Pathophysiology of ulceration

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The exact pathophysiology of ulcer development has not been established. Originally , it was thought that static blood within the superficial veins led to hypoxia, which caused tissue death (stasis ulcers). This was not confirmed by investigation of venous oxygen saturation, which was found to be higher in ulcerated limbs. This led to the concept of arteriovenous fistulae, which were thought to develop in response to the high venous pressure; however, this could not be confirmed. High venous pressure was found to be associated with a pericapil - lary infiltrate. This includes fibrin and other proteins, which are known to lead to fibrosis. It was hypothesised that these ‘cu ff s’ could act as an impediment to di ff usion of oxygen and nutrients. Leukocytes were found to be reduced in the blood retur ning - from legs with venous hypertension. This decrease in leukocyte passage was shown to increase if short-term venous hyperten - sion was induced by application of a tourniquet. This led to the conce pt of white cell ‘trapping’, which, however, has not been confirmed by further investigation. Polymor phonuclear leuko - cytes were not found within the tissues, but increased numbers of mast cells, monocytes and lymphocytes have been found in - periulcer tissues. Reactive oxygen species are increased in the ulcer environ - ment and these may generate fr ee radicals, leading to tissue

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the fibroblasts in the ulcer surrounds are also abnormal, being in a ‘senescent’ state. Growth factors may be inhibited, leading to poor repair, and their absence may also lead to ulceration. It is debated whether these factors are the cause or e ff ect of an ulcer. At present, ambulatory venous hypertension is the only accepted underlying cause of venous ulceration. This also explains why venous ulcers are never seen in the upper limb. It is important to try to define the exact mec hanism of ulcer development. V enous hypertension may be the result of primary valve incompetence of the saphenous veins, incompetence of the perforating veins or incompetence or obstruction of the deep veins.

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