# The role of the immunoinflammatory response after cardiac arrest

Commentary on

**The role of the immuno-inflammatory response in patients after cardiac arrest**

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Cardiac arrest is a good (experimental) model for research in ischaemic tolerance [1], so it has been used often during the last 20 years in animal models. The (global) ischaemic event induced by cardiac arrest is followed by a phase of ischaemic preconditioning if the initial phase was of shorter or even stroke/complete ischaemia if the initial phase was of longer duration [2]. During the last years, there has been great effort in different research groups all over the world to find out the underlying pathophysiological mechanisms involved after these events or at least to find out some risk factors either in the brain or in the whole body [3, 4].

In the present issue of *Archives of Medical Science*, Anna Samborska-Sablik *et al.* present an interesting manuscript that deals with the role of the immunoinflammatory response after cardiac arrest in humans [5]. It is one of the very few studies in humans after cardiac arrest and is therefore of special interest [6]. The authors could find a significant correlation of the post-cardiac-arrest immunoinflammatory response (hs-CRP and IL-6) with the patients' clinical state and also with the prediction of survival [5]. Such data are in line with previous research in the brain [7, 8] and open the door to so-called "immunomodulatory therapy". In the brain such research is advanced, but we need a better understanding of the brain and immune system after such ischaemic events [9]. For the whole body, nearly no data exist so far. However, Anna Samborska-Sablik *et al.* have now provided a better insight into the clinical relevance of such research [5]: They show the relevance of this immunoinflammatory response for Glasgow Coma Scale (GCS) and Acute Physiology and Chronic Health Evaluation II (APACHE II) score: two of the major scores used in clinical daily practice. Therefore the immunoinflammatory response after ischaemic events is not only an experimental phenomenon (as many others), but also of great clinical importance [10, 11]. What are now the therapeutic implications of these findings? In experimental studies, preventive antibiotic treatment dramatically improved survival and outcome after ischaemic events, and even reduced

the organ's infarct size area [9, 12]. Apart from these experimental studies there are several clinical trials with promising results in this direction [12]. Smaller events of shorter duration can be protective – for example in the postoperative period – for further ischaemic events [7].

In this context, the oxygen-conserving reflex has gained more interest [13, 14] and can explain – at least partly – why the brain is only damaged after a very long duration of cardiac arrest. Nevertheless, the article of Samborska-Sablik *et al.* [5] goes deep into the pathophysiology of different organs. Further clinical research on this topic will open widely the doors to interesting therapeutic modalities and will help to resolve one of the great problems in internal medicine. Let's go on this way!

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