EDITORIAL

HEATING AND CHRONIC LYMPHEDEMA

The value of heat in the management of disease is as old as the hills and as mysterious as any ancient myth. One can imagine the dinosaurs basking in warm and humid water, relying on heat to mobilize their heavy limbs like the lizards of the Galapagos Islands seeking the sun after a cold night. Heat itself has not always been given the credit for any good effects. Fever therapy has been interpreted as a stimulus of endogenous steroid production and might now be thought of as a role for interleukins facilitating complex biochemical pathways. The Turkish bath and sauna are sometimes thought of as a form of catharsis due to a "good sweat."

The Editors have agonized over the paper from China (see page 20) which concerns the value of heat in the management of lymphedema seeking from the authors both proof and explanation. We are satisfied that many sufferers are benefiting but, as for explanation, this is still mysterious. Heat, of course, has metabolic effects and this, together with enhancement of blood flow, is offered as an explanation for phlogistic effects countering inflammation. The hot poultice is now less often prescribed in the Developed World but hot cupping is a popular alternative therapy in many countries and an indirect stimulus to blood supply is sometimes used as an explanation of counter irritants.

In lymphedema, the role of blood supply is equivocal. Flooding the tissues and washing through the interstitium may be good when the lymphatics are functioning but may overload a failing system. An answer to the question "Why does heat work?" is needed, and not just for lymphedema. For instance, such therapy is used for psoriasis (1) which, if anything, is a disease characterized by an excess of metabolic stimuli within the skin.

Cellular mechanisms encouraging phagocytosis and proteolysis and the activation of immunological attacks on organisms damaging the lymphatics, even altering the environment of a parasite sitting in the proximal lymphatics by flooding them with tissue products they have successfully walled themselves off from, can all be conceived as mechanisms for the good effect of heating.

The relationship between the lymphatic system and the blood flow contributing to tissue edema has not been studied in experiments in which sustained high skin temperature can be shown to redistribute blood flow (2).

In this age, when immunological or biochemical processes are regarded as the explanation of almost anything, one modality must not be ignored and that is the transmission of mechanical forces (3). Movement of the tissues is enhanced by warming and this is the basis of the use of hot water, wax or mud baths, used by rheumatologists for joint diseases and masseurs for skin and muscular disorders. The "facial" of the beauty parlor is also often a combination of heat and massage for the dispersal of both deep and superficial mischief.

One can conceive that the lymphatic system would work more efficiently in an environment which is not made stodgy by edema and many of the body "fluids" are
susceptible to rheological explanations of their dispersal, i.e., flowing better when warm.

The problem in lymphedematous tissues is that the interstitium is full of materials that cannot be shifted and almost all such materials, whether lipid or protein, are made more liquid by warmth and almost all pathways, whether blood vessels or low resistance channels in the interstitium, are decongested by mechanisms encouraging a lowering of the viscosity of their contents. Viscous materials shift more easily when they are warmed and when they are stirred. This Editor has no difficulty in at least imagining that heat ought to be beneficial in lymphedema because of its effects on prelymphatic pathways. Any benefit on lymphatics themselves then follows.

Mechanical properties of all biological tissues are sensitive to change in temperature. Temperature alters rheology, cold makes tissue stiffer, and the well-known reduced distensibility of blood vessels and increased viscosity of blood in sclerodematous tissues is one explanation of Raynaud’s phenomenon. Even the transmission of the arterial pulse to the tissues is likely to be enhanced by counteracting the vasoconstrictor effects of cooling and the changes in tissue compliance that are known to characterize edematous tissues (4). Salter 1981 (5) estimated that for every 1°C decrease in dermal temperature there was a loss of compliance of 14%. It is simply unknown whether lymphedematous tissues are more or less affected by changes in temperature but techniques exist for testing such and, hopefully, it will not be long before we have an explanation of Chang et al’s observations.

REFERENCES


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