decrease length of stay to manage costs, premature discharge could lead to an increased rate of readmission (5), paradoxically increasing overall costs for a hospital system. Furthermore, as payment bundles for MI might soon extend to 90 days (3), our study was one of the first to address the risk and cause of readmission in the 31- to 90-day window. Our finding of overwhelming non-CV readmissions in this time highlighted the challenges for cardiologists in reducing the rate of these readmissions. Close collaboration with other clinical teams, such as primary care and internal medicine specialists dealing with chronic disease processes, might be needed to affect these readmissions. We acknowledged limitations inherent to a single-center retrospective study.

The risk of readmission after MI was highest immediately after discharge, particularly for CV-related readmissions and dropped by almost one-half within 15 days of discharge. Efforts to prevent readmission need to account for this change in risk over time and prevention strategies should begin early during the index admission. A more nuanced approach based on the risk and cause of readmission over time might be a more preferred method to improve quality of MI care and to hold hospitals accountable for readmissions after MI.

*Umesh N. Khot, MD
Michael J. Johnson, MD
Ashley M. Lowry, MS
Jeevanantham Rajeswaran, PhD
Samir Kapadia, MD
Mehdi H. Shishehbor, DO, MPH, PhD
Venu Menon, MD
Stephen G. Ellis, MD
Pamela Goepfarth, RN, MBA
Eugene H. Blackstone, MD

*Heart and Vascular Institute Center for Healthcare Delivery Innovation J2-4
Cleveland Clinic
9500 Euclid Avenue
Cleveland, Ohio 44122
E-mail: khotu@ccf.org

http://dx.doi.org/10.1016/j.jacc.2017.06.043

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REFERENCES

1. Dunlay SM, Weston SA, Killian JM, Bell MR, Jaffe AS, Roger VL. Thirty-day rehospitalizations after acute myocardial infarction: a cohort study. Ann Intern Med 2012;157:11–8.

2. Dharmarajan K, Hsieh AF, Lin Z, et al. Diagnoses and timing of 30-day readmissions after hospitalization for heart failure, acute myocardial infarction, or pneumonia. JAMA 2013;309:355–63.

3. Notice of proposed rulemaking for bundled payment models for high-quality, coordinated cardiac and hip fracture care. Center for Medicare and Medicaid Services, 2016. https://www.cms.gov/Newsroom/MediaReleaseDatabase/Fact-sheets/2016-Fact-sheets-items/2016-07-25.html. Accessed April 27,2017.

4. Blackstone EH, Naftel DC, Turner ME. The decomposition of time-varying hazard into phases, each incorporating a separate stream of concomitant information. J Am Stat Assoc 1986;81:615–24.

5. Kociol RD, Lopes RD, Clare R, et al. International variation in and factors associated with hospital readmission after myocardial infarction. JAMA 2012;307:66–74.

Coronary Artery Disease Affects Symptomatology of Aortic Valve Stenosis

Although the exact pathogenesis of aortic valve stenosis (AVS) is incompletely resolved, it is becoming increasingly clear that a complex interplay between genetic predisposition and environmental factors drives progressive mineralization and fibrosis of the aortic valve (1). Cross-sectional observational studies have shown that AVS shares some risk factors with atherosclerosis. In addition, the metabolic syndrome and hypertension have been associated with the progression rate of AVS (2). In a recent article published in the *Journal*, Yan et al. (3) performed a "big data" study among 1.12 million individuals, of whom 20,995 people developed severe AVS during a median follow-up of 13 years, indeed an impressive study for which the authors need to be commended. The authors show that classical cardiovascular risk factors (hypertension, diabetes, and dyslipidemia) were independently associated with incident severe AVS, which was defined as hospitalization for AVS and/or surgical or transcatheter aortic valve replacement (AVR) (3). Current clinical guidelines recommend AVR in symptomatic patients with severe AVS (4,5). However, symptomatology is driven by a tradeoff between the severity of AVS and coronary artery disease (CAD). Patients with CAD can already become symptomatic from moderate AVS, whereas AVS patients without CAD can remain asymptomatic or paucisymptomatic despite having severely elevated transaortic gradients. In addition, among CAD patients undergoing coronary bypass surgery, concomitant AVR is sometimes performed for moderate AVS. In the latter case, the surgical indication is driven by CAD, not by AVS severity. Thus, CAD may confound the association between the classical risk factors and AVS treatment indications. It would therefore be helpful if the authors could provide in future work additional clinical data on the AVS hospitalizations and treatments that composed the primary outcome of their study. First, to what extent did the patients with risk factors undergoing AVS treatment require concomitant revascularization? Second, following stratification of patients with and without CAD, are hypertension, diabetes, and dyslipidemia still associated with AVS in different subgroups? Such additional clinical data would enhance our understanding and could shed further light on the association between these risk factors and AVS in patients with and without CAD.

***S. Matthijs Boekholdt, MD, PhD**
Benoit J. Arsenault, PhD
Patrick Mathieu, MD, MSc

*Academic Medical Center
Department of Cardiology, Room F3-239
PO Box 22660
1100 DD Amsterdam
the Netherlands
E-mail: s.m.boekholdt@amc.uva.nl

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REFERENCES

1. Bosse Y, Mathieu P, Pibarot P. Genomics: the next step to elucidate the etiology of calcific aortic valve stenosis. J Am Coll Cardiol 2008;51:1327–36.

2. Capoulade R, Clavel MA, Dumesnil JG, et al. Impact of metabolic syndrome on progression of aortic stenosis: influence of age and statin therapy. J Am Coll Cardiol 2012;60:216–23.

3. Yan AT, Koh M, Chan KK, et al. Association between cardiovascular risk factors and aortic stenosis; the CANHEART aortic stenosis study. J Am Coll Cardiol 2017;69:1523–32.

4. Nishimura RA, Otto CM, Bonow RO, et al. 2017 AHA/ACC focused update of the 2014 AHA/ACC guideline for the management of patients with valvular heart