Venous ulcers are the most common and severe skin wounds occurring in the lower legs. These wounds are painful, hard to heal and have a 78% chance of recurrence after two years. Currently, there are no preventative approaches for ulcers; they are treated after they develop. Once diagnosed with a venous ulcer, it usually reoccurs and patients are required to seek treatment for the rest of their lives. Such high recurrence rate and the need for long term wound care lead to frequent visit to the wound clinics, and result in huge financial burdens on the patients, family and society. To move toward prevention, a need exists to understand the pathology of venous ulcer formation so that we can identify populations at risk of developing venous ulcers and implement preventive measures at an early stage.

The goals of this research were to: i) determine the blood flow patterns in the lower legs between patients with venous ulcers and a healthy population; ii) quantify the hemodynamic parameters that contribute to the measured blood flow differences between ulcer patients and a healthy sample; iii) understand the progressive changes within the skin tissue when undergoing inflammation and edema during the ulcer formation.

The first goal was addressed by experimental work conducted in the wound clinic. A blood perfusion test was performed on the lower legs of the ulcer patients and a healthy control group. External normal, and combined normal and shear loads were applied to stimulate changes in the blood flow. The blood perfusion profile under no load, load, and recovery after loads were analyzed. Results showed that the ulcerated legs were significantly different than healthy legs in the response to load (blood perfusion decrease under loading), as well as in the reactive hyperemia (the spike in blood perfusion upon the removal of loads). More interestingly, the legs that did not have open wounds but from the same venous ulcer patients exhibited an intermediate trend between the wounded and healthy legs, suggesting a progressive change existed during ulcer development.

Using a Windkessel based model together with experimental data, the patient-specific hemodynamic parameters: blood vessel resistances and compliances were obtained. Results indicated the significant differences in the localized vessel resistance and compliance between ulcerated legs and healthy legs. This suggests a threshold/range of the parameters existed between healthy, non-ulcerated legs (from same patient), and ulcerated legs. These patient-specific parameters have the potential of identifying population at risk of developing venous ulcers.

Finally, a Finite Element model explored the tissue internal stresses and pressure changes due to inflammation, a symptom associated with venous ulcer development. Two inflammation identifiers: glycosaminoglycans (GAGs) and sodium were used in a series of parametric studies.
Results showed that with the GAGs and sodium accumulation, skin tissue produces an increased fluid pressure, resulting in tissue ischemia. Furthermore, such accumulation also increased tissue elastic stress, which leads to tissue damage and wound formation. The Finite Element model demonstrated the detailed pathological events that happen between inflammation, edema and skin ulceration.

With both experimental and numerical approaches, this research improved the understanding of venous ulcer pathology, exposed the internal changes in vessel compliances and resistances, tissue pressure and stress during ulcer formation, and predicted the onset of venous ulcers. This work laid a solid foundation for developing better preventive measures and clinical care in the future.

Persons with disabilities please contact the Mechanical Engineering office at 517-355-5131 to request accommodations.