

Bone Stimulation by Low Level Laser - A Theoretical Model for the Effects

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The anecdotal and researched evidence for the effects of Low Level Laser Therapy (LLLT) on the stimulation of bone have been reported for over 20 years. This has been in the form of local as well as systematic effects – including contra-lateral effects. Reports of stimulation of rabbit radii fractures and mice femurs were made as early as 1986 and 1987 with irradiated bones healing faster than controls and contra-lateral non-treated fractures similarly demonstrating faster healing times. Over the following decade and a half, further studies have also investigated and demonstrated that LLLT is effective for the stimulation of bone tissue.

The reasons for this have been attributed to the general effects of LLLT and its ability to increase the rates of healing through mitochondrial ATP production and alteration in the cellular lipid bi-layer. Additional hypothesis include the subsequent capacity of irradiated cells to alter their ion exchange rate and thus influence the catalytic effects of the specific enzymes and substrates. These in turn initiate and promote the healing process completing the cascading cycle of events.

In the area of bone specific research, Dr. Tony Pohl of the Royal Adelaide Hospital in South Australia, has provided a new theory that postulates that the majority of fluid transfer and exchange within living bone is predominantly influenced by the lymphatic circulation. LLLT is well documented and known as having effects that influence the lymphatic circulation and wound healing process. A coupling of these two areas of theory can demonstrate a positive description and explanation of the predominant effects of LLLT in bone stimulation. In reality, LLLT's effects on bone may well be a further consequence of its actions on the lymphatic circulation.

Reports of stimulation of Rabbit radii fractures were made by Tang in 1986 and similar reports by Trelles in 1987 on mice femurs. In both situations the irradiated bones healed faster than the controls. In another study by Hernandez-Ros, in 1987, LLLT demonstrated stimulation of fresh fractures on Sprague-Dawley rats that were fractured bilaterally. The unexpected results of this study were that the contra-lateral fractured non-treated limb also healed faster than the control group. Over the following decade and a half further studies (Yamada 1991; Pyczek, Sopala et al. 1994; Ozawa 1995; Horowitz 1996; Yaakobi 1996; Saito and Shimizu 1997) have also investigated and demonstrated that LLLT is effective for promoting the stimulation of bone healing. Recently Nicolau and colleagues (2002) from Brazil demonstrated the positive effect of LLLT on the stimulation of bone in mice with latent promotion of bone remodeling at injury sites without changes in bone architecture, increased bone volume and increased osteoblast surface through increased resorption and formation of bone with higher apposition rates. A positive effect on bony implants has been

demonstrated by Dörtbudak (2002) and Guzzardella (2003). The effect of laser irradiation on osteoblastic cells has been reported by Yamamoto (2001) and Guzzardella (2002).

The reasoning for this amelioration in all experimental circumstances, based on electron microscopy as well as macroscopic histological evidence, was concluded to be due to i.a. improved vascularisation as a consequence of blood vessel formation, absorption of the haematoma, macrophage action, fibroblast proliferation, chondrocyte activity, bone remodeling from increased osteoblastic activity and deposition of calcium salts.

These changes and evidence based studies attribute the macro- and microscopic effects to the known and accepted general actions of LLLT and its ability to increase rates of healing through stimulation of ATP production, (Karu 1989; Smith 1990) promoting repair and polarization of the cellular lipid bilayer (Fenyo 1990) as well as LLLT's capacity to affect cells through alterations in their exchange rate of ions (Robinson and Walters 1991) and influences the catalytic effects of the specific enzymes and substrates (Pouysseguer 1985; Karu 1988) which in turn initiate and promote the healing process.

More recent work by Dr. Tony Pohl, an internationally renowned Orthopaedic Surgeon from the Royal Adelaide Hospital in South Australia and lecturer at the Adelaide and South Australian Universities, has given rise to a new theory on bone circulation through reconsideration of fluid and protein transfer within bone (Pohl 1999). This theory suggests that the general understanding of the circulatory action within bone has been incorrect. Pohl postulates that the majority of fluid transfer and exchange within the living bone is predominantly influenced by the lymphatic rather than the vascular circulation. This is justified through studies on bone fluid input and output levels that have demonstrated that the venous and arterial aspect of circulation alone cannot account for the demonstrated levels of output nor the presence of free radical molecules which exceed those of the vascular input. Furthermore, the diameter of large protein cells within the bone exceed the diameter of the vessels that form the terminal aspects of the circulatory system making it impossible for them to have been delivered via this system. Consequently, an additional circulatory system must be present that will account for both the increased output and the presence of the large diameter protein cells as well as the free radicals.

If LLLT is then considered within the context of this new theory on bone circulation and the contribution of the lymphatic circulation then a further logical reasoned deduction for the action of LLLT on bone stimulation can be made. LLLT has a well documented and known effect influencing the lymphatic circulation. This has been demonstrated from the early works of Lievens, (1985) that demonstrated the influence of "Laser Irradiation" on the motricity of the lymphatic system and on the wound healing process. This is supported by several wound studies that demonstrate that the levels of protein rich exudates in non-healing wounds increase markedly from exposure to LLLT. This demonstrated action is determined to be as a result of the increase in lymphatic circulation (Robinson and Walters 1991; Gabel 1995). More recent work at the Flinders Medical Center in Adelaide South Australia has been completed and presented at the World Association of Laser Therapy conference in Tokyo Japan (Anderson, Carati et al. 2002). This study has demonstrated the positive effects of LLLT on the lymphatic circulation and its consequential benefits to the post mastectomy patient.

An understanding of the existing knowledge of the effects of LLLT on the lymphatic system combined with the hypothesis of bone fluid transport provides a coupled theory that would demonstrate a positive description and explain of the predominant effects of LLLT in bone stimulation.

In the trauma situation of direct or indirect damage to the bone, including fractures and periosteal induced damage such as stress fractures, the tissue damage leads to compromises that include but are not limited to, physical blockage from the trauma and waste / debris, increased fluid and circulatory viscosity from added cellular content within the lymphatics, lower speed motility and energy deficit in the tissue and cells from the loss of ATP production as a general effect from the trauma, cell changes and inability of mitochondria to function at the normal higher level to promote self repair and regeneration.

LLLT with its known general effects and specific direct effects on the lymphatic system would act to stimulate mitochondria ATP that increases cellular and circulatory motility as well as directly influencing lymphatic flow. LLLT also promotes increased permeability in interstitial tissue and facial layers (Gabel 1995) reducing stagnation and blockage. These actions would assist the increase in lymphatic flow and consequently the circulation within the affected bone. There is also a hypothetical potential that the presence of LLLT by increasing lymphatic circulation does so by virtue of an increase in the diameter of the lymphatic vessels, not just by increased flow rates within the vessel at an unchanged diameter. This diameter increase, if definitively present, would also explain the presence of large diameter protein cells within the normal bone circulation that cannot be attributed to the vascular circulation and would additionally explain a facilitated process for removal of debris and larger protein cells passing out of traumatized areas that is additionally stimulated by the use of LLLT.

Stimulation of bone healing by LLLT has till now has been generally classified as a consequence of the general healing effects of LLLT. In reality LLLT's effect on bone may well be a further consequence of its actions on the lymphatic circulation.

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