HYPERTHERMIA AND PHYSIOTHERAPY: A GUIDE TO INDICATIONS AND DOSIMETRY

Dr Stefano Della Villa – Dr. Domenico Creta
Isokinetic - Bologna

Introduction

It has been known since historical times that the application of heat can have beneficial effects on the organism.

Since ancient times man has used heat as a therapeutic element to solve many of the types of pain from which he suffered, using hot-humid compresses, mud and sand baths, warming pads, hot baths, the "moxa"in acupuncture, adopting modalities which we would define today as exogenous heat therapy.

But what is heat? Heat is a form of energy, more precisely thermal energy and can be obtained by the transformation of another form of energy (mechanic, chemical, electric, etc.). The reverse process is also true, that is the transformation of thermal energy (heat) into another form of energy (for example mechanical energy) (thermal machines such as the internal combustion engine work on this principle). (28)

The passage of heat from one body to another or the simple application of heat on a body can cause the temperature of the body to rise, due to the increase in motion energy (kinetic energy) by the particles of which it is made up of.

The continuing application of heat causes a change in the state of the body (for example, from a solid state to a liquid one or from a liquid one to a gaseous one) due to the transformation of the links that exist between the particles themselves in their various states of aggregation (potential energy).

What has been said up to now is true for what regards normal chemio-physical reactions which involve inorganic matter. If instead we are dealing with biological tissue and living organisms, it is a completely different matter especially with regard to the reversal of the reactions.

In fact if a too high level of heat is applied or for too long a time, irreversible changes and\or denaturation could occur on the part of the proteins, the enzymes, and the catalysts resulting in biological damage. (44) (69)

What has been said up to now explains the ancient use of heat for therapeutic and analgesic reasons and the complex notions of biology, chemistry, biochemistry and physics on which is based the use of heat for medical purposes.

Ancient populations used heat because they realised that it had beneficial effects for certain pathologies, but they did not know the mechanisms underlying its action; empirically they realised that the skin was an obstacle in the application of deep heat and consequently the use of heat was limited to the treatment of topical pathologies.

A further limit to the precise use of heat is represented by the system for regulating body heat of the individual which tends to bring the tissue temperature back down to its physiological temperature (37°C in man) and whose precise mechanism was certainly not understood in detail in ancient times.

Moreover, if we consider the fact that the increase in the number of chemical reactions that occur in a unit of time is directly in proportion to the surrounding temperature where these reactions take place, we realise that heat applied to biological systems increases the number and the speed of the biochemical reactions, influencing the activity of the single cells and of the system of which they are a part.

The biological effects of heat

The application of heat to biological tissue brings about reflex physiological reactions. (44)

It is important to distinguish the use of topical heat and that of deep heat because they have different responses. (3) (18)

In the first case, given that the means used cannot allow the heating of human tissue unless through an undesirable overheating of the skin, heat can only be applied on the surface. In such a case the reflex vasomotor reactions, through stimulation of the thermic receptors in the skin and the pain receptors, would bring about a vasoconstriction of the muscle arterioles and of the inactive organs, with changes in the haematic flow on the surface and dilatation of the subcutaneous and cutaneous arteriolar bed. (1) (31) (41)

This topical vasodilation induced by the heating of the tissues is aimed at increasing the dispersion of heat from the skin surface, cooling down the zone, and trying to bring the skin temperature back to its physiological levels. (54)

Such behaviour however, which is completely natural and aimed at conserving the homostasis of the system, goes against the goal we have set ourselves of heating the tissue.

The limits of topically applied heat result obvious from this and more so the limits tied to the heating techniques which encounter in the skin an easily damaged biological barrier when subjected to a thermal rise.

Deep heat however activates a series of reflex vasomotor reactions which behave in a substantially different way and which come about for the very reason that heating

takes place in the tissues in depth and in the muscle tissue in particular, as well as in the cutaneous and subcutaneous tissues.(62)

The revolutionary fact consists in, with the use of new equipment, the heat being able to penetrate in depth, without damaging the skin, to monitor the temperature both on the surface and in-depth, to keep the set temperature levels constant, to have a homogenous distribution of heat, to be able to repeat the treatment at a later date with the exact same conditions guaranteed, and all this under conditions of safety and comfort for both patient and doctor.

Apart from the distictive features and innovations that the Hypertherm equipment has brought with it and which have allowed problems to be solved which for many years were seemingly unsurmountable, from a medical point of view what interests us is the transfer of heat and the biological responses so caused, independantly of the type of energy employed and of the technique used.(11) (20) (21) (58)

But how does heat bring about biological effects, and what are the physiological premises of tissue response to heat?

Why is pain, oedema, joint stiffness, etc., reduced or why do they disappear? (6)

The application of heat to a biological system of tissues in a living organism causes changes in blood flow due to dilatation of the arterioles and the capillaries.

Such an increase in flux is due to both a direct effect, given by the rise in temperature, and to reflex mechanisms. The latter involve the thermic receptors which when stimulated by heat activate local axonic reflexes and more complex central responses which come under the hypothalamic control of the temperature.

When the heat penetration is deep the increase in in-depth blood flow has the purpose of cooling down the area because the blood temperature is lower than the temperature of the area of tissue under treatment (for example, measured temperature 39.5°C; physiological blood temperature 37° - 37.5°C).

Such an increase in the locoregional blood flux, which comes about by an arterial and venous vasodilatation and by the opening of the entire vasal bed (microcirculation), causes the speed of the haematic flux to diminish and a consequent increase in the contact time of the blood in the capillary bed.

The increase in contact time is expressed in an increase of gaseous exchanges, of metabolites, of ions between the vasal bed and the perivascular spaces and the interstice, with a greater removal of the products of cellular catabolism, of necrotic deposits, result of the lysis of degenerate cells.

The increase in temperature brings with it a higher uptake of free O2 thanks to the dissociation of the oxygen from the haemoglobin being made easier. (43) (60)

This higher uptake of free oxygen is essential in order to satisfy the increased metabolic needs of the cells, in which, still due to this thermal rise, can be seen an increase in the number and the speed of the chemical and biochemical reactions, which can only be sustained by increased availability of O2 and metabolites.

As already mentioned the increase in haematic flux causes a removal of the catabolites and the products of cell degradation, causing the death of the more seriously damaged cells.

Cells that are damaged or ill because of some form of pathogenic or traumatic noxa have in fact a reduced resistance in the face of further stressful stimuli and a significant thermal rise is sufficient to cause their necrosis. (39) (65)

In this way the cell necrosis itself causes the release of chemiotaxis substances, of growth-factors that represent a powerful regenerative and\or reparative stimulus.

What is important and needs to be emphasized, is that the deep penetration of heat causes the vasodilatation of the inner arterioles, which are dilated by input that originates centrally and the purpose of this reaction would be to cool down the area.

Since the blood however is less capable of cooling down the area than Hypertherm is of heating the tissue, we have a global increase in temperature of the locoregional tissues and the therapeutic temperature thus induced is maintained within a range of set values.

After all, the blood in-depth acts like a bag of water against the skin and tries (and to a certain extent succeeds) to cool down the area to bring the temperature back to its physiological levels. (Tab 1.)

The effects of heat on the skeletal muscle apparatus

Alongside the finer points of the effect on cells and tissues that have been described above, there are also more obvious macroscopic effects which allow the use of such therapeutic modalities for the treatment, at least symptomatic, of numerous inflammatory, traumatic and degenerative illnesses of the skeletal muscle apparatus.

The increase in blood flow which, we repeat, is at the basis of the effects induced by heat, causes an increase in the extensibility of the collagene. (73)

This effect is obvious if we think of the molecular structure of collagene tissue where collagene fibres and elastic fibres are variously organised among themselves. (5). Between these proteins there are in fact complex chemical links which are weakened by a targeted increase in temperature with the result that the fibres can then slide more liberally across each other, without subjecting the structures involved (tendons, ligaments, articular capsula) to an excessive increase in tension during forced stretching. (4) (10) (15) (34) (36)

The muscle tissue, which contains numerous vessels and a thick network of capillaries, when subjected to heat, sees a significant increase in the amount of blood at its disposal with the metabolic effects already described above.

This increase in the amount of blood means better contractile efficiency since the activity of the ATPase enzyme increases which is able to split the phosphoric links making more energy available. (12) (47) (67)

Moreover, thanks to the increased haematic flux, the pH is normalized, the variations of which affect the activity of numerous enzymes including that of ATPase.

With an increment in the flux, through an increase in the amount of ions and sodium in particular, a normalization of the electrolytes is obtained, which if not balanced seem to be the principal cause of cramp.(66)

Moreover the muscle contains a delicate and complex system of connective supports whose base molecule is represented by collagene and so the use of heat allows the myofibrils to slide more easily over each over and allows an increased extensibility and elasticity of the muscle over all. It is enough to consider the fact that stretching exercises are performed more smoothly if the muscle has been previously warmed-up. (49) (69) (70)

With regard to joint stiffness we can say that the therapeutic effects, which consist in recovery of the range of motion with greater joint fluidity and a diminishing of painful symptoms, are due to a positive response to deep heat therapy in the tissues which control joint movement. (7) (8) (9) (26) (71) (72) (74) (75)

The reduction in muscle spasm is attributable to a reflex mechanism in which are involved complex receptor structures such as the Golgi tendon organ, the neuro-muscular spindles, and the gamma fibres, the secondary afferent fibres. (16) (17) (27) (53)

The Golgi tendon organ and the neuro-muscular spindles participate in addition to the regulation of the muscle tone and the degree of tension of the tendons.

The Golgi tendon organ has as one of its basic actions that of inhibiting muscle contraction; when subjected to heat its frequency increases resulting in an accentuation of muscle inhibition.

The neuro-muscular spindles are instead responsible for muscle tone with their basal frequency; if subjected to heat there is a decrease in frequency with a subsequent relaxing of the muscle. (60)

A relaxing of the muscles and an inhibition of contraction are noted in conclusion when the neurosensorial structures responsible for contractile function control are subjected to heat. (60) (61)

With regard to the analgesic effect, it can be seen that heat is able to inhibit or diminish the frequency of the fine afferent fibres which seem to be those responsible for transmitting pain impulses. (24) (60) (61)

Finally the diminishing of the inflammatory infiltrates, the draining and the resolution of the exudates and the oedemas can be attributed, as already described above, to an increase in the capillary permeability which facilitates these exchanges in addition to a mechanical drainage sustained by the increment in haematic flow. (Table 2.)

Pathologic physiology of the response to trauma

To exploit correctly the effects of heat means having a perfect knowledge of tissue anatomy.

Tissues differ in that each has its own distictive anatomic structure and a vascularization specific to each tissue type. (51) (59)

This means that for each type the effects of the heat will be different and consequently the moment, the duration and the modalities of heat application will also be different.

For the above reasons, different anatomy, different vascularization, it will mean that the tissues will also have different repair capabilities. (30) (52) (68)

Skin, for example, is capable of repairing a superficial injury, even if there has been a significant loss of substance, by an epithelial regeneration; the nervous tissue or the cartilaginous tissue is much less capable if at all of acting in this way. (40)

What all tissues have in common, however, in response to a pathogenic noxa or a trauma is an inflammatory phase. (50) (56)

This fundamental and essential way of reacting of the organism is the same whether it is the muscle tissue which has been damaged rather than bone tissue, while what can vary is the final result of the inflammatory process. (30) (68)

The acute inflammatory response is given by a series of common biological events, distinguished by four fundamental phenomena, that is *rubor* (reddening), *calor* (rise in heat in comparison to the surrounding area), *tumor* (tumefaction) and *dolor* (pain). (13)

It is necessary to add as a fifth fundamental sign of inflammation, of particular interest in physiatrics and rehabilitation, the functio laesa that is to say the disturbance of normal functions.

If we had to define inflammation we would say that it represents that combination of local alterations in the circulation of the blood, the vessels and the tissues which arise under the influence of a pathogenic stimulus and which as a rule aim to eliminate the irritating action.(13) (30) (68)

The latter is represented by local stimuli of various kinds, mechanical, thermic, chemical, electrical, radiant, infectious, etc.

What is very interesting from a biological point of view is the compulsoriness of the various stages, which result in being programmed for and focused on one purpose only, that of restoring the homeostasis by means of a compulsory iter which generates a temporary unbalance. (13)

If we want to summarise the various phases of the inflammatory process, we can say that there is a first phase, the acute phase, which begins from the moment in which the tissue undergoes abuse and persists for the first 2-3 days.

During this phase the vasomotor reactions are the most important because the first thing that happens is a vasodilatation followed by a tightening-up and then a closing of the vessels.

The dynamics of local circulation have the purpose of bringing a large quantity of blood, of serum which, through the increased permeability of the capillaries, diffuses to the outside, thus diluting the concentration of pathogenic agents, flooding the territory with agglutinins, precepitins, lysins which are needed to link and neutralise the bacteria or the toxins that these produce. (30) (68)

The large amount of fibrine forms a thick grid whose function is to trap the above mentioned pathogenic substances.

Finally in the area of damaged tissue leucocytes appear, the white cells which are needed for the lysis of the bacterial cells or the necrotic ones.

The following phase which directly proceeds the previous one, is the sub-acute phase. (13) (30)

During this phase features the proliferation of granulated tissue which is a transitory form of tissue responsible for the process of tissue repair.

This tissue is characterized by the presence of other cells acting as phagocytes, the scavenger cells which "clean" the tissue of the cell detritus, of the damaged cells or of the leucocytes who have in turn absorbed bacteria or viruses.

During this phase there is also the neo-formation of blood vessels, in order to bring new substances from the vascular stream given that an anabolic phase is beginning at this stage. Moreover the blood can enter into contact with the macrophages of the vascular stream, thus helping to produce antibodies.

Still during this phase that persists up to the 5^{th} or 6^{th} day, cells of mesenchymal origin are to be found (depending on the type of tissue fibreblasts, osteoblasts, etc.) which begin the considerable synthesis and depositing of collagene fibre. (13) (30) (68)

In this way the phase of synthesis begins which tends to substitute the loss of tissue substance with the depositing of connective tissue which with time will tend to, at least from a functional point of view, assume the behavioural characteristics of the tissue which it has substituted.

This synthetic phase begins about the 6^{th} day of the inflammatory process and persists up to the 28^{th} day; at first the synthesis of collagene is very accentuated, the metabolic tissue rate is increased and is sustained by the increased haematic supply. As the days pass, the synthesis of collagene decreases as do the number of vessels which occlude and then disappear.

Finally there is the remodelling phase, the last, which from the 28^{th} day persists until at least the 120^{th} day and whose main feature is the spatial reorganisation, the remodelling according to the force lines of the newly synthesized collagene fibres, in order to restore the original functions of the tissue.

This detailed analysis of the inflammatory process makes clear some fundamental aspects which should be taken as guide lines for a correct use of heat (29):

1. in the acute phase (0-3 days) the use of heat is contraindicated because important vasomotor phenomena are already in course. Often, above all in traumatic damage, the vessels are ruptured and this is followed by a haematic overflow; the vasodilatory effect induced by the heat interferes with the normal processes of coagulation and all this is even more evident in a muscle sprain where there is a true haemorrhage which would continue with the application of heat.(22) (23)
2. in the sub-acute phase (3^{rd} - 6^{th} day), by which time the vasomotors have become stabilized, we can begin to apply heat to the tissues with however great care and not using too high temperatures and heat levels because the reflex responses thus obtained can easily cause either the pain or the focus of inflammation to return.
3. In the synthetic phase (6^{th} – 28^{th} day), the use of heat is at its best; the initial crisis has passed, there is a vasal neo-formation with the new capillaries

able to respond by reflex to the heat stimulation. From a medical point of view, it seems methodologically correct to sustain the haematic supply to tissue in which important synthetic processes are taking place and whose basal metabolism has increased because this sustains the tissue repair. (15)

4. The same is true for the remodelling phase (28^{th} – 120^{th} day and beyond) because, even if the neo-vessels have by now completely disappeared, it is however possible to obtain an increase in the locoregional haematic flux avoiding those conditions which could encourage degenerative phenomena, the root cause of which seems to be a reduced vascularization. (13) (32) (48) (Tab.3).

Therapeutic indications

In the light of what has been said above it is understandable why deep heat treatment with Hypertherm is indicated for the treatment of a large number and many types of pathologies.

Taking into account the biological time needed for tissue repair, with regard to acute pathologies, and that many kinds of chronic pain of the skeletal muscle apparatus are due to a vascular degeneration of the tissues, we can conclude that treatment with Hypertherm is indicated in sub-acute and chronic pathologies, of the inflammatory or degenerative kind, in the compact connective tissue (tendons, joint capsula, ligaments), bone tissue, muscle tissue and cartilaginous tissue and the anatomic areas where these tissues are variously present together. (14) (45) (46) (55)

PATHOLOGY GROUPS

Tendons	**Muscles**	**Osteo-cartilage**	**Neural**
tendonitis	Contractures	contusions	neuroma
peritendonitis	Contusions	distortions	canalicular syndromes
tendosynovitis	Sprains	arthrosis	
insertional tendinosis	Ossifying myositis	periostitis	
tendinosis		fractures	

Based on our experience we can affirm that when we subject patients with recent pathologies to the treatment with Hypertherm their painful symptoms are sometimes accentuated especially at the end of the first session. (35). At this point at least two questions must arise; whether it is the case that we have began the treatment too early on, and if this is so we are still in the acute phase of the inflammation and whether we have used suitable temperature levels for that particular patient, with

regard to his or her personal pain threshold and with regard to the type of pathology from which he or she is suffering.

Before the patient is subjected to a treatment with Hypertherm it is essential that he or she undergoes a physiatric medical examination in order to make an exact diagnosis and to pinpoint any conditions which constitute contraindications with regard to the application of heat. (2)

Pathologies in the acute phase, serious cardiocirculatory problems, insufficient microcirculation (diabetes), tumours, TBC, local or general infectious diseases, pace-maker wearers, skin infections inthe area to be treated, pregnancy (if the pathology is located near the abdomen) are all explicitly contraindicated for treatment with Hypertherm.

There are instead contraindications relative to situations in which the treatment can be carried out but cautiously, with a more careful monitoring of the reactions and the conditions of the patient during the heat therapy session. These contraindications are the following: the presence of ischaemia in the tissues, obesity, a prosthesis or metal implant, the presence of cartilage growth, thrombosis or haemorrhagic diseases, the locoregional areas of anaesthesia.

The main guide-line for a correct use of heat therapy is strictly related to the stages the illness takes; if it is in the sub-acute phase, gentle heat will be applied, while for chronic pathologies it can be stronger.

Finally we would like to mention a few future uses of Hypertherm, in particular in the treating of senile osteoporosis and in the healing process of fractures. (38) (42)

Medical studies have in fact been carried out on a group of patients suffering from senile osteoporosis with a low turn-over, with densitometer bone values of the rhachis below the norm and pain in the lumbar region, who were subjected to two cycles of deep heat treatment each lasting 10 days in a period of one year. The data obtained and published in a specialized scientific journal allowed a comparative study to be made between this group and another group of patients of the same type who were not subjected to endothermia treatment. (42)

This study showed a significant improvement for the better with regard to the pain symptoms of the treated patients in comparison to the untreated patients and the statistics are also significant in the fact that the bone density was seen to improve in the treated patients and worsen in the untreated ones.

Another study, this time experimental, was conducted on rabbits in order to evaluate the effect of Hypertherm on the healing process of fractures after the surgical application of two catheters in the femur to measure the inner temperatures and to stimulate bone-growth. (37) (38)

Without going into details, in conclusion the study pointed out how the researchers, by applying deep heat in association with the mechanically abused bone tissue, were able to stimulate a more abundant, more rapid and more organised depositing of bone tissue, compared to the rabbits with femoral catheters that were not subjected to endothermic treatment. These results could be extended to man and give rise to a question, whether those pathologies which have already seen a powerful stimulus of bone-growth, for example in fractures, can benefit in any way

from endothermic treatment, avoiding or limiting the occurence of complications such as osteonecrosis, delays in consolidation or pseudoarthrosis.

New and extremely fascinating fields of research are thus opening up aimed at pinpointing new pathologies on which heat could have a healing effect while at the same time curing the symptoms, contributing in this way, together with all the other rehabilitation methodologies, both physical and manual, to the complete functional recovery of the patient.

Tables

Tab.1: Biological effects of heat.

↑tissue temperature
vasodilatation (microcirculation)
↑haematic flow ; ↓speed ; ↑contact time
↑O2 free
↑supply of metabolites
↑number and speed of chemical and biochemical reactions
↑removal of catabolites and exudates
↓discharge of pain receptors
modest "cell-killing"

Tab.2: effects of heat on the skeletal muscle apparatus

↑blood flow
↑collagene extensibility
↑contractile muscle efficiency
↓joint stiffness
↓muscle spasm
↓inflammatory infiltrates, oedema, exudates
↓pain

Tab.3: inflammation phases

BIOLOGICAL PROCESSES	PHASES
0-3rd day - vasomotor reactions + exudates + leucocoytes + fibrine	acute
2nd-4thday – macrophages for phagocytes + fibreblasts + neovessels	
5th-6thday- granulation tissue and neovessel max. expansion	sub-acute
6th-28thday- collagene synthesis and then ↓ neovessels	synthesis
28th-120thday- reorganisation and remodelling of neocollagene	remodelling

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