Better evaluation of electric shock injuries

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Biophotonic techniques can monitor the pathophysiology of electrical burns.

Electrical injuries have a variety of causes but are most common in the workplace where typical injuring voltages are 1–10kV. Such injuries are devastating at and beyond the point of contact. Contact points are often completely carbonized, with both tissue and bone removed, and necrosis (death of tissue) can spread well beyond the main site of injury. Damage to cutaneous tissues depends on the site of impact, the strength of the field, and the exposure time. At present, the extent of electrical injury is often diagnosed by visual and tactile inspection. If we could develop direct, non-invasive diagnostics for tissue close to the entry and exit sites of electrical injury, clinicians would have important additional information when selecting an operative strategy.

Various imaging techniques have been used to evaluate soft tissue injury. Magnetic resonance imaging (MRI) is a valuable tool for visualizing the extent of deep tissue damage and guiding surgical procedures. Furthermore, functional MRI can be used to monitor the physiological behavior of an organ system, but, unfortunately, this technique is very costly. Thermography has been used to assess burn depth in correlation to temperature, but its susceptibility to ambient temperature and time of injury restrict its suitability for real clinical use. Ultrasound can distinguish between necrotic and viable tissue—heat damaged tissue has significantly different acoustic impedance compared to healthy tissue—but has the disadvantage of requiring contact with the patient. Finally, laser doppler imaging (LDI) can be a useful and non-invasive tool for evaluating burn wounds by measuring blood perfusion (flow through the wound).

We combined off-the-shelf imaging tools (laser doppler, thermal imagers) and custom-made devices (our spectral imaging system, see Figure 1) to determine tissue viability after electrical injury. First, hemoglobin volume fraction combined with an assessment of blood motility is useful to determine the presence of ischemic regions (i.e., with an inadequate blood supply) or hematoma (blood clots). In addition, the status of hemoglobin oxygenation in the local capillary bed can pinpoint hypoxic regions (that is, with too little oxygen), as well as inflammation. Methemoglobin is an abnormal form of oxidized hemoglobin that does not release oxygen to body tissues, and its concentration in the skin, together with excretion of hemoglobin in the urine, indicates the breakdown and damage of tissue and organs.

We studied tissue injury to understand the local and systemic mechanisms of physiologic damage that occur in an electrical injury. We used a combination of these imaging techniques and chemical analysis to assess damage to tissues from electric shocks. We used a spatial frequency domain imaging (SFDI) system to assess pathophysiologic changes in the wounds at different time periods. SFDI allowed us to do some crude topographic sectioning of tissue and quantify pathological changes in tissue oxygen saturation, hemoglobin volume fraction, and...
methemoglobin as compared to healthy subjects. Additionally, we used a co-registered (i.e., all imagers look at the same part of the sample) LDI system to monitor tissue perfusion to highlight the presence of a hematoma or an ischemic area due to vascular break-down.

We also used a thermal camera to monitor skin temperature during electrical shocks. This measurement correlates with finite element models (FEM) of electrical current transfer into biological media, which attempt to establish the depth of injury. Our FEM model takes tissue perfusion into account, which is a primary source of cooling during thermal damage. We also conducted plasma cytokine analysis to monitor plasma protein concentration. Altogether, we assayed 80 different proteins, including vascular endothelial growth factor and interleukin-10. We chose proteins to analyze primarily because of their role in systemic inflammatory responses, which we expected would indicate systemic pathophysiology from injury. We also analyzed gene expression, and used a rat animal model to show that our system can determine levels of injury after a 1000V electrical shock.

Our studies showed an interesting, albeit complex, picture of electrical injury, where several pathological events occur contemporaneously. First, we found that high current levels (up to 6A) generated by the electrical shock find preferential pathways (tendons, vessels, muscle structures) within the body from the two poles. Current flowing through these tissues is converted quickly into heat, the only alleviating factor being the presence of vessels that have a cooling effect. Tissues and cells are damaged at temperatures above 42°C. In addition, genes involved in dermatological disease and connective tissue networks are upregulated, showing a systemic damage to skin structure. At the same time, areas of low perfusion appear near the wound, pointing to ischemia and hematoma due to blood vessel damage. For a shock lasting 20s, methemoglobin concentration increases in the hours following the injury, while oxygen saturation in the skin decreases. Both effects can be related to ischemia and vasculature damage.

Saving adjacent tissue remains a key goal of surgical intervention for this condition. With direct non-invasive diagnostics, we hope to develop methods to better assess the severity of tissue damage, both local and systemic, in order to better predict optimal treatment strategies and possibly improve limb salvage.

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References