Neutrophils belong to white blood cells (aka leukocytes) circulating in our blood. The cells form one of the first lines of defense when our body becomes infected with pathogens (pathogenic microorganisms) such as bacteria. Neutrophils belong to granulocytes because they carry numerous granules inside their cytoplasm. These granules are full of different proteins which can be used to fight pathogens. Upon infection neutrophils clear pathogens by their either intracellular or extracellular killing. One of the latter strategies is related to formation (within the neutrophil), and subsequent ejection, of so called neutrophil extracellular traps (NETs). These are large structures composed of neutrophil genetic material (loose DNA; in cells it is packed into chromatin in which DNA binds to histones) which serves as a backbone of the whole structure. To this DNA proteins from neutrophil granules (e.g. enzyme neutrophil elastase), and from nucleus (histones), attach. Functionally, NETs are compared to spider webs or fishing nets as they catch and immobilize pathogens. For the above reasons, formation of NETs helps to control spread of infection, for example, they entrap and kill some bacteria. But the problem with NET formation is that they persist in blood vessels, where they are formed, long after pathogens are cleared from circulation and they now damage our own bystander tissues. Such problem is observed in numerous disorders and one of them is systemic inflammation or sepsis. This is a life-threatening condition often accompanied by presence of pathogens in blood during which (multi)-organ dysfunction can occur. For this, in the current project we aim to explain how these NETs are removed from blood vessels and why this process is prolonged or inadequate during some diseases such as sepsis. We will perform our studies using a particular type of microscopy – *in vivo* or intravital microscopy. This technique allows to observe and record processes occurring in real time in blood vessels of live mice. We will reveal time required for complete NET removal, identify cells involved in the process of NET decomposition and mechanisms that facilitate this process. Knowing mechanisms of a given process is the first step towards development of strategies that can control it, e.g. prevent or inhibit. For this, we also want to test how some pharmacological treatments might help to detach NETs from vasculature. We have already tested some of them in the past, and here we propose new strategies to remove NETs from vasculature. Overall, our project will explain how NETs are removed from vasculature and will help to understand why it is not efficient or impaired.