et al (1) commented specifically on three cases of pulmonary embolism following 31F bicaval dual-lumen (Avalon ELITE; Maquet, Rastatt, Germany) catheter removal which they had seen. In our case series, there were not any clinically demonstrated pulmonary embolism following cannula removal, either with dual lumen bicaval cannulae or single lumen cannulae.

We agree that the prevalence of DVT following ECMO is of great concern, particularly following cannulation of the upper extremities. The risks and the therapeutic implications of DVT of the upper extremities are increasingly recognized (3). Given our experiences and those of others, including Staudacher et al (1), we think that the prevalence of upper extremity DVT following bicaval dual lumen cannulation is likely to contribute to significant morbidity. We would recommend that manufacturers, regulators, or bodies such as the Extracorporeal Life Support Organisation form a prospective registry to better understand the true prevalence and consequences of thromboembolic disease following ECMO cannulation.

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The Richmond Agitation-Sedation Scale Should Not Be Used to Evaluate Neurologic Function

To the Editor:

We were quite astonished to read the article by Vasilevskis et al (1) published in a recent issue of *Critical Care Medicine*. To us, there is no point in using the Richmond Agitation-Sedation Scale (RASS) to evaluate neurologic dysfunction when this scale was developed to adapt sedative therapy (2) and is, therefore, therapy dependent. By contrast, the aim of the Sequential Organ Failure Assessment (SOFA) score (3) is to evaluate the degree of organ dysfunction by using a system that minimizes as much as possible the effect of therapeutic interventions; the notable exception to this

general rule is the cardiovascular component, which uses vasopressor doses, because it is impossible to avoid a treatment-related variable for this system. The SOFA score includes the assumed Glasgow Coma Scale (GCS) score, that is, the GCS that the patient would have in the absence of any sedation, to evaluate neurologic function. If the GCS is assessed as is, without taking into account the effect of sedation, its prognostic value will be skewed. If the GCS score is not mentioned, one should consider that it is 15 of 15.

The RASS not only is a marker of brain dysfunction/damage but can also be just a measure of the degree of sedation. Although the score proposed by Vasilevskis et al may provide similar prognostic information to the GCS, it will vary considerably depending on the sedation practices used in different ICUs. As an example, a GCS score of 3–5 as a result of organic brain injury will certainly have a different prognostic implication than a similar GCS score in a patient receiving deep sedation for agitated delirium.

We acknowledge that people may not always collect the data for the SOFA correctly, but this is not a reason to discard the system. As an analogy, none would suggest discarding the electrocardiogram just because some people cannot read or interpret it! It is obvious that the RASS has prognostic value, as does the use of sedative agents or mechanical ventilation; this is not at all surprising. However, using the RASS to replace the GCS in the evaluation of neurologic status does not make much sense.

Drs. Vincent, Takala, and Moreno were involved in the development of the Sequential Organ Failure Assessment score, which could be conceived as a potential intellectual conflict of interest. Dr. Marshall served as a board member for AKPA Pharma Inc. Dr. Sakr has disclosed that he does not have any potential conflicts of interest.

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