

# HBOT in RSD

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Post-traumatic Sudeck's Syndrome is a reflex sympathetic dystrophy which consists of pain and tenderness, usually in a distal extremity, associated with vasomotor instability, swelling and trophic skin changes arising after trauma. The severity of the syndrome is frequently unrelated to the severity of the injury and the dystrophy of often appears after minor trauma. The classic X-ray shows patchy bone demineralization.

In its early stages, it is either unrecognized, misdiagnosed, and often mistreated so the patient may have a prolonged and severe disability. No treatment, hitherto has proved to be very successful, once the disease has become established: various forms of physiotherapy, systemic administration of drugs ( anti-inflammatory agents, vasodilators, steroids, calcitonin ), peripheral chemical sympathectomy, infiltration of painful areas with local anesthetics, sympathectomy and sympathetic blocks, section of the sensory nerves or of the dorsal roots of the spinothalamic tract ( in intractable cases) have been reported in the literature.

Despite any or all of these measures, many patients improve little or not at all, so that their symptoms persist for months or years. Some patients have attempted suicide because of all the psychological and economical problems related to the disease. The etiopathology of the condition is uncertain. The present pathogenic hypothesis is that after an injury to the limb there is an initial vasomotor reflex spasm and, in a second phase, a loss of vascular tone with persistent vasodilatation and rapid bone resorption. Chronic irritation of peripheral sensory nerve secondary to trauma and soft tissue sets up abnormal activity of internuclear neuronal pool and continuous stimulation of sympathetic motor efferent fibers.

Hypoxia and acidosis lead undifferentiated mesenchymal cells and younger fibroblast to proliferation and quicker maturation ( a state which requires lower oxygen consumption ) with abnormal fibrous tissue production, edema organization and joint stiffness. Reflex vasomotor disturbances, resulting in hypoxia, catabolite production and acidosis stimulate sensory nerve termination and close a vicious self sustaining cycle.

The use of HBOT in the treatment of post-traumatic Sudeck's Syndrome is rational. In fact, hyperbaric oxygenation induces vasoconstriction and reduce edema: this counteracts vascular stasis and venous repletion, increases depresses osteoblast activity and mineralization, reduces fibrous tissue formation. HBOT seems to break the vicious self sustaining cycle of reflex sympathetic dystrophy, because normalization of local tissue oxygen tension, pH and water interstitial content stops abnormal sensory nerve stimulation and efferent vasomotor activity.

## References

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