

TABLE 1 Modified Penn Classification of Ischemic Presentations in Patients With Acute Type A Aortic Dissection

| Class | Definition |
|----------------|---|
| Penn Class Aa | Absence of branch vessel malperfusion or circulatory collapse |
| Penn Class Ab | Branch vessel malperfusion with localized malperfusion |
| Subclass Ab-1 | Localized malperfusion without involving critical organs of subclass Ab-2 |
| Subclass Ab-2 | Major cerebral, mesenteric, and coronary malperfusion |
| Penn Class Ac | Generalized malperfusion because of circulatory collapse |
| Penn Class Abc | Both localized and generalized malperfusion |

associated with adverse outcomes following surgical repair (3) (see references 2, 8, and 12 in Chien et al. [5]). Of note, class Aa + Ab-1 (odds ratio [OR]: 0.17, 95% confidence interval [CI]: 0.064 to 0.444; $p < 0.001$) and class Ab-2 + Ac + Abc (OR: 5.94, 95% CI: 2.251 to 15.671; $p < 0.001$) were respectively relieving and exacerbating independent factors for 30-day mortality (5.8% vs. 26.7%, $p < 0.001$). Thus, it seems that a mixed group of modified Penn Ab-2 + Ac + Abc is more similar to the meaning of “complicated” ATAAD and the mixed group of modified Penn Aa + Ab-1 is closer to the meaning of “uncomplicated” ATAAD. In short, we think the validation of modified Penn classification could be easier to apply and more adequately point out patients with higher risk of in-hospital mortality after surgical repair.

Notably, a prevalence of 20% to 35% of patients with ATAAD with shock (defined as generalized ischemia) was reported by a number of investigators. Nevertheless, Czerny et al. (1) did not describe if they included this major confounder, hemodynamic shock, and have not included it as variable in the multivariate analysis. This was a serious problem because hemodynamic shock is strongly related to post-repair in-hospital mortality. This flaw calls into question the methodology of investigation and its subsequent results. Thus, the in-hospital mortality of patients with organ-specific malperfusion might be overestimated or underestimated during statistical analysis, depending on whether the hemodynamic shock is presented or not in patients with organ-specific malperfusion. Finally, since generalized malperfusion is the strongest risk factor of post-repair in-hospital mortality in patients with ATAAD by many investigators (2,4) (see references 1, 3, and 5 in Chien et al. [5]), we strongly recommend the investigators consider the impact of generalized malperfusion as a major confounder of pre-operative malperfusion on the operative mortality risk for patients with ATAAD in future analyses.

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REPLY: Reconsidering the Impact of Pre-Operative Malperfusion on Acute Type A Dissection



The Modified Penn Classification

We thank Dr. Li and colleagues for their interest in our paper (1). The genuine rationale for classifications is to simplify complexity and, as the investigators state, the Penn classification is an excellent risk stratification tool to predict mortality on the ischemic pattern at clinical presentation (2). However, any sub-segmentation – despite that it might be justified and may fit – does not always help to make complex issues easier to understand and does not always direct the focus of a physician on a clinical path where the diagnostic and therapeutic aim is reached more rapidly. In general, a classification has to be easy to understand and self-explanatory, as we are well aware that not all variations of a highly dynamic process can be mapped. This is also the case when

looking at widely used risk stratification tools such as EuroSCORE II and the STS (Society of Thoracic Surgeons) score (3).

There are examples regarding acute type B aortic dissection: It is now widely accepted to categorize acute type B aortic dissections into complicated and uncomplicated cases. Nearly every physician directs his focus on the extent of malperfusion when being confronted with the diagnosis “complicated acute type B aortic dissection,” as it is simple, very clear, and logical (for simplicity we scotomize the other, more rare reasons for why an acute type B aortic dissection may be complicated).

Recently, we focused on morphological predictors in patients with primary uncomplicated acute type B aortic dissection turning into complicated type B aortic dissection mainly by developing malperfusion within the initial days after the acute event. We were successful in identifying the location of the primary entry tear—either at the concavity or the convexity of the distal aortic arch—as being decisive for presence and absence or risk of developing malperfusion. Consequently, we suggested a sub-classification into B1 and B2 (according to the location of the primary entry tear). However, our intent was not mirrored by current literature as this sub-classification is not used, presumably because even this small amendment was too difficult to be implemented into clinical routine (4).

As the investigators correctly state “generalized malperfusion” and “shock” are very important surrogates and, in addition, mutually dependent. The pre-operative variables collected in the GERAADA (German Registry for Acute Aortic Dissection Type A) registry are the use of catecholamines, the presence or absence of hemodynamically relevant pericardial effusion, as well as the need for pre-operative cardiopulmonary resuscitation. In addition, a detailed inventory of malperfused organs is provided being presence or absence of coronary, cerebral, spinal, visceral, renal, and peripheral malperfusion. The presence or absence of shock or generalized malperfusion is strongly related to these surrogate parameters. Therefore, further adjustment is not strictly necessary for the purpose of prediction. Concerning interpretation of effects, we agree that omission of important variables may introduce bias. However, this was not the case due to the above-mentioned reasons. Retrospective inclusion of new variables after having seen the result itself has the potential for bias, which has to be weighed against other potential biases. Therefore, we are convinced that our path of analysis is an appropriate one for the predefined aim of the study.

Regarding the investigators’ use of terms, it is very important to realize that shock per se is defined by “generalized malperfusion”; therefore, it is questionable to use both terms as, in addition, there are several forms of shock (such as cardiogenic and septic shock) and each might be applicable in patients with an acute type A aortic dissection cardiogenic shock (when sustaining tamponade or coronary malperfusion) or septic shock (when sustaining visceral malperfusion).

Summarizing, it was our intent to direct the focus of physicians on a very simple thing: the presence or absence of malperfusion. To be able to act before reacting becomes necessary and to enable planning to resolve malperfusion in advance before bail-out becomes necessary by defining the lowest common denominator for enabling a common language, which is in our opinion both “complicated” and “uncomplicated” acute type A aortic dissection.

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