

INFLAMMATION

Inflammation is a fundamental physiological process of soft tissues responding to injury. External force or internally disruptive chemical or biological agents may injure (stress) soft tissues, with the result that blood vessels and other soft tissues in or near the injured area will exhibit a dynamic complex of cytological and histological reactions. These reactions are local in nature. They are designed to cause the destruction or removal of injurious material (bacteria, foreign matter or chemicals) and to promote morphological responses that lead to repair and healing. The cardinal signs of soft tissue inflammation are rubor, calor, tumor (swelling), dolor (pain), relatively high skin resistance overlying the inflamed tissues and often some impairment of function.

Inflammation is a defensive reaction to all sorts of tissue injury and the names of numerous inflammatory conditions commonly designated by the suffix *-itis* (bursitis, tenosynovitis, and capsulitis).

Whenever a body tissue is cut, burned, infected or otherwise damaged, enzymes in the local area of injury are activated to free bradykinin from large precursor molecules present in the blood and various other soft tissues. The primary function of bradykinin is to increase the sensation of pain. It does this by filling special receptor sites on free pain nerve endings which causes them to fire a barrage of intense afferent pain impulses that amplify the sensation of pain already produced by the injury. At the same time, the bradykinin sensitizes free nerve endings, making them hypersensitive to heat and light touch, creating an overall sensation of *soreness*. The secondary function of bradykinin is to promote the production of histamine. It does this directly by binding to mast cells in the area and provoking them to release histamine; it does this indirectly through the amplification of the pain process mentioned above. Nerve centers in the brain or spinal cord, designed for this purpose, respond to the amplified pain impulses by sending efferent impulses to appropriate nerve endings housed in the involved soft tissues, causing them to release *substance P* (a neuropeptide), which also binds to mast cells and further boosts histamine production.

The function of the histamine is to increase blood flow into the involved area by dilating

arterioles and increasing capillary vessel permeability by enlarging the spaces between capillary vessel wall cells while simultaneously constricting venules to prevent fluid outflow. The enlarged spaces between the capillary wall cells allow fluid, white blood cells, kallikrein, and bradykinin precursors to leak through them into the interstitial space. This combined with the restricted venule outflow, causes interstitial space congestion and consequent soft tissue swelling.

The released bradykinin precursors cause additional bradykinin to be constituted. This additional bradykinin binds itself to the membranes of nearby cells and sets in motion a chain reaction in which polyunsaturated fatty acids that help to make up the tissue cell membranes are released. These polyunsaturated fatty acids are then used to produce prostaglandins. Prostaglandins stimulate the surrounding tissues to cause blood vessel dilation and to facilitate further capillary bed permeability. Additionally, the prostaglandins bind to receptor sites on the involved free pain nerve endings to promote additional pain impulse production and to stimulate additional bradykinin production. This is important, since enzymes present in blood generally deactivate bradykinin almost as soon as it is produced. This means that bradykinin must be continually released to sustain the inflammatory reaction. The inflammatory process is, after all, designed to continue until any infection is fully suppressed or until the source of irritation has been removed and the healing process begun.

Soft tissue inflammation is determined through assessment of the cardinal signs of inflammation, including pain on palpation, raised soft tissue temperature, swelling, redness and the presence of relatively high skin resistance over the supposed deep tissue site. The first four symptoms will vary in appearance, sometimes failing to appear to casual observation and at other times becoming blatantly apparent. The author has found that the most reliable and objective measurement of inflammation available in the clinical setting, to date, is differential skin resistance (DSR) survey. The skin resistance overlying the area of deeper tissue inflammation will be relatively high when compared with surrounding skin area. If treatment is successful, the high level of

The author has (through observation) come to the conclusion that prolonged exposure to “burning” action of prostaglandins promotes the production of what may be termed “adhesions”. That is, the body thinks that it has been burned, so it floods the area with collagen fibers. These fibers would ordinarily form a “scar”, but no “scar” is necessary, so these fibers start hooking tissue layers together. Many of these layers are meant to slide over one another, but when stuck together by the adhesions they can no longer slide, and the pulling of one tissue layer against another serves as a continuing source of irritation.

The treatment of soft tissue inflammation should be directed at:

- Removing or avoiding the causal source of inflammation through bracing, casting, splinting, taping, strapping or rest.

- The breaking of adhesion formations (soft tissue manipulation) in the inflamed zone and surrounding areas.
- Facilitating the decomposition and inhibition of the production of the inflammatory chemicals through the application of effective anti-inflammatories (through phonophoresis or topical application).
- Improving circulation in the involved tissues through ice packing, low frequency electrical stimulation, adhesion breaking or milking-massage.
- Improving strength in the involved musculature to prevent reinjury and to improve vascular function through electrical toning or exercise.

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