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Lost in perfusion

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The recently published article by Conzelmann *et al.*, based on the GERAADA registry, provides further multicentre evidence from a large number of patients that organ malperfusion represents a key factor for mortality in acute Stanford type A aortic dissection (AD), with death rates increasing proportional to the number of affected organs [1, 2]. This concept critically applies also to Stanford type B AD, where non-surgical options play a primary role. In our strive to continuously improve outcome, these findings primarily imply that tailored strategies to revert organ malperfusion should start as early as possible in conjunction with aortic repair, and should take advantage of an expert multidisciplinary 'aortic team'. A major challenge in front of us, however, is represented by the unclear definition of organ malperfusion syndromes in AD. This negatively affects both clinical practice and research, and leads to poor recognition of the substantial clinical heterogeneity of patients with AD. Unfortunately, in spite of its leading role in identifying perfusion as a key pathophysiological and prognostic factor, the GERAADA registry itself does not provide any provisional definition of malperfusion syndromes applicable to clinical practice and to further studies, as the diagnosis of malperfusion was operator-/centre-specific [1–3].

In the current era, a large amount of data exploring organ perfusion three-dimensionally can be made available for patients with AD in a timely fashion and already in the Emergency Department. These data, awaiting systematic record and exploration, include objective assessment of neurological status (NIH Stroke Scale, Glasgow Coma Scale, Modified Rankin Scale), ECG findings, bedside ultrasonography of the heart and vessels, biomarkers exploring organ damage (myocardium: troponin; kidney: cystatin C, neutrophil gelatinase-associated lipocalin, creatinine; visceral organs: lactate dehydrogenase, amylase, transaminases; muscle: creatine kinase, lactate dehydrogenase), microperfusion (lactate), systemic inflammation (white blood cells, C-reactive protein), thrombotic burden (D-dimer, fibrinogen, platelets) and increasingly detailed imaging data. For standardization, these variables could be classified in predefined bundles of organ damage. A relevant model providing readily applicable methods and criteria could be sepsis, another multiorgan malperfusion killer [4].

In front of us, we should pave our way towards two major objectives. First, meta-analytic studies combining large registries of AD now have a chance to provide clinicians, especially emergency physicians from non-specialized centres, with simple assessment tools based on few unequivocal variables (e.g. age, gender, haemodynamics, coma, necessity to resuscitate) for the identification of the sickest patients. This will favour well-balanced clinical management and communication with patients' family members, while limiting heroic therapeutic decisions. Second, a new era of multidimensional evaluation and treatment of organ perfusion with measurable criteria and end-points has come, which will allow us to continuously improve the outcomes of AD.

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Reply to Morello *et al.*

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We thank Morello *et al.* for their interest in our work [1–3]. Their demand for a 'provisional definition of malperfusion syndromes' is well comprehensible, but this was neither the primary intention of the GERAADA (German Registry for Acute Aortic Dissection Type A) registry, nor possible to achieve by the collected parameters of the registry. The variables for the GERAADA registry were defined long time before the onset of the registry in July 2006, comprising the presence or absence of coronary, cerebral, spinal, visceral, renal and peripheral malperfusions as well as neurological symptoms. Thus, for the purpose of predicting lethal risk factors, the registry includes a detailed inventory of malperfused organ systems. A more detailed collection of 'malperfusion' parameters—as specified by the authors (e.g. biomarkers and imaging data)—may be worthwhile. But as we had already mentioned in our paper (see Limitations section), the limited amount of data for the GERAADA registry was deliberately designed to keep the data recruitment as simple as possible. Concerning data input into registries, the quality and reliability of too many variables is questionable and might introduce undesirable bias. Furthermore, to analyse questions, where multiple parameters or interactions of parameters should reveal a significant impact, a cohort of 2137 patients is most likely underpowered. Sometimes less is more; therefore, we are convinced that our analyses with the limited amount of data are appropriate for the predefined aim of this study. And it was one of our intentions to direct the focus on one important issue: the presence or absence of malperfusion syndromes, which has a tremendous impact on the patient's fate.

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