Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

decades since the introduction of the lung allocation score, it is still not optimized.

We agree with the authors and discuss it in our original paper that the different strategies to address increased calculated panel reactive antigen patients in other transplant disciplines has had limited success. Furthermore, desensitization protocols represent a valid alternative for some patients, and in some hands, they have shown success. However, current desensitization protocols lack rigorous validation and consensus remains elusive for a specific, safe, well-tolerated, durable, and effective desensitization protocol in lung transplantation, making it impossible for ubiquitous application across all patients.

Nonetheless, we do believe that the current allocation method should be challenged and optimized for all recipients. We are encouraged by the ongoing efforts of the Organ Procurement and Transplant Network to develop a transparent and objective allocation system based on continuous distribution that takes in to account disadvantaged groups of recipients. We hope that these efforts lead not to broader geographic sharing, but rather to a smarter geographic sharing of our national donor resources. This smarter sharing will balance clinical acuity with many other important aspects of transplantation and minimize disparities in organ allocation.

We are grateful for the authors' interest and concern for our mutual patients, and we urge the transplant community to continue developing a fair and equitable allocation system with policy while we continue to work on the same goal through medical advances such as desensitization.

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MISCELLANEOUS

COVID-19 Disease and Cardiac Surgery: Reciprocal Interrelations

TO THE EDITOR: The interesting paper by Fukuhara and colleagues[1] merits some patho-physiologic observations. After surgery, the innate immune system weakens in its cell and humoral components for an increased release of cortisol and catecholamines, and for anesthetic-analgesic medications. This involves preferentially cardiac surgery, mainly if performed with cardiopulmonary bypass and hypothermia, as a consequence of the ischemia-reperfusion injury, that, followed by a great release of reactive oxygen species and proinflammatory cytokines, electively damages the pulmonary capillaries and increases their permeability.[2] In COVID-19 infection these structures are subjected to an "endotheliitis," consisting in infiltration by neutrophils and mononuclear cells and a consequent thrombosis.[3,4] It explains the great incidence of pulmonary complications, until a severe pneumonia, in the course of this viral infection, preexisting or occurring in the early postoperative period.[1] COVID-19 disease can evolve toward a "hypersensitive vasculitis," where a dysregulated adaptive immunity causes an abnormal production of immunoglobulins and antibodies, that, infiltrating the arterial walls of small/medium sized vessels, trigger an "immunothrombosis," with the subsequent danger of organ malperfusion or acute ischemia.[4] This dangerous condition affects cardiac surgery more often, especially in case of postoperative low cardiac output syndrome. Moreover, in severe COVID-18 disease, platelets, activated to repair endothelia, perform a "covercytosis" against virions, in agreement with their myeloid lineage and direct origin from megakaryocytes modified in their genome with mRNA fragments derived from the SARS-CoV-2ZN1 gene. They demonstrate also a greater power of aggregation and adhesion to fibrin filaments, and favor production of microcomplexes with neutrophils, monocytes and T lymphocytes.[5] This pathology not only affects the pulmonary microvessels, but also, in the COVID-19 hypercoagulative milieu, causes thromboembolic complications in medium/large sized vessels, differently expressing, according to their hemodynamics, from mural thrombi, occasionally discovered, to a complete thrombosis. In surgery, all this increases the risk of thrombo-embolic events, and after a cardiopulmonary bypass specifically affects the pulmonary vessels and further deranges the hemostatic system. In perspective, we have to consider another complication of COVID-19 infection, consisting of an intramural hematoma, caused by endothelial ulceration and necrotic-inflammatory lesions of the tunica media, evolving in an acute dissection, if not protectively restrained and

converted into a mural thrombus by activated platelets and thanks to a hypercoagulative state.

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COVID-19 and Aortic Dissections: Collaboration (Among Disciplines and Centers) Is Key

REPLY TO THE EDITOR: We thank Manenti and colleagues[1] for their insightful comments concerning the pathophysiology, in particular, the immune dysregulation and vascular inflammation, of coronavirus disease 2019 (COVID-19) and its implications for the management of patients with concomitant cardiac surgical emergencies, such as an acute type A aortic dissection (ATAAD).

Our understanding of COVID-19, a complex and multiform disease process, is still incomplete but rapidly evolving. Since our report[2] in April of a patient with both ATAAD and COVID-19 and an unfortunate dismal outcome, we know that cardiac surgery centers worldwide have faced the similar challenge of choosing and delivering the "best" treatment strategy for patients with a combination of these two deadly diseases.

In examining our experience at the University of Michigan and the experiences of those of Wuhan and Changsha (China), we found that some patients with both ATAAD and COVID-19 can still survive and recover with operative management.[3] Similarly, our colleagues from Yale[4] and Ghent (Belgium)[5] showed that operative management of ATAAD in COVID-19 patients can be successful. These collective outcomes have made us hopeful that our persistent efforts to learn about and adapt to the current pandemic will help ensure the well-being of our patients.

It is important to recognize that strategies addressing the notable increased risk of proinflammatory features associated with cardiopulmonary bypass in COVID-19 patients undergoing cardiac surgery, as described by Dr Manenti, have been proposed, primarily attempting to mitigate the adverse effects of hypercoagulability and hyperinflammation. As a result of the current limited clinical data regarding the optimization of such strategies, collective sharing of information will be more crucial than ever. There is no doubt that this pandemic has imposed a heavy toll around the world. We hope that with continued scientific investigations and sound public health policies, it will be under control soon. We strongly believe that collaboration among centers and different specialties is key to achieving this goal.

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The Risk of Reintervention of the Trifecta Bioprosthesis

TO THE EDITOR: We read with great interest the article by Lam and colleagues[1] addressing the rate of reintervention especially due to structural valve degeneration (SVD) of 3 different bioprostheses, the Carpentier-Edwards Magna Ease (Edwards Lifesciences, Irvine, CA), Trifecta (St Jude Medical, St Paul, MN), and Mitroflow (LivaNova, London, United Kingdom),