investment was necessary. The impact of those strategies was analyzed by multivariate logistic regression (unpublished data). Risk-adjusted hospital mortality declined (from 11.5% to 3.6%, $P = .003$) after these strategies were implemented. Survival benefit was particularly evident in high-risk patients (Euroscore > 7), in which operations performed prior to the quality program were independently associated with greater mortality (odds ratio = 4.8; 95% confidence interval 1.6–17.4; $P = .005$). There was no difference in low-risk patients (Euroscore < 6). Therefore, multimodal strategies in our center remarkably improved the outcomes of high-risk cardiac surgery patients, as opposed to low-risk patients, which are probably less dependent on the system.

Quality in cardiac surgery is ultimately a surgeon's responsibility and should be part of the training of young surgeons. We would like to congratulate Dr Stamou and colleagues on their excellent work on an extremely important subject.

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References

1. Stamou SC, Camp SL, Stiegel RM, Reames MK, Skipper E, Watts LT, et al. Quality improvement program decreases mortality after cardiac surgery. *J Thorac Cardiovasc Surg.* 2008;136:494-9.
2. Ribeiro ALP, Gagliardi SPL, Nogueira JLS, Silveira LM, Colosimo EA, Nascimento CAL. Mortality related to cardiac surgery in Brazil, 2000–2003. *J Thorac Cardiovasc Surg.* 2006;131:907-9.

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Reply to the Editor:

We appreciate Dr Atik's kind comments and thoughtful questions. Additionally, his efforts are to be recognized and commended. Our quality improvement program (QIP)^{1,2} was developed to address opportunities to reduce mortality and mitigate the risk of

major morbidity (nosocomial infections, acute renal failure, hemorrhage, reoperations, etc) through elimination of variation while reliably applying the best available evidence as well as best practices. Regarding the comment about severity scores, The Society of Thoracic Surgeons (STS)—National Cardiac Database allowed us to compare our performance to national averages and guided our efforts. STS developed definitions for key data elements, providing a common language and standards. STS also developed risk-adjusted mortality and morbidity algorithms to provide benchmark measures that helped in the construction of our standard operating procedure. STS risk scores were routinely recorded in our QIP efforts and used to calculate expected mortality and compare it with the national average. However, reporting risk scores was beyond our focus. The most important finding of our article was that by building a QIP, a measurable and significant decrease in mortality can be accomplished, and on that regard, our article was among the first to report such a finding.

We tend to disagree with Dr Atik's comments that QIPs follow institutional or regional/national initiatives. A QIP can follow both national/regional initiatives and at the same time be tailored to each institution's needs and particularities. Our QIP was structured based on STS guidelines and best evidence practices but was also adjusted to our institution's particular goals.

An important finding of our work is that a QIP did not improve dramatically the outcomes on diabetic patients, as previously reported.³ Similarly, Van Den Berghe and colleagues³ demonstrated that with their intensive insulin therapy protocol, they were able to reduce mortality of all medical/surgical intensive care unit patients, except those with a prior history of diabetes. Our investigation corroborated this finding that diabetics are a high-risk subset of patients on which future QIP efforts should focus. The presence of small target vessels (diffuse coro-

nary artery disease) frequently encountered in diabetic patients, the higher risk for wound infection, and the higher risk of mortality compared with nondiabetic patients as well as the resistance of diabetic patients to improve with QIP initiatives provided the rationale for our proposition.

We tend to agree with Dr Atik's statement that "because processes of care vary widely among cardiac surgery programs, there are no 'recipes' that can be universally applied, due to system-based particularities." However, common denominators of successful QIP efforts include a dedicated multidisciplinary team with common goals, a systems-based approach, accurate communication, adherence to strict protocols, and monitoring compliance. In this respect, some universally applied rules may apply. Regarding the reasons for the change in outcomes, we believe there is a multifactorial etiology: mainly the absence of a dedicated multidisciplinary team, the lack of a monitoring tool to measure compliance to the protocols, and individualized rather than a team approach to clinical problem solving during the initial period before development of QIP. In contradistinction, our systems-based approach, standardization, team building, consistent and accurate communication, and active management of change and quality accounted for a recent decline of 48% in mortality.

The financial impact (investment and savings) of QIP is indeed crucial. We previously demonstrated that by implementing QIP, a significant increase in the early extubation rates, a decrease in intensive care unit stay, and resource utilization can be accomplished.² We are in the process of analyzing the specific financial impact of our QIP, and this will be the focus of a future publication. We believe that by using a unique database linking clinical and financial outcomes through a focus on quality, cost containment in cardiac surgical care can be achieved by identifying and

reducing costly complications, improving efficiencies of care, and reducing resource utilization.

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References

1. Stamou SC, Camp SL, Stiegel RM, et al. Quality improvement program decreases mortality after cardiac surgery. *J Thorac Cardiovasc Surg.* 2008;136:494-9.
2. Turner SL, Stamou SC, Stiegel RM, et al. Quality improvement program increases early tracheal extubation and decreases pulmonary complications and resource utilization after cardiac surgery. *J Card Surg.* In press.
3. Van den Berghe G, Wilmer A, Milants I, Wouters PJ, Bouckaert B, Bruyninckx F, et al. Intensive insulin therapy in mixed medical/surgical intensive care units: benefit versus harm. *Diabetes.* 2006;55:3151-9.

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TRANSMISSION OF HUMAN T-LYMPHOTROPIC VIRUS TYPE I BY BILATERAL LIVING-DONOR LOBAR LUNG TRANSPLANTATION To the Editor:

Human T-lymphotropic virus type I (HTLV-I) is an etiologic agent for adult T-cell leukemia/lymphoma and HTLV-I-associated myelopathy/tropical spastic paraparesis. Most HTLV-I infections are attributable to transmission from mother to child or to sexual contact later in life, whereas transfusion is perhaps the most efficient mode of viral transmission. Seroprevalence of HTLV-1 among high-risk populations is 10% to 30% in the

southern area of Japan, such as Okinawa, and approximately 5% in the Caribbean region compared with 1% in Europe and the United States.

There have been several reports of HTLV-1 transmission in association with organ transplantation (Table 1).¹⁻⁴ However, no report describes a transmission of HTLV-1 by lung transplantation. We describe the first case of HTLV-1 transmission from a seropositive donor via lung transplantation.

A 38-year-old woman had abnormal shadows on a chest x-ray at a healthcare examination. Video-assisted thoracoscopic surgical lung biopsy was performed, and usual interstitial pneumonia was diagnosed. Despite some drug treatments, she had dry cough and dyspnea that gradually worsened. In 2003, at the age of 42 years, she was considered eligible for bilateral living-donor lobar lung transplantation at Okayama University. The patient was negative for anti-HTLV-I antibody. The younger sister was positive less than 16 times for anti-HTLV-I antibody. Because the patient's condition was severe and deteriorating, she was considered to be in urgent need of lung transplantation. In addition, there are no reports of HTLV-I transmission by lung transplantation. Therefore, lung transplantation was performed using lungs from an elder brother and a younger sister in August 2003. Before the transplant, the lungs were flushed with 1 L of Euro-Collins solution, which consists of 115 mmol/L monopotassium, 10 mmol/L sodium, and 3.6% glucose, both antegradely and retrogradely.⁵

The patient's general condition improved after the lung transplantation. In November 2008, her oxygen saturation was 98% to 99% at room air. However, serologic follow-up demonstrated that she had acquired HTLV-I infection posttransplantation. Antibody titers were as follows: 256 times after 9 months, more than 8192 times in August 2006, 2048 times in April 2008.

Remesar and colleagues¹ reported the first case of HTLV-1 infection transmitted by organ transplantation (a child who received a kidney from her infected mother). The seroconversion was observed 83 days after transplantation. Nakatsuji and colleagues² described a kidney transplant recipient who presented with tropical spastic paraparesis 4 years posttransplantation. Toro and colleagues³ reported the transmission of HTLV-1 from a single donor to 3 organ (2 kidneys and 1 liver) transplant recipients, who subsequently developed tropical spastic paraparesis within 2 years posttransplant.

Although the lungs were pretreated with preservative solution, transmission of HTLV-1 could not be prevented. Approximately 5 years posttransplant, our patient demonstrates neither neurologic nor hematologic evidence of HTLV-I-related disease. However, this patient should be followed up for adult T-cell leukemia/lymphoma and tropical spastic paraparesis.

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TABLE 1. Review of the literature about transmission of human T-lymphotropic virus type I after solid-organ transplantation

First author	Year	Age and sex	Organ	Clinical features	References
Remesar	2000	Daughter	Kidney	None	1
Nakatsuji	2000	50 M	Kidney	HTLV-1-associated myelopathy	2
Toro	2003	53 F*	Kidney	Subacute myelopathy	3
Toro	2003	55 M†	Kidney	Subacute myelopathy	3
Toro	2003	44 F‡	Liver	Subacute myelopathy	3
Zarranz	2003	54 F*	Kidney	Myelopathy	4
Zarranz	2003	57 M†	Kidney	Myelopathy	4
Zarranz	2003	44 F‡	Liver	Myelopathy	4
Yara	2008	42 F	Lung	None	Present report

HTLV, Human T-lymphotropic virus type I. *, †, ‡The same patients.