a New York Times article describing our work. Dr Anthony Atala has a much better claim to this milestone achievement.

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Reply to the Editor:

We thank Dr Macchiarini for commenting on our editorial published in the *Journal*, and we acknowledge his team's motivation and efforts to advance tracheal replacement.

In his response to our editorial, Paolo Macchiarini refers to several publications, thus undoubtedly convincing many readers of his views. However, in not one of these articles have mortality rates been published. Furthermore, we cannot follow his suggestion to rely on an unpublished article to obtain this information. Nor indeed can we refer to its content, although we have been in a position to read it. However, the unfortunate results after some of the treatments with "bioengineered" tracheas

have reached investigative journalists of *Science*¹ and other media.^{2,3}

More important, the purpose of our editorial was to inform the scientific community that regeneration of a viable trachea resulting from applying bone marrow cells to a decellularized or a synthetic scaffold in the absence of any blood supply is based on hope and belief and not on scientific evidence. None of the publications that Macchiarini cites in his response provide scientific evidence for his claims. We therefore strongly warn against further unethical human experimentation. The ongoing clinical trials will show whether or not this warning was justified.

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MODIFIABLE RISK FACTORS FOR ACUTE KIDNEY INJURY AFTER CORONARY ARTERY BYPASS GRAFTING

To the Editor:

We read with interest the recent article by Ng and colleagues¹ that identified modifiable risk factors for acute kidney injury (AKI) after coronary artery bypass grafting (CABG) in an Asian population. They showed that preoperative anemia and intraoperative lowest hematocrit were potentially modifiable risk factors independently associated with

postoperative AKI. In the design of this study, however, some important data regarding patient perioperative management, such as intraoperative hemodynamic changes, fluid volume, and use of vasoactive medicines, were evidently missing. It has been shown that intraoperative systolic blood pressure decrease relative to baseline is independently associated with postoperative AKI in patients undergoing CABG.² Furthermore, the combination of intraoperative hemodilution anemia and hypotension can synergistically act to increase the risk of AKI after cardiac surgery.³ Campbell and associates⁴ have demonstrated that fluid volume before cardiopulmonary bypass can contribute significantly to intraoperative hemodilution anemia and that restricting fluid volume before cardiopulmonary bypass can attenuate intraoperative hemodilution anemia and decrease the need for transfusion in patients undergoing CABG. In addition, perioperative inotropes, vasopressors, antiarrhythmics, and diuretics may also influence development of AKI after cardiac surgery. We therefore argue that optimizing perioperative management, such as intraoperative avoidance of excess fluid volume, hypotension, and renal arterial vasoconstrictive drugs, should be importantly modifiable factors in decreasing the occurrence of postoperative AKI in patients undergoing CABG. We believe that the results of this study would have been more informative had these factors been taken into account.

Ng and colleagues¹ did not mention the specific timing of postoperative creatinine measurements. It was also unclear whether continuous creatinine measurements were performed. It is therefore difficult to determine whether the cases of AKI reported in this study were due to intraoperative or postoperative factors. Although serum creatinine lags behind acute changes in renal function, AKI (defined by serum creatinine >10 mmol/L greater than normal values)